ICK, NOT SICK! HORMONAL MODULATION OF PHYSIOLOGICAL AND BEHAVIORAL RESPONSES TO PATHOGEN THREAT

by

JENNA MICHELLE LUNGE

A dissertation submitted in partial fulfillment of the
requirements for the degree of

DOCTOR OF PHILOSOPHY IN PSYCHOLOGY

2026

Oakland University
Rochester, Michigan

Doctoral Advisory Committee:

Lisa L. M. Welling, Ph.D., Chair
Andrea T. Kozak, Ph.D.
Melissa M. McDonald, Ph.D.

To the students I have had the privilege to teach, and to those I have not yet met –

Your curiosity and love of learning have inspired me to be a better scholar, educator, and person. Learning alongside my students has been my greatest motivation and my greatest reward.

You've made this whole thing worth it.

-Dr. Lunge

ACKNOWLEDGMENTS

I would like to thank my advisor, Lisa Welling, for her mentorship throughout this dissertation and the decade that has led to it. Thank you to the other members of my committee, Andrea Kozak and Melissa McDonald, and to all the Psychology faculty for their wisdom and support over the years. I have had the privilege to learn alongside some incredible friends during graduate school, several of whom I would like to acknowledge. To Ginny Mitchell and Liz Taylor, for the conversations that shaped my pedagogy and inspired my growth as an educator. To Kat Sheetz, for the countless hours spent coworking, sorting spit, running my ELISAs when I was teaching, and making my graphs prettier in *R*, and for proving that all of this is more fun with a friend. And to Taylor Elam, Emma Walquist, and Gina Montalto, who met me with encouragement when I had doubts, levity when I needed it, and showed me that even on the hard days, there is always room for laughter–your friendship has healed me in ways I can't adequately describe. Thank you to my parents and family for their love, confidence, and pride–no matter what I'm doing, you're my biggest fans. Finally, thank you to my husband, Ryan May. For building a life with me in Michigan. For taking care of our home, our pets, and me, and carrying more than your share so I could do this. For your genuine interest in my work and the pride you so clearly have when sharing it with others. Thank you for showing me that life should still be lived during a PhD. It may have taken me longer to get here, but because of you, I got to live a lot of life along the way.

Jenna Michelle Lunge

ABSTRACT

ICK, NOT SICK! HORMONAL MODULATION OF PHYSIOLOGICAL AND BEHAVIORAL RESPONSES TO PATHOGEN THREAT

by

Jenna Michelle Lunge

Adviser: Lisa L. M. Welling, Ph.D.

The Behavioral Immune System (BIS) helps reduce infection risk through responses such as disgust and pathogen avoidance. The Compensatory Prophylaxis Hypothesis (CPH) proposes that immunomodulation associated with heightened progesterone increases BIS reactivity to offset this vulnerability. This dissertation examined how endogenous progesterone and exogenous progestin (via oral contraceptives [OCs]) shape behavioral and physiological responses to pathogen threat across three studies. It further tested the prediction that behavioral and physiological defenses would show complementary coordination under low vulnerability and compensatory coordination under conditions of heightened vulnerability.

In Study 1, pathogen-salient cues increased state disgust, and progestogenic activity shaped when individual differences in perceived infectability (PI) were associated with pathogen avoidance motivation. In Study 2, pathogen cues again increased disgust, which in turn predicted greater pathogen avoidance and reduced inflammatory reactivity (IL-6), supporting a compensatory pattern of coordination between behavioral and physiological responses. These effects were moderated by PI and

hormonal context, with the compensatory pathway emerging among naturally cycling luteal-, but not follicular-, phase women, consistent with the CPH, and across OC users, although attenuated at higher levels of progestogenic activity. In Study 3, there was little evidence that pathogen threat or hormonal context reliably predicted intergroup attitudes.

Across studies, state disgust emerged as the most consistent indicator of BIS activation and had a central role in linking pathogen threat to downstream defensive responses. Overall, this dissertation supports a flexible, context-dependent coordination between behavioral and physiological immune responses and refines the CPH by demonstrating that hormonal influences shape pathogen defense.

TABLE OF CONTENTS

TABLE OF CONTENTS—Continued

TABLE OF CONTENTS—Continued

TABLE OF CONTENTS—Continued

TABLE OF CONTENTS—Continued

APPENDICES

LIST OF TABLES

LIST OF FIGURES

LIST OF FIGURES—Continued

LIST OF ABBREVIATIONS

BIS	Behavioral Immune System
DC	Dendritic cell
PAMP	Pathogen-associated molecular pattern
PRR	Pattern recognition receptor
TNF-α	Tumor necrosis factor alpha
IL	Interleukin
IL-1β	Interleukin-1 beta
CRP	C-reactive protein
PGE_2	Prostaglandin E_2
HPA	Hypothalamic-pituitary-adrenal
APC	Antigen presenting cell
Th	T helper cell
Tfh	T follicular helper cell
Treg	Regulatory T cell
IFN-γ	Interferon-gamma
TGF-β	Transforming growth factor-beta
NK	Natural killer
HC	Hormonal contraceptive
H1N1	Influenza A
MERS	Middle East Respiratory Syndrome
CoV	Coronavirus

SARS	Severe acute respiratory syndrome
COVID-19	Coronavirus Disease 2019
CPH	Compensatory Prophylaxis Hypothesis
NC	Naturally cycling
IUD	Intrauterine device
OC	Oral contraceptive
PI	Perceived infectability
MPA	Medroxyprogesterone acetate
IRB	Institutional Review Board
PVD	Perceived Vulnerability to Disease
DPSS–R	Disgust Propensity and Sensitivity Scale–Revised
DS–R	Disgust Scale–Revised
OCD	Obsessive Compulsive Disorder
C-DIS	Culpepper Disgust Image Set
SPA	Situational Pathogen Avoidance
EE	Ethinyl estradiol
FE	Ferrous fumarate
LNG	Levonorgestrel
µg	Micrograms
Mg	Milligrams
NE	Norethindrone
NEA	Norethindrone acetate
NG	Norgestimate

M	Mean
SD	Standard deviation
F	Critical F-value
t	Critical t-value
p	Critical p-value
n^2	Eta-squared effect size
HC4	Heteroskedasticity-consistent
CI	Confidence interval
VIF	Variance inflation factor
ALLO	Allopregnanolone
sIgA	Salivary secretory immunoglobulin A
GR	Glucocorticoid receptor
DES	Desogestrel
OD	Optical density
LOD	Limit of detection
HRP	Horseradish peroxidase
TMB	Tetramethylbenzidine
nm	Nanometers
CV	Coefficient of variation
ΔIL-6	IL-6 reactivity (post-test minus baseline)
ANCOVA	Analysis of covariance
LMM	Linear mixed-effects model
ANOVA	Analysis of variance

REML	Restricted maximum likelihood
POC	People of Color
SR2K	Symbolic Racism 2000 Scale
SR-Immigrants	Symbolic racism toward immigrants
SR-POC	Symbolic racism toward People of Color
GENE	Revised Generalized Ethnocentrism Scale
MANCOVA	Multivariate analysis of covariance

CHAPTER ONE

INTRODUCTION

Preventing harm from bacteria, viruses, and other microorganisms is a fundamental physiological priority. Although immune system needs vary across organisms, all living organisms – from prokaryotic bacteria and archaea to plants and animals – exhibit defenses against immune challenge (see Rimer et al., 2014). At its most primitive, for example among prokaryotic cells, immune responses protect from pathogenic infection (Tock & Dryden, 2005). Vertebrates possess more complex immune systems that also detect cellular damage, facilitate tissue repair, and maintain immunological memory. Relatively recently, researchers have identified a psychological component of infection prevention known as the behavioral immune system (BIS; Schaller, 2011; Schaller & Park, 2011). The BIS is a disease avoidance system composed of cognitive, emotional, and behavioral responses that function to *prevent* infection from environmental pathogens. Responses of this system detect and evaluate the presence of pathogenic sources in an environment, motivate psychological states and behavior to avoid pathogens, and ultimately manage long-term risk of exposure to infection and disease.

The BIS is a coordinated effort of *proactive* and *reactive* outputs to limit harm from environmental pathogens (reviewed in Ackerman et al., 2018). Proactive responses reduce long-term risk by motivating behaviors such as basic hygiene (e.g., bathing with soap and water; Curtis & Biran, 2001) and preferences for healthy-looking mates,

including traits like facial symmetry, sexual dimorphism, and averageness (reviewed in Little et al., 2011; Welling, 2025), which are indicative of immunocompetence (Foo et al., 2020; Rhodes et al., 2007). These preferences may reduce exposure to illness (e.g., Jones et al., 2001; Thornhill & Gangestad, 2006) and increase offspring viability through enhanced immune function (reviewed in Tybur & Gangestad, 2011).

Reactive BIS responses occur after exposure to cues of immediate pathogen risk (e.g., bodily secretions, rotting food). These include physical withdrawal, watery eyes, nausea, retching, and vomiting, which either prevent exposure to pathogens or expel pathogens that may have already entered the body. The dominant reactive emotion associated with the BIS is disgust, which likely evolved to motivate these pathogen-avoidant behaviors. Although violations of social norms (e.g., stealing) and behaviors that can threaten reproductive success (e.g., infidelity) do elicit moral disgust and sexual disgust, respectively (e.g., Tybur et al., 2009), disgust is most frequently reported alongside exposure to potentially pathogenic sources (reviewed in Oaten et al., 2009). Given that the most common elicitors of disgust (reviewed in Curtis & Biran, 2001) do readily carry and transmit parasites, there is a clear role of contagion prevention in disgust.

BIS reactions are context-sensitive and vary based on transmission risk. For example, although viewing wound maggots readily induces disgust (Kamber et al., 2020), other ectoparasites (i.e., parasites that live on or in the skin of their host) are more commonly associated with feelings of itch, motivating behavioral outputs such as scratching to physically remove the ectoparasite (reviewed in Kupfer & Fessler, 2018). Because the presence of maggots necessitates a nearby source of decay or decomposition

(e.g., rotting flesh, spoiled food), disgust is likely motivated in the presence of maggots, but not necessarily other ectoparasites, to prevent the ingestion of those rotting foods and the pathogens within them. In contrast, itch is strongly motivated upon cues of nearby ectoparasites, such as others' scratching (Ogden & Zoukas, 2010) or when viewing lice, scabies, or bed bugs (e.g., Kamber et al., 2020).

Individual differences in disgust propensity (likelihood of feeling disgust) and disgust sensitivity (degree of distress) predict disease-avoidance behaviors (van Overveld et al., 2006). People high in disgust propensity and disgust sensitivity are less likely to engage in disgust-inducing tasks, such as drinking from a dog's bowl or eating horse meat (van Overveld et al., 2010), and engage in more frequent and lengthier handwashing after exposure to a pathogen threat (Thorpe et al., 2011; Tolin et al., 2006). In the wake of the COVID-19 pandemic, higher disgust sensitivity predicted disease avoidance behaviors such as social distancing, handwashing, and disinfecting (Shook et al., 2020).

Together, proactive and reactive BIS responses work to mitigate immediate and long-term infection risk by motivating cognitive, affective, and behavioral mechanisms to prevent pathogen exposure. When pathogen exposure occurs, the physiological immune system responds to defend the body against infection and disease via its two major branches: the innate and adaptive immune systems. The innate immune system is the body's first line of defense against pathogens, and it acts rapidly and nonspecifically using physical defenses (e.g., skin, secretions), antimicrobial cells, and messengers that quickly induce inflammation and coordinate neural changes in response to infection, tissue damage, or certain imbalances of homeostasis (e.g., autoimmune disorders, diabetes). Over time, the adaptive immune system develops a targeted response that is

pathogen-specific and capable of retaining immunological memory of microorganisms that have been encountered.

Cytokine Signaling and the Immune Response

Innate immune cells, such as dendritic cells (DCs) and macrophages, detect certain patterns that are associated with pathogens (e.g., bacterial flagella, viral spike proteins) called pathogen-associated molecular patterns (PAMPs) via pattern recognition receptors (PRRs). When PRRs bind PAMPs, the detecting cell becomes activated and triggers a massive release of cytokines, which are a class of small, potent signaling proteins. Cytokines act locally, amplify their release, and circulate to different target organs, producing peripheral and central physiological changes (reviewed in Lokau & Garbers, 2020).

Cytokines initiate the acute phase reaction, a coordinated inflammatory response with local, systemic, and central components (reviewed in Pecchi et al., 2009). Locally, proinflammatory cytokines such as tumor necrosis factor alpha (TNF-α), interleukin (IL)-1 beta (IL-1β), and IL-6 initiate inflammation by, for example, increasing vascular permeability (Gardiner et al., 1998; Spriggs et al., 1988) via nitric oxide production (Rosenkranz-Weiss et al., 1994; Satoh et al., 1997; Shindo et al., 1995), allowing immune cells to access the site of infection. Cytokines also diffuse into the bloodstream to recruit immune cells to the site of infection or damage (e.g., Fenton et al., 2002) and to mediate the broader immune response (reviewed in Medzhitov, 2007). Ultimately, the local response increases inflammation with blood flow to target areas, improving cellular recruitment and promoting phagocytosis (i.e., the process by which immune cells called phagocytes engulf and ingest foreign particles, such as pathogens) to eliminate

pathogenic material and debris. IL-6 and other cytokines also reach the liver, inducing the systemic acute phase reaction (reviewed in Gruys et al., 2005), which amplifies inflammation through cellular recruitment, protein transport, detoxification, complement activation (i.e., a cascade of proteins that enhance inflammation and phagocytosis), coagulation, and tissue repair (reviewed in Cray, 2012; Mantovani & Garlanda, 2023). In the liver, IL-6 binds to hepatocytes (functional liver cells) and induces the synthesis of acute phase proteins such as C-reactive protein (CRP), which promotes phagocytosis (e.g., Kindmark, 1971) and further cytokine release (Ballou & Lozanski, 1992).

Cytokines act on the central nervous system to trigger the central acute phase reaction, inducing fever, stress response, and sickness behaviors such as shivering (reviewed in Pecchi et al., 2009). Fever enhances the proliferation and activity of immune cells (e.g., Capitano et al., 2012), inhibits pathogen replication (Kluger & Rothenburg, 1979), and is observed alongside inflammation in all vertebrates during immune challenge (Boltaña et al., 2013; Nahas et al., 2016; reviewed in Evans et al., 2015). Indeed, individuals in cold environments or administered antipyretic (fever-reducing) medications do not generate fever responses and are at greater risk of fatal infections (reviewed in Kluger, 1986). Although IL-6 alone is only weakly pyrogenic (i.e., it does not induce fever when centrally injected; Lacroix & Rivest, 1998; LeMay et al., 1990), it sustains fever (e.g., Chai et al., 1996; Nilsberth et al., 2009; Rummel et al., 2011). Cytokines signal the brain through vagal nerve pathways at low concentrations (i.e., the inflammatory reflex; Breder et al., 1988; see also Binshtok et al., 2008; Hagenacker et al., 2010; Steinberg et al., 2016; Watkins et al., 1995) and via blood-brain barrier signaling at higher concentrations (Capuron & Miller, 2011), where they induce prostaglandin E_2

(PGE_2; a small inflammatory lipid mediator). PGE_2 crosses into the hypothalamus and increases the thermoregulatory set-point, producing fever and associated behavioral and physiological responses (Blomqvist & Engblom, 2018). Proinflammatory cytokines also activate the hypothalamic-pituitary-adrenal (HPA) axis, leading to glucocorticoid release (e.g., cortisol), which suppresses cytokine expression and limits inflammation (reviewed in Chrousos, 1995; Rivest, 2001). In this way, anti-inflammatory activity of the HPA-axis restrains inflammation, prevents excessive tissue damage, and restores homeostasis once pathogens and infection are cleared.

Beyond coordinating inflammation, cytokines link the innate and adaptive immune systems by influencing T cell differentiation. Specifically, crosstalk between the innate and adaptive systems by way of cytokines enables an immune response that is highly tailored to the pathogens first encountered by immune cells at the site of infection. The innate immune response rapidly develops as a first-line defense against infection, whereas the adaptive immune system takes 4-7 days to mount a targeted and longer-lasting response driven by T cells (i.e., T lymphocytes; e.g., Soderberg et al., 2005). Upon PAMP detection through their PRRs, antigen-presenting cells (APCs)—mainly DCs, macrophages, and B cells (i.e., antibody-producing cells of the adaptive immune system)—internalize pathogens (i.e., endocytosis), generate antigens, and present them on major histocompatibility complex class II proteins. APCs then travel to the secondary lymphoid organs (e.g., lymph nodes, spleen) and present antigens and polarizing cytokines (i.e., cytokines that influence the differentiation and functional state of other cells) to activate and shape the differentiation of T cells. T cells differentiate into a mature effector cell subtype based on signals from the type and quantity of antigens and

polarizing cytokines present in the environment (reviewed in Malissen et al., 2014), as well as the lineage (i.e., the developmental pathway) of the APC and the unique epigenetic regulators present in the specific T cell subtype (reviewed in Dutta et al., 2021). In other words, APCs identify the invading threat based on the PRR that has been activated and relay this information to T cells via antigens and polarizing cytokines, in turn promoting T cell differentiation into a subtype that maximizes clearance of the identified threat.

T cells derive from common precursor cells, but differentiate into T helper cells (Th cells, also known as $CD4^+$ T cells) or cytotoxic T cells (also known as $CD8^+$ T cells) under genetic and epigenetic control (Hosokawa & Rothenberg, 2020). Mature Th cells guide the adaptive immune response by releasing cytokines to activate and coordinate other immune cells, and cytotoxic T cells kill infected or abnormal cells by inducing apoptosis (i.e., programmed cell death).

Th cells differentiate into distinct effector lineages with specialized immunological functions shaped by polarizing cytokines present during activation (reviewed in Chatzileontiadou et al., 2021), including Th1, Th2, Th17, T follicular helper (Tfh) cells, and regulatory T cells (Tregs). Models of *human* Th differentiation consistently demonstrate that specific cytokine environments direct lineage commitment whereby, in combination with cooperating polarizing cytokines, IL-12 and interferon-gamma (IFN-γ) promote Th1 differentiation, IL-4 promotes Th2 differentiation, IL-6 and transforming growth factor-beta (TGF-β) together promote Th17 differentiation, and TGF-β alone promotes Tregs (reviewed in Hilligan & Ronchese, 2020; Schmitt & Ueno, 2015). These polarizing cytokines are released in response to pathogen recognition and

determine which type of Th cell a naïve (undifferentiated) T cell will become based on the immune context.

Each Th lineage has a distinct role in coordinating immune responses and activating other effector cells (e.g., B cells, cytotoxic T cells), with characteristic cytokine secretion profiles and specific pathogen targets (reviewed in Chatzileontiadou et al., 2021). Th1 cells predominantly secrete IFN-γ, IL-2, and TNF-α, which promote responses to intracellular pathogens like viruses and certain bacteria (reviewed in Elenkov & Chrousos, 1999). These cytokines drive cell-mediated immunity (i.e., destruction of pathogens or infected cells) by priming macrophages for phagocytosis (Pace et al., 1983) and activating cytotoxic T cells and natural killer (NK) cells, which are innate immune cells that destroy infected or abnormal cells (Mosmann & Coffman, 1989). In contrast, Th2 cells secrete IL-4, IL-5, and IL-13 in response to toxins, allergens, and large extracellular parasites (reviewed in Hammad et al., 2022). These cytokines promote humoral immunity (mediated by large molecules in the extracellular fluids) by facilitating antibody production and suppressing Th1-mediated activities such as macrophage activation and proinflammatory cytokines (reviewed in Mantovani et al., 2004). Th17 cells secrete IL-17 and IL-22 to induce inflammation and are implicated in the pathogenesis of autoimmune disorders (e.g., psoriasis, rheumatoid arthritis; reviewed in Hilligan & Ronchese, 2020) and other immune-mediated diseases (e.g., asthma, inflammatory bowel disease; reviewed in Tesmer et al., 2008). Tfh and Treg cells provide broader immunomodulatory functions that are not restricted to particular pathogens (reviewed in Crotty, 2019; Shevyrev & Tereshchenko, 2020). Additional Th subsets (e.g., Th3, Th5, Th9, Th22, Th25) have also been described (see Osum & Jenkins, 2023),

though their functions are beyond the scope of this dissertation.

Interactions Between the Physiological and Behavioral Immune Systems

During immunological challenge, the body prioritizes expending energy on fever and inflammation, and animals exhibit sickness behavior (proposed by Hart, 1988; Kent et al., 1992), a suite of behavioral changes such as lethargy, social withdrawal, and decreased appetite that conserves energy for immune function (reviewed in Dantzer, 2001). Energetically costly activities (e.g., searching for food or mates) are deprioritized, whereas behaviors that conserve or generate heat (e.g., rest, shivering) are prioritized. By de-emphasizing activities unrelated to immediate survival, sickness behavior also minimizes further exposure to pathogens.

APCs that detect pathogens and initiate the acute phase reaction also secrete cytokines that shape adaptive immune responses. The adaptive immune system provides pathogen-specific responses characterized by antibody production, cytotoxic capabilities, and immunological memory. Cytotoxic and antibody-producing cells require activation by Th cells, which differentiate into effector subtypes in response to APC-derived polarizing cytokines and subsequently coordinate downstream immune response. In all, cytokines coordinate the innate and adaptive immunity, as well as broader physiological and neurological processes.

Cytokines communicate peripherally to coordinate the innate and adaptive responses, but they also communicate with the brain to induce the central acute phase reaction, namely fever, HPA activity, and sickness behavior. The central response is *reactive*, beginning only after pathogen exposure, and is extremely energetically costly. For example, a 13% increase in metabolic rate is needed to induce just a 1º C increase in

human body temperature (reviewed in Dantzer, 2004), and a metabolic increase averaging between 9-30% occurs from increased lymphocyte activity after pathogen exposure (Straub et al., 2010). Metabolic energy could be conserved if individuals engaged in disease-avoidant behavior to avoid contact with pathogens in the first place.

Evidence linking the BIS and its physiological counterparts is mixed. Some studies find that disgust is associated with reduced infection risk (Cepon-Robins et al., 2021) and reduced inflammation (e.g., oxidative stress; Gassen et al., 2018), whereas others suggest disgust initiates early response immune activity to protect the body more rapidly from infection, should pathogen exposure occur (Oaten et al., 2009). Schaller and colleagues (2010) provided the first experimental evidence of inflammatory reactivity to visual cues of pathogens (e.g., sneezing, skin lesions), which was later supported by other research finding increased inflammation after exposure to pathogen-relevant stimuli (e.g., Keller et al., 2022; Schaller et al., 2010; Stevenson et al., 2011, 2012). However, some studies have found increased inflammation, but no differences between pathogen threat and non-pathogen threat conditions (Gassen et al., 2019; Stevenson et al., 2015).

Differentiating between *trait* and *state* disgust helps clarify BIS-immune system interactions. Trait disgust reflects the tendency to experience disgust across varied situations and refers to the stable influence of predispositions for disgust as a motivator for pathogen avoidant cognitions and general behaviors. State disgust involves current disgust feelings that may motivate pathogen avoidance. Unlike trait disgust, state disgust is reactive to situational shifts in environmental pathogen load (Makhanova et al., 2021). Importantly, studies that find support for interactions between the physiological and BIS often experimentally manipulate cues to the environmental pathogen load (e.g., images

depicting infection; Schaller et al., 2010) and measure behavioral immune reactivity at the state level (e.g., situational pathogen avoidance motivations; Makhanova et al., 2021).

Research has not yet clearly disentangled inflammatory reactivity from the general emotion of disgust (i.e., a *trait* manifestation of the BIS) compared to current feelings of disgust from engaging with pathogen-related stimuli (i.e., a *state* manifestation of the BIS). Nevertheless, trait disgust is important for behavioral immune activity. Individuals vary in their perceptions of how susceptible to infection they are, and this perceived vulnerability to disease has been shown to influence the intensity of pathogen avoidance motivations in the presence of potential pathogen threats (see Neuberg et al., 2011). For example, compared to individuals with low perceived vulnerability to disease, individuals high in perceived vulnerability to disease have stronger preferences for faces digitally altered to appear healthy (Welling et al., 2007), greater contamination-related obsessions and compulsions (Brady et al., 2021), and more disease-avoidance behaviors in the wake of COVID-19, such as social distancing, handwashing, and disinfecting (Shook et al., 2020).

Progesterone as an Anti-Inflammatory Hormone

One line of research evaluating the relationship between the physiological and behavioral immune systems involves the immunomodulatory effects of progesterone (reviewed in Raghupathy & Szekeres-Bartho, 2022; Zwahlen & Stute, 2023), a steroid reproductive hormone produced abundantly during pregnancy and in lower quantities during the luteal phase of the ovulatory cycle. Progesterone prepares the endometrium for implantation and facilitates an immunological shift that suppresses the development of an immune response toward fetal antigens (Tafuri et al., 1995). Progesterone-induced

immunomodulation extends beyond the uterine environment by profoundly impacting women's systemic immunocompetence and potentially contributing to behavioral adaptations that compensate for decreases in immune function.

At the cellular level, progesterone suppresses the inflammatory activity of various APCs. It reduces macrophage phagocytosis (Barañao et al., 1991), nitric oxide production, and Th1-polarizing cytokines (Arruvito et al., 2008; Menzies et al., 2011). Similarly, progesterone also inhibits cytotoxic NK cell activation (Faust et al., 1999). Progesterone-treated DCs produce fewer proinflammatory cytokines (e.g., TNF-α, IL-1β, IL-6, IL-12), but increase their anti-inflammatory cytokine production (e.g., IL-10; Butts et al., 2007; Jones et al., 2010). In addition to suppressing the Th1-like inflammatory cytokine profile of APCs, progesterone also directly manipulates naïve Th cell differentiation by inducing Th2 (Piccinni et al., 1995) and Treg (Lee et al., 2011) subsets. These immunosuppressive effects are vital for successful implantation and support a broader framework of immunomodulation during early pregnancy.

Typically, Th1 responses are cytotoxic and target intracellular pathogens (reviewed in Elenkov & Chrousos, 1999), whereas Th2 responses enhance antibody production to combat extracellular pathogens and promote tissue repair (reviewed in Mantovani et al., 2004). A balance between Th1/Th2 cells is necessary for normal immune responses, and perturbations to this balance contribute to the pathology of allergic, autoimmune, and inflammatory diseases (reviewed in Rahimi et al., 2019). A Th1-dominant response is usually considered essential for a robust immune defense against infection. In pregnant women, however, it poses a threat to the semi-allogeneic fetus, which contains antigens recognized by the immune system as foreign and harmful

(see Wegmann et al., 1993). Adaptively, progesterone mediates a shift in T cell polarization that inhibits Th1 cells (Veenstra van Nieuwenhoven et al., 2002) and promotes a state of Th2 dominance during pregnancy and, to a lesser extent, the luteal phase (Lissauer et al., 2015; Piccinni et al., 1995). Th2 cytokines like IL-4, IL-5, and IL-13 further reduce the risk of immune-mediated damage to the fetus by suppressing the Th1 response and instead promoting antibody-mediated immunity (reviewed in Wang et al., 2020).

The Th1/Th2 shift during pregnancy is an oversimplification, however, and improved understanding of Th lineages has led to a greater focus on Th17 cells and Tregs. High concentrations of IL-6 and TGF-β drive naïve Th cells to differentiate into the proinflammatory Th17 cells. In conjunction with proinflammatory cytokines like IFN-γ, IL-21, and IL-17, Th17 cells activate and recruit proinflammatory cells (e.g., NK cells, neutrophils, macrophages), activate the acute phase response, amplify the Th1 response, and further promote the Th17 response via positive feedback (reviewed in Osborne et al., 2019; Tang & Hu, 2023). Th17 cells defend against invasion from extracellular pathogens and fungi but, like the Th2 response, the presentation of paternal antigens from the semi-allogeneic fetus to naïve T cells drives a Th17 response that would be harmful to the fetus (Fu et al., 2014). As early as the luteal phase, progesterone suppresses Th17 differentiation (Nakashima et al., 2010) and promotes Tregs (Lee et al., 2011), which inhibit APCs, innate and adaptive effectors, and secrete regulatory cytokines (TGF-β, IL-10, IL-35) to further promote tolerance (Hays et al., 2019; Yang et al., 2023; Grover et al., 2021; Goodman et al., 2012; Vignali et al., 2008). Thus, progesterone helps maintain pregnancy by regulating Th1/Th2 and Th17/Treg balance

(reviewed in Saito et al., 2010).

Still, pregnancy is not uniformly anti-inflammatory. Rather, the maternal immune response is dynamic, with localized inflammation being necessary for implantation and labor. Principally, the window of implantation (i.e., 6-10 days post-ovulation during the luteal phase) requires a controlled and localized inflammatory response to successfully establish pregnancy. The follicular and periovulatory phases promote the expansion of proinflammatory cytokines, APCs (reviewed in Robertson et al., 2018), and Tregs (Arruvito et al., 2007), which begin to accumulate in the endometrium. Seminal fluid further enhances receptivity, driving additional proinflammatory mediators and T cells to the endometrium (Robertson et al., 2009; Sharkey et al., 2012) and priming T cells with paternally-derived antigens (Tilburgs et al., 2008). Mild inflammation during implantation enables trophoblasts (the fetal cells forming the outer layer of the conceptus) to invade the endometrium (Kaislasuo et al., 2020; Salama et al., 2020), but inflammation must be quickly constrained. By suppressing Th1 and Th17 activity (reviewed in Robertson et al., 2018) and driving Th2 (Piccinni et al., 2015) and Treg (Moldenhauer et al., 2009) differentiation, Tregs facilitate an anti-inflammatory shift that supports implantation and promotes immunological tolerance of the semi-allogeneic fetus. These immunological shifts function to prevent local inflammation, which is necessary for implantation, from escalating into a systemic immune response that could cause fetal rejection. Although pregnancy maintains a Th2- and Treg-dominant state, it shifts back to a Th1/Th17-dominant profile to trigger labor (Martínez-García et al., 2011; reviewed in Orefice, 2021).

Outside these key events, uncontrolled inflammation predicts adverse pregnancy

outcomes and inflammatory responses during pregnancy are complex. Elevated Th1 cytokines (IFN-γ, IL-2, and TNF-α) are associated with spontaneous abortion (e.g., Chaouat et al., 1990; reviewed in Raghupathy, 1997) and preeclampsia (reviewed in Saito & Sakai, 2003), whereas elevated Th2 cytokines (IL-4 and IL-13) are associated with healthy pregnancies (e.g., Chaouat et al., 1995; reviewed in Raghupathy, 2001). Recurrent pregnancy loss is marked by systemic Th1 (Ng et al., 2002) and Th17 (Ghaebi et al., 2019) dominance after implantation failure, and uterine tissue from miscarriage has elevated concentrations of Th1 cytokines (Jin et al., 2011).

Although progesterone's immunomodulatory effects are crucial for establishing and maintaining pregnancy, this shift comes with significant trade-offs. Progesterone-induced immunomodulation leaves women more susceptible to many infections, as the immune system's ability to mount an effective antibacterial response is compromised. Women in their luteal phase are more susceptible to candidal vaginitis, an infection caused by the opportunistic yeast *Candida albicans*, which can evade immune clearance and is more prevalent during immune suppression (Kalo-Klein & Witkin, 1989). This increased susceptibility to *Candida* is modulated by progesterone-mediated immune reactivity; women in their luteal phase exhibit a poorer immune response and lower germination inhibition of *Candida*, and women using hormonal contraceptives (HCs) containing progestin produce less variable immune reactivity to *Candida* over time. Th1 suppression–a prerequisite for maintaining a healthy pregnancy–inhibits the effectiveness of the maternal immune response when challenged by pathogens that rely on the Th1 response for clearance (i.e., intracellular pathogens; see Smith, 1999). Pregnant women are more susceptible to infection from toxoplasmosis, listeriosis, and Q fever (a

foodborne illness derived from intracellular pathogens; Avelino et al., 2003; Goulet et al., 2012; Silk et al., 2012), and experience more severe cases of many diseases caused by intracellular pathogens (e.g., chlamydia, Influenza A [H1N1], malaria, Middle East Respiratory Syndrome [MERS]-Coronavirus [CoV], and severe acute respiratory syndrome [SARS]; Alfaraj et al., 2019; Baud & Greub, 2011; Lam et al., 2004; Taylor et al., 2011; van Kerkhove et al., 2011).

Maternal infection can lead to adverse pregnancy outcomes. Several pathogens (e.g., *Toxoplasma gondii*, rubella, cytomegalovirus, herpesvirus, *Listeria monocytogenes*, human papillomavirus, hepatitis virus, Zika virus) readily transmit vertically from mother to fetus (reviewed in León-Juárez et al., 2017) and are associated with miscarriage or preterm birth (reviewed in Heydarifard et al., 2022). The exact mechanism causing adverse fetal outcomes after placental transmission is unknown for many pathogens, however one process involves a reactive maternal Th1 immune response (reviewed in Heydarifard et al., 2022). Even non-transplacental infections (e.g., influenza) are associated with fetal risks (reviewed in Silasi et al., 2015).

Although data on SARS-CoV-2 infection susceptibility in pregnancy are limited (see Jamieson & Rasmussen, 2022), pregnant patients in one hospital that screened for SARS-CoV-2 among the pre-surgical population were 15.7 times more likely to have an asymptomatic infection compared to the general surgical population (Kelly et al., 2021). Pregnancy is most certainly associated with more severe cases of COVID-19, and numerous reports find that SARS-CoV-2 infected pregnant women are 3 times more likely to be admitted to the intensive care unit and nearly 2 times more likely to die from COVID-19-related complications than age-matched nulliparous women (reviewed in

Jamieson & Rasmussen, 2022). COVID-19 diagnosis is also associated with adverse pregnancy outcomes, including a greater risk of miscarriage (e.g., Balachandren et al., 2022), preterm birth (reviewed in Khalil & Al-Humadi, 2020), and stillbirth (reviewed in Allotey et al., 2020). The risk for stillbirth was particularly strong during the dominance of the Delta (B.1.617.2) variant within the United States (DeSisto et al., 2021).

The Compensatory Prophylaxis Hypothesis

The shifted immune response during pregnancy, along with the resulting increased infection risk, underscores the importance of behavioral adaptations to reduce women's vulnerability to infection. Greater sensitivity to disgust and increased pathogen-avoidance behaviors during periods of greater infection risk could help lower pathogen susceptibility. The Compensatory Prophylaxis Hypothesis (CPH) contends that times of high progesterone (i.e., luteal phase, pregnancy) and progesterone-induced immunomodulation are accompanied by increases in the behavioral immune response, such as greater disgust sensitivity and avoidance of pathogen sources (e.g., feces, raw meats; see Fessler, 2001). Progesterone mediates the shift in the immune response to support pregnancy success and is hypothesized to drive increases in disgust sensitivity when immune suppression is most pronounced (e.g., Bressan & Kramer, 2022). This behavioral adaptation offsets the immunomodulatory effects of progesterone, helping to compensate for women's heightened infection risk during the luteal phase and pregnancy.

Increases in disgust sensitivity have been clearly established among pregnant women, who report the greatest disgust sensitivity during the first trimester of pregnancy (Fessler et al., 2005; Zelaźniewicz & Pawłowski, 2015; but see Dlouhá et al., 2023), a time that is also characterized by the most frequent symptoms of nausea and vomiting

(e.g., Lacroix et al., 2000). Nausea and vomiting during the first trimester are potentially prophylactic responses that defend against food-borne pathogens (reviewed in Fessler, 2002). Recently, Kaňková and colleagues (2022) found direct support for the Compensatory Prophylaxis Hypothesis among pregnant women in their first trimester; greater disgust was associated with lower serum cytokine concentrations, including the Th1 cytokines IFN-γ, IL-2, and TNF-α, as well as a proinflammatory mediator of the innate immune system, IL-1β (but also TH2 cytokine IL-4).

Support for progesterone-mediated compensatory prophylaxis among nonpregnant women is less consistent. The luteal phase is associated with more manifestations of the BIS than are the follicular and menstrual phases; women in their luteal phase report more intense disgust to pathogen-relevant phrases (e.g., "touch feces"; Liu et al., 2023) and exhibit greater preference for healthy-looking faces (versus unhealthy-looking faces; Jones et al., 2005). Higher levels of progesterone in naturally cycling (NC) women have been associated with greater self-reported disgust sensitivity during the luteal phase (Zelaźniewicz et al., 2016), more intense ratings of disgust on stimuli depicting sources of pathogens (e.g., an open wound, crowded public transit; Fleischman & Fessler, 2011), and more pathogen-avoidance behaviors in public restrooms (Fleischman & Fessler, 2011). Among women actively fighting an infection, those in their luteal phase, compared to their follicular phase, report greater pathogen disgust, more contamination-related obsessions, and greater disgust toward pathogen-related images, including in studies using luteinizing hormone tests to confirm cycle phase and ovulation timing (Miłkowska et al., 2019, 2021). However, other research has not replicated this relationship between progesterone and increased disgust (Fessler &

Navarrete, 2003; Jones et al., 2018), and meat consumption (a potential pathogen source readily avoided during pregnancy; reviewed in Fessler, 2002; Flaxman & Sherman, 2000) is not reduced among women in the luteal phase (Fleischman & Fessler, 2011). Furthermore, Olatunji and colleagues (2020) found greater self-reported disgust among women in the *follicular* phase, which is characterized by relatively low progesterone levels.

It is possible that progesterone-induced behavioral prophylaxis is not best captured by questionnaires evaluating disgust at the trait level, but rather is better measured at the state level after the induction of a pathogen-threat. Studies finding no increases in pathogen disgust coinciding with increases in progesterone level (Jones et al., 2018) or the luteal phase (Fessler & Navarrete, 2003; Zelaźniewicz et al., 2016) assessed self-reported trait pathogen disgust. Questionnaires evaluating women's chronic tendencies to experience disgust may not be an appropriate measurement of progesterone-mediated compensatory prophylaxis. Studies that present images of pathogen threats (Fleischman & Fessler, 2011; Jones et al., 2005; but see Olatunji et al., 2020) or that include an assessment of women's current state of health (Miłkowska et al., 2019; see also, Miller & Maner, 2011; but see Tybur et al., 2020a) find greater support for state compensatory increases in BIS responses. Although the current literature is limited, it remains probable that pathogen threat must be salient to elicit increases in the behavioral immune responses that compensate for progesterone-induced immunomodulation.

Hormonal Contraceptives

HCs, which supply exogenous progestin (often combined with exogenous

estrogen), may influence inflammation and the BIS. The efficacy of hormonal contraception is established through central and local mechanisms (reviewed in Benagiano et al., 2004). Centrally, synthetic hormones interrupt the hypothalamic-pituitary-gonadal axis by suppressing the secretion of luteinizing hormone and follicle stimulating hormone from the pituitary gland through negative feedback, which prevents development of the ovarian follicle and inhibits ovulation (reviewed in Bastianelli et al., 2018). Locally, HCs thicken the cervical mucus, reduce ciliary motion in the fallopian tubes, and thin the endometrial lining, which together hinder implantation and sperm penetration and transport (reviewed in Brown & Allen, 2020).

All progestins also induce activity at the progesterone receptor and are full or partial progesterone agonists. Because progesterone induces immunomodulation, in part through activity at the progesterone receptor (e.g., Arruvito et al., 2008; Butts et al., 2007; reviewed by Hapgood et al., 2018), exogenously administered progestins should be capable of altering the immune system. Indeed, the anti-inflammatory action of progestins is highlighted by the clinical use of HCs to manage pain associated with endometriosis, a chronic inflammatory condition characterized by growth of endometrial glands and tissue outside of the uterus (reviewed in Gezer & Oral, 2015). Progestins such as norethindrone (Grandi et al., 2016b) and dienogest (Grandi et al., 2016a), for example, exert numerous anti-inflammatory effects on endometrial tissues, such as the inhibition of proinflammatory cytokines IL-1β, IL-6, IL-8, and TNF-α. The anti-inflammatory capabilities of progestin are not isolated to the endometrium (reviewed in Hall & Klein, 2017). For example, *in vitro*, norgestrel (Jones et al., 2010) and levonorgestrel (Quispe Calla et al., 2016b) inhibit the activation of DCs, prevent their secretion of

proinflammatory cytokines, and suppress cytotoxic and helper T cells activation and proliferation.

Because some progestins within HCs circulate systemically (reviewed in Bick et al., 2021), including at low levels from certain intrauterine devices (IUDs; see Ewies, 2009), it is likely that HC use is associated with immunological impairment. Concerningly, women using oral contraceptives (OCs) containing both progestin and synthetic estradiol (i.e., combined OCs) exhibit numerous disturbances to their immune function, which are hypothesized to contribute to an increased susceptibility to diseases, including sexually transmitted infections (reviewed in Brabin, 2004). Women using combined OCs and those using the contraceptive NuvaRing (a contraceptive ring containing ethinyl estradiol and the progestin etonogestrel that is inserted into the vagina) have fewer Langerhans cells (early response antigen-presenting macrophages) in the vaginal epithelium than non-contracepted controls (Michel et al., 2015). OC users also have impaired systemic NK cell activity (e.g., Auerbach et al., 2002; Scanlan et al., 1995; Yovel et al., 2001). Unlike cytotoxic T cells, NK cells do not need to be activated by antigens to induce their cytotoxic effects, and so they respond much more rapidly if not suppressed. Moreover, women using OCs have an anti-inflammatory serum cytokine profile, with reduced levels of the proinflammatory cytokine IL-8, but greater concentrations of the anti-inflammatory Th2 cytokine IL-13 compared to non-contracepted women (Giraldo et al., 2008). The same study found that neutrophils isolated from OC users are less phagocytic than those from non-contracepted women. Taken together, progestin-induced immunomodulation may result in decreased immune functioning that could leave OC users, and possibly users of other HCs, more susceptible

to infection and disease.

It is possible that HC users display heightened behavioral immune activity to compensate for the immunomodulating effects of progestins in their HCs. Compared to NC women, OC users are worse at identifying facial expressions associated with disgust (Hamstra et al., 2014), suggesting blunted activity of the BIS, whereas others have found they have stronger preferences for healthy-looking faces (Jones et al., 2005), suggesting heightened activity of the BIS. Even more inconsistently, others found no significant difference between OC users and NC women in identifying disgust (Kimmig et al., 2022). These contradictory results suggest that the relationship between HC use and behavioral immune activity may be more complex than initially thought, warranting further investigation to clarify these effects.

Overview

This dissertation investigates the complex interplay between the physiological immune system, progesterone-mediated immunomodulation, and the cognitive, affective, and behavioral mechanisms of the BIS. It addresses the conditions under which these systems operate in complementary or compensatory ways by examining how trait-level vulnerability (e.g., perceived infectability [PI]) and hormonal status (endogenous progesterone or synthetic progestin exposure) shape behavioral and physiological responses to pathogen threat. Specifically, the current dissertation directly evaluated the CPH across three studies involving NC women and users of OCs. In Study 1, the CPH was examined among OC users during either their active pill phase (containing progestin) or their reminder pill phase (no progestin) to evaluate whether synthetic progestin exposure shaped state disgust and pathogen avoidance motivations following exposure to

pathogen-salient cues. Study 2 extended this work by assessing both physiological and behavioral immune responses in OC users and NC women before and after a pathogen threat manipulation. This study tested whether the physiological and behavioral immune systems operate in complementary or compensatory ways depending on hormonal context and PI. A direct marker of physiological immune activity, salivary IL-6 (a pleiotropic cytokine involved in inflammatory responding), was assayed to examine immune reactivity following pathogen threat exposure. Finally, Study 3 drew from the same sample as Study 2 and focused on broader social-cognitive dimensions of the BIS. It examined whether pathogen salience, hormonal context, and individual vulnerability were associated with intergroup attitudes, including symbolic racism and ethnocentrism.

Across all studies, individual differences in PI were assessed to determine how trait-level perceptions of infection risk shape behavioral and physiological immune responding. Together, this three-study series provides a comprehensive test of the CPH and offers new insight into how hormonal and dispositional factors interact to regulate pathogen defense across behavioral, immunological, and social domains.

CHAPTER TWO

STUDY 1

There are currently no studies that directly assess the mediating effect of perceived vulnerability to infection on the relationship between progesterone and compensatory prophylactic behaviors. Olatunji and colleagues (2020) recorded pathogen-avoidance behaviors (e.g., unwillingness to touch the floor in front of a toilet in a public restroom) after a disgust induction among women in their follicular or luteal phase who were assigned to engage in either emotional suppression or emotion reappraisal regulation strategies. The authors measured disease vulnerability perceptions to check for group-level differences in trait disgust sensitivity between the four groups, and because the groups did not significantly differ in perceived vulnerability to disease, this trait was not included in later analyses. Importantly, differences in perceived vulnerability between all participants in the follicular phase and all participants in the luteal phase were not reported. Although this study found that women in the follicular phase engaged in more pathogen avoidance behaviors than women in the luteal phase, it did not directly assess whether women's perceived vulnerability to disease mediated the relationship between ovulatory cycle phase (i.e., estimated progesterone level) and state pathogen avoidance.

The immunomodulatory effects of progestins may be influential for HC users' behavioral immune reactivity. Limited research has identified some associations between HC use and accurately detecting facial expressions (Hamstra et al., 2014; Kimmig et al., 2022), but these studies investigated all OC users as one group and the unique

characteristics of the synthetic compounds within the contraceptives were not considered.

HCs are formulated with one of several forms of progestin that differ in chemical structure. Although all progestins bind to the progesterone receptor, they are broadly categorized into two groups with distinct biological activities: (1) those with structural similarities to progesterone, and (2) those with structural similarities to testosterone. Progesterone-related progestins are further divided into pregnanes (e.g., medroxyprogesterone acetate [MPA]) and 19-norpregnanes (e.g., nomegestrol acetate, segesterone acetate/nestorone), whereas testosterone-related progestins are further divided into estranes (e.g., dienogest, etynodiol diacetate, norethindrone, norethindrone acetate, norethindrone enanthate) and gonanes (e.g., desogestrel, levonorgestrel, norgestimate, norgestrel). Drospirenone is a newer progestin, derived from spironolactone, that more closely reflects the biological activity of endogenous progesterone than other progestins (Krattenmacher, 2000). Even within the same class, the biological activity of individual progestins can differ widely. There are several domains that may contribute to unique consequences of HCs on immune function and compensatory prophylactic behaviors, including (1) the route of administration, which influences bioavailability and serum concentration dynamics; (2) the pharmacodynamics of the progestin, involving its receptor binding affinities and metabolic pathways, (3) the temporal dynamics of OC phases, corresponding to fluctuations in progestin between active pill and reminder pill phases, and (4) the dose of the progestin, which determines the magnitude and duration of physiological effects.

Route of Administration

The concentration of a drug that is available to induce action within the tissues

(i.e., bioavailability) is determined by its route of administration and pharmacokinetics. Pharmacokinetics refers to the influence of the body's physiological processes on a drug, including its absorption, distribution, metabolism, and elimination. OCs are the most popular form of HC in the United States (Daniels & Abma, 2020). The progestin-only pill (i.e., the "mini pill") supplies synthetic progestin, but, unlike other OCs, does not reliably inhibit ovulation due to the low dosage (Duijkers et al., 2022). Combined OCs contain progestin and ethinyl estradiol (or the recently available synthetic estrogens estetrol or estradiol valerate) and generally contain reminder pills (i.e., nonmedicated "sugar" or "placebo" pills or pills containing iron) at the end of the pack.

OCs, like all orally administered pharmaceuticals, undergo first-pass metabolism in the liver – significantly reducing the concentration of the medication – before being absorbed into circulation and distributed to tissues (reviewed in Stanczyk et al., 2013). Most orally administered progestins reach their maximally available concentration within 1-3 hours and then steadily decrease in concentration at a rate dependent upon their half-life (i.e., the time required for the body to clear 50% of the maximum serum concentration), which is generally around 20 hours (reviewed in Bick et al., 2021). As such, OCs require daily administration to maintain their efficacy, resulting in serum concentration spikes every 24 hours for compliant users. OCs cause dynamic shifts in serum concentration and availability unlike that of other contraceptive administrative routes (e.g., intrauterine devices, injectables), which take longer to reach maximum concentration, have longer half-lives, and maintain more stable – albeit steadily decreasing – concentrations until eliminated (reviewed in Bick et al., 2021). Meaningful differences in progestin bioavailability between routes of administration necessitates

considering OC users separately from users of other non-oral forms of hormonal contraception.

Progestin Pharmacodynamics

While pharmacokinetics considers the effects of the body on a medication (i.e., absorption, distribution, metabolism, and excretion), pharmacodynamics refers to the effects of the medication on the body. Pharmacodynamics investigates the mechanisms of action of a medication, including its receptor binding, potency (i.e., the dose needed for a given effect), and efficacy (i.e., the maximum effect possible), to identify the optimal dose for achieving a desired clinical effect. The contraceptive efficacy of progestins is mediated primarily through activity at the progesterone receptor (reviewed in Africander et al., 2011). Progesterone receptors are distributed on cells throughout the nervous system, including immune cells (reviewed in Dressing et al., 2011). However, progestins bind to the progesterone receptor to varying degrees. For example, compared to the affinity of endogenous progesterone for the progesterone receptor, norethindrone binds 130% more strongly and levonorgestrel binds over 300% more strongly, whereas drospirenone binds only 20% as strongly (Philibert et al., 1999).

Notably, the actions of progestins are not limited to the progesterone receptor. Many progestins bind to other steroid receptors to varying degrees, including the androgen receptor, glucocorticoid receptor, and mineralocorticoid receptor (Louw-du Toit et al., 2017), contributing to many of the undesirable side effects of HC use (e.g., weight gain, bone density loss; reviewed in Africander et al., 2011). Progestins are subclassified into four generations, determined by when they were introduced into the market, which differ in their activity on other steroid receptors. First, second, and third

generation progestins (e.g., MPA, norethindrone, levonorgestrel, norgestimate, desogestrel) are androgenic progestins due to their agonistic activity at the androgen receptor, whereas fourth generation progestins (e.g., dienogest, drospirenone) have potent antiandrogenic properties (reviewed in Stanczyk et al., 2013).

Investigations of pharmacodynamics are contributing to an improved understanding of the progestin-specific side effects of HCs (e.g., Gravelsins et al., 2023; Grøntvedt et al., 2017; reviewed in Mitchell & Welling, 2020). However, limited research has evaluated how these factors influence the BIS. One study (Menting-Henry et al., 2022) considered the androgenicity of progestins in its sample, but found no differences in identification of disgust expressions between users of androgenic progestins, users of anti-androgenic progestins, or NC women. This study did not report other unique qualities or effects of the OCs (e.g., dose). At this time, no other studies have investigated how the unique pharmacodynamic properties of progestins influence BIS reactivity.

Temporal Dynamics of Oral Contraceptive Phases

OCs are commonly taken in two phases shifting between pills containing synthetic hormones and pills that contain no synthetic hormones. Most progestin-only pills (e.g., Errin, Nora-Be) contain 28 days of active pills without a reminder pill phase, but are also available with 24 active tablets followed by 4 reminder pills (i.e., Slynd). Most combined OCs contain 21 days of active pills followed by 7 days of reminder pills, but they are also available with 24 active and 4 reminder pills (e.g., Loestrin 24 Fe, Yaz), 21 active pills but no reminder pills (e.g., Junel 21), or in 91-day formulations with 84 days of active pills followed by 7 days of reminder pills (e.g., Seasonale) or low-dose

estradiol pills (Seasonique). Women in their active pill phase have relatively low amounts of estradiol and artificially inflated levels of progestin that can, in most combined OC formulations, approximate the effects of endogenous progesterone during the mid-luteal phase (Grøntvedt et al., 2017; Odell & Molitch, 1974). OC users who take reminder pills (or who skip the reminder pills, but still wait 4-7 days before beginning active pills) consequently shift between days of having high doses of synthetic progestin and days without synthetic progestin. Given progestin's immunomodulatory effects, this shift could result in distinct manifestations of the BIS that differ between these phases. Interestingly, when combined OC users were grouped by pill phase into those currently using their active pill versus those currently using their reminder (or off pill), women taking their active pill showed more accuracy in an affective responsiveness task (i.e., selecting the appropriate emotional facial expression to match a provided emotion-inducing scenario) when assessing happy, sad, angry, fearful, disgusted, and neutral faces (Radke & Derntl, 2016). However, this study did not report whether there were phase-related differences in recognition accuracy for disgust relative to other emotions individually.

Dose and Relative Potency

OCs are also available in formulations at varying doses. Progestin-only pills are monophasic, meaning the active pills all supply the same dose of synthetic hormones throughout the pack. Combined OC formulations can be monophasic, biphasic (i.e., active pills that supply two phases with different doses of synthetic hormones), triphasic (i.e., active pills supply three phases with different doses of synthetic hormones), or quadriphasic (i.e., active pills supply four different phases of synthetic hormones).

Multiphasic OCs generally increase in progestin throughout the pill pack, although some brands (e.g., Natazia) adjust the dose of synthetic estradiol between phases. Moreover, OC brands often produce several formulations that differ somewhat in progestin dosage. For example, drospirenone is supplied at 3 mg per day in combined OCs but at 4 mg per day in progestin-only pills. Norgestimate, on the other hand, is generally supplied at 250 mcg in monophasic combined OCs, but is supplied as low as 180 mcg during phase one of triphasic OCs. Progestins prescribed at higher doses induce greater physiological effects than the same progestin at a lower dose. Similarly, it is important to consider whether the dose of progestin contained in women's OC pills has psychobehavioral influences.

Pharmacokinetics and pharmacodynamics determine the clinical effect of a medication at a certain dose. However, HCs are prescribed in a range of doses, even for the same progestin, which can lead to different biological outcomes. To better capture these variations, researchers studying the psychological effects of oral contraceptives have recently adopted a coding system that quantifies the relative activity of each formulation at the progestogenic, androgenic, and estrogenic receptors (e.g., Hampson et al., 2022; see relative potency table in Dickey & Seymour, 2021). These activity values are derived from *in vivo* bioassays and receptor-binding studies in both humans and laboratory animals (Chihal et al., 1975). This methodology considers both pharmacokinetic factors (e.g., metabolism, half-life) and pharmacodynamic properties (e.g., receptor binding, synergistic or antagonistic effects between progestins and synthetic estradiol), enabling meaningful comparisons across formulations with different hormonal compositions or dosages. It also enables comparisons between monophasic and

multiphasic formulations because activity values reflect the compounding hormonal activity across a full 28-day span of the OC formulation. As such, hormone activity values reflect each formulation's bioactivity at the relevant hormone receptors. Importantly, this methodology forgoes grouping users by administration route, androgenicity, or even progestin type, and instead allows researchers to assess activity-dependent effects of progestin in a linear fashion. This approach is especially crucial for accurately evaluating the effects of progestins in samples that include low-dose or multiphasic formulations.

The Current Study

This study investigates the influence of synthetic progestins on the behavioral immune response by examining how OC users respond to pathogen-related cues. Participants using OCs completed a pathogen disgust manipulation during either their active or reminder pill phase, followed by a series of questionnaires assessing BIS reactivity through state disgust and pathogen avoidance motivations. To test for linear associations between progestogenic activity and BIS outcomes, each participant's OC formulation was coded for its progestogenic activity, which reflects its cumulative receptor activity across the 28-day formulation.

It was hypothesized that participants exposed to the pathogen-salient experimental condition would report greater state disgust and stronger pathogen avoidance motivations than those in the pathogen-free control condition. Among those in the experimental condition, individuals in their active pill phase were expected to exhibit greater BIS reactivity compared to those in the reminder pill phase. Progestogenic activity was hypothesized to influence this relationship, such that participants using more

progestogenic formulations were expected to show stronger disgust responses and pathogen avoidance motivations than those using less progestogenic formulations. These relationships were expected to be influenced by participants' perceptions of their own disease vulnerability. Specifically, participants who viewed themselves as more vulnerable to disease were expected to exhibit the highest levels of disgust and pathogen avoidance motivations, especially in the experimental condition. Conversely, participants in the control condition were expected to exhibit the lowest BIS reactivity, regardless of progestogenic activity.

In all, this study aimed to clarify whether the strength of individual vulnerabilities, determined by psychological (perceived vulnerability) and immunological (progestogenic activity) traits, contribute to variation in BIS reactivity, operationalized by emotional (state disgust) and motivational (pathogen avoidance) measures, under ecologically threatening conditions.

Method

Design

This study employed a between-subjects experimental design. Participants were randomly assigned to either a pathogen-salient or pathogen-free condition using the Culpepper Disgust Image Set (C-DIS; Culpepper et al., 2018). OC users were categorized by pill phase (active, reminder), and each OC formulation was coded continuously for progestogenic activity. Perceptions of disease vulnerability were examined as an individual difference moderator of state BIS responses. The primary dependent variables were state disgust and pathogen avoidance motivations.

Participants

Cisgender women (i.e., women who were assigned female at birth) who were at least 18 years old (M = 19.96, SD = 3.08) and currently using an OC pill (N = 341) were recruited from Oakland University's Psychology Subject Pool (SONA). Participants were currently taking active pills (i.e., the first 21-24 pills that contain synthetic hormones to inhibit ovulation; n = 314) or reminder pills (i.e., the hormone-free pills at the end of the pill pack that coincide with menstruation; n = 27). Participants using reminder pills had taken *at least* 2 complete days of reminder pills to allow time for synthetic hormones to be metabolized and cleared from the body.

Prior to analyses, 33 participants who were in their active pill phase were excluded for not completing the BIS reactivity measures (n = 20), having no variability in responses across measures (n = 11), or for currently breastfeeding (n = 2). This left 308 participants for analyses (281 active pill phase, 27 reminder pill phase). Participants were not pregnant or breastfeeding and did not use another form of hormonal contraception in addition to their OC pill. Participants were predominantly White (81.5%; 9.1% Black, 4.9% multiracial, 3.9% "other", .6% preferred not to answer), heterosexual (83.8%; 12% bisexual, 2.3% homosexual, 1.9% "other"), and in an exclusive romantic relationship (59.4%; single 39.9%, nonmonogamous .6%).

Materials

Prescreener and Demographic Questionnaires

Participants first completed questions used for prescreening purposes (i.e., sex assigned at birth, current HC use, current pill phase) before providing informed consent and beginning the study. Participants provided demographic information (i.e., age, sexual orientation, relationship status, ethnicity), and information about their hormonal history

(i.e., their menstrual history, current use of hormone replacement therapy, pregnancy status, whether they have given birth in the last year, and whether they are currently breastfeeding).

Oral Contraceptive Questionnaire

OC information was provided using a drop-down menu containing the brand names of OCs available as of May 14, 2023. Participants whose OC was not listed on the first drop-down menu were taken to a second drop-down menu listing the names of generic OC formulations. Those using an OC that was not included in either drop-down menu were instructed to write-in the medication information listed on their OC pill pack (i.e., progestin type and strength, synthetic estrogen type and strength). They were asked which day of their pill pack they were currently on (to determine OC phase), how many hours it had been since they took their most recent pill, and the length of time they had been using their current brand of OC in years and months.

Perceived Vulnerability to Disease Questionnaire

The Perceived Vulnerability to Disease Questionnaire (PVD; Duncan et al., 2009) assesses trait disgust sensitivity with 15 items (α = .808) that are rated on a 7-point scale from 1 (Strongly disagree) to 7 (Strongly agree). The PVD contains two subscales that measure self-perceived susceptibility to infectious disease (i.e., Perceived Infectability [PI]; e.g., “If an illness is ‘going around’, I will get it.”; α = .892) and general discomfort in the presence of pathogenic material (i.e., Germ Aversion; e.g., “I prefer to wash my hands pretty soon after shaking someone’s hand”; α = .685). The convergent and discriminant validity of the PVD is well-established (see Duncan et al., 2009) and it has strong test-retest reliability (r = 0.954; Díaz et al., 2016). The PI subscale was used as a

trait measure of BIS sensitivity to assess how dispositional perceptions of infection vulnerability influence situational reactivity to pathogen salience.

Disgust Propensity and Sensitivity Scale–Revised

The Disgust Propensity and Sensitivity Scale – Revised (DPSS-R; Fergus & Valentiner, 2009; van Overveld et al., 2006) contains 12-items that assess two factors of trait disgust: disgust propensity (i.e., one's general tendency to experience disgust) and disgust sensitivity (i.e., how intense the disgust is if it is experienced). Disgust Propensity (α = .836) is assessed with statements such as "I experience disgust" and "I feel repulsed", and Disgust Sensitivity (α = .784) is assessed with statements such as "I think feeling disgust is bad for me" and "When I feel disgusted, I worry that I might pass out." Participants' agreement with each statement was rated on a 5-point scale from 1 ("Never") to 5 ("Always"). The convergent and divergent validity of the DPSS-R has been established; the measure is strongly correlated with measures of contamination fear (e.g., Padua Inventory – Contamination Obsessions and Washing Compulsions subscale; Burns et al., 1996), is more strongly correlated with disgust-related phobias than fear-related phobias (Fergus & Valentiner, 2009), and is not correlated with reports of injection avoidance (Olatunji et al., 2007a). The DPSS-R also has strong predictive validity and participants with high scores on the DPSS-R are less likely to perform disgust-eliciting behaviors (e.g., drinking from a dog's water bowl, touching a band-aid with a red spot; van Overveld et al., 2010). The DPSS-R was used to test for pre-existing differences in participants' trait disgust sensitivity before activation of the BIS.

Disgust Scale–Revised

The Disgust Scale – Revised (DS-R; Haidt et al., 1994; modified by Olatunji et

al., 2007b) assesses trait disgust propensity with 27-items (α = .860; not including 2 distractor items) rated on a 5-point scale from 0 (Strongly disagree) to 4 (Strongly agree). The DS-R can be organized into three subscales that measure core disgust (i.e., disgust related to aversive stimuli, such as rotting meat entering the mouth), animal-reminder disgust (i.e., disgust caused by stimuli reminding one of their animal nature), and contamination disgust (i.e., fear and disgust towards stimuli that may have contagions). Core disgust (α = .737) is assessed by 12 items and contains statements such as, "Even if I was hungry, I would not drink a bowl of my favorite soup if it had been stirred by a used but thoroughly washed flyswatter" and "If I see someone vomit, it makes me sick to my stomach." Animal-reminder disgust (α = .787) is assessed by 8 items such as, "It would bother me to be in a science class, and to see a human hand preserved in a jar" and "It would bother me tremendously to touch a dead body." Contamination disgust (α = .575) is assessed by 5 items and includes statements such as, "I never let any part of my body touch the toilet seat in public restrooms" and "I probably would not go to my favorite restaurant if I found out that the cook had a cold." To maintain reliability, subscales with poor internal consistency were not analyzed individually. The DS-R has good construct validity, as participants with obsessive compulsive disorder (OCD) score higher than non-anxious participants on the total scale, and participants with OCD that have washing concerns score higher than those without washing concerns on the Core Disgust and Contamination Disgust subscales (Olatunji et al., 2007b). The DS-R also has good convergent validity and is correlated with validated measures of disgust (e.g., Disgust Emotion Scale; Kleinknecht et al., 1997) and contamination fear (e.g., Padua Inventory – Contamination Obsessions and Washing Compulsions subscale; Burns et al.,

1996), even after controlling for state anxiety (Olatunji et al., 2007b). Because individuals with higher scores on the PVD tend to report higher levels of disgust sensitivity (e.g., Stevenson et al., 2021), the DS-R was used as a covariate when assessing trait-state models of BIS reactivity. Given its stronger internal consistency relative to the subscales, the DS-R total score was retained for analysis.

Culpepper Disgust Image Set

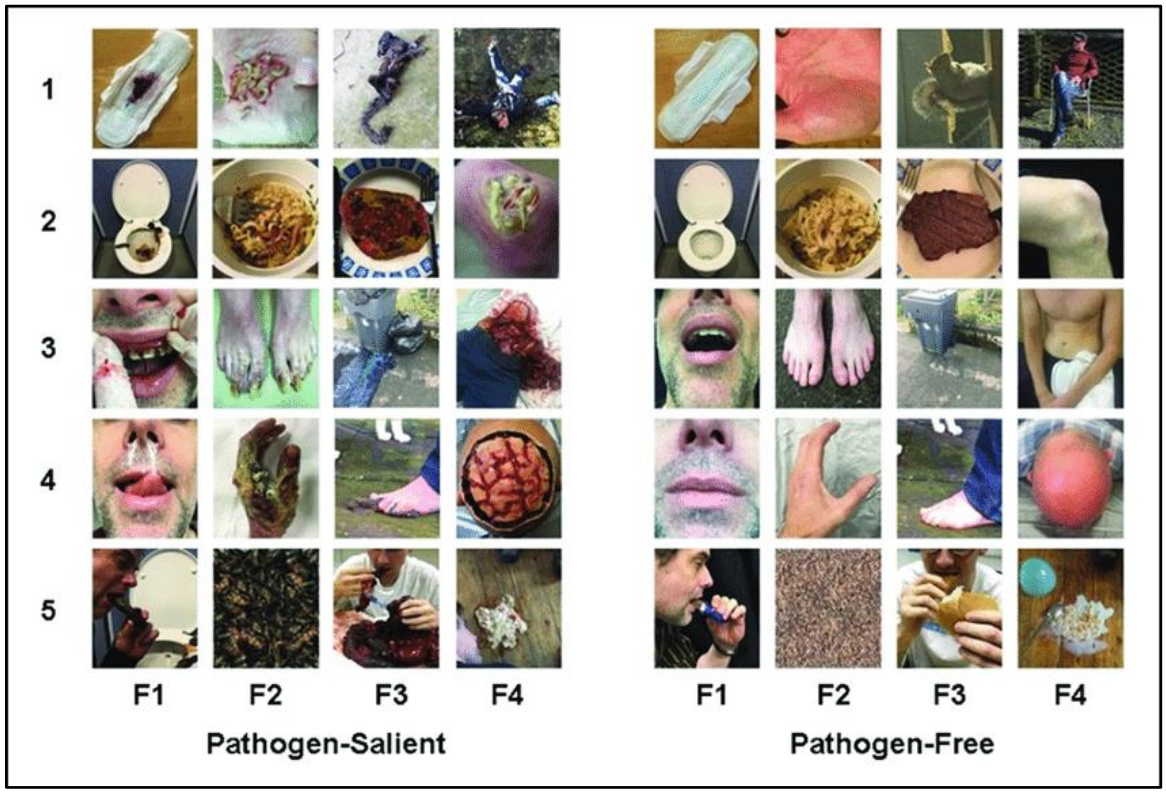
The Culpepper Disgust Image Set (C-DIS; Culpepper et al., 2018) was used as the pathogen disgust manipulation task. Participants were randomly assigned to rate images of either (1) pathogen-salient stimuli or (2) matched pathogen-free stimuli. Each set is comprised of 20 images that were generated to either prime pathogen disgust (i.e., pathogen-salient stimuli; α = .938) or to not prime pathogen disgust (i.e., pathogen-free stimuli; α = .914). Images within the randomly assigned set were presented to participants on separate pages in a random order and participants were instructed to rate each image for disgust on a 7-point scale (anchors: 0 = "not disgusting at all", 6 = "extremely disgusting"). The image sets are paired such that each pathogen-salient image has a closely matched control image that is relatively free from pathogen cues (e.g., an image of a festering leg wound is matched with an image of an uninjured leg; see Figure 2.1).

Disgust ratings for the pathogen-salient image set (M = 4.53, SD = 1.11) are significantly higher than the pathogen-free image set (M = 1.67, SD = 1.01; $t(306) = -23.66$, $p < .001$), demonstrating that the pathogen-salient stimuli effectively activate disgust mechanisms, whereas the pathogen-free stimuli do not. The C-DIS pathogen-salient image set is strongly positively correlated with other stimuli used to induce disgust (e.g., Curtis images; Curtis et al., 2004), and the difference in scores between the

pathogen-salient and pathogen-free image sets in both the C-DIS and Curtis images are positively correlated, demonstrating good convergent validity. Importantly, the difference scores between the pathogen-salient and pathogen-free stimuli are significantly larger for the C-DIS compared to the Curtis images, suggesting the C-DIS is a more effective task for manipulating disgust (Culpepper et al., 2018).

Figure 2.1

Culpepper Disgust Image Set Stimuli.



Note: Pathogen-salient images (left) were each paired with a relatively pathogen-free control image (right).

Behavioral Immune System Reactivity

BIS reactivity was assessed in two ways: (1) a single-item question, "How disgusted do you feel right now?", rated on a 0-100 sliding scale, and (2) the Situational Pathogen Avoidance (SPA) scale (Makhanova et al., 2021). The single-item question assesses the emotional component of pathogen avoidance, whereas SPA assesses the cognitive and motivational mechanisms of pathogen avoidance to social situations where pathogen transmission is likely. Both measures capture BIS reactivity to situational pathogen cues. The SPA contains 10 items (α =.863) that are rated on a 7-point scale (anchors: 1 = Strongly disagree, 7 = Strongly agree), including "Right now, it would make me uncomfortable to touch a door handle in a public restroom" and "Right now, I would be grossed out if I shook a stranger's hand." Participants responded to the statements with their attitude *right now at this moment* to assess their *current* state of pathogen avoidance and BIS reactivity. The convergent and discriminant validity of the SPA has been established and it is positively correlated with other validated measures of trait pathogen avoidance (e.g., Germ Aversion and PI subscales of the PVD; Duncan et al., 2009), whereas it is not correlated with measures that are not theoretically related to pathogen avoidance (e.g., social desirability; see Makhanova et al., 2021). The SPA also has good predictive validity and is predictive of negative reactions toward an obese target, which is consistent with previous research (e.g., Miller & Maner, 2011).

Progestogenic Activity of OCs

Because of the pharmacokinetic (e.g., metabolism, half-life) and pharmacodynamic differences between OC formulations, they induce different levels of activity at the progesterone receptor. Each OC formulation was assigned a progestogenic

activity value reflecting its average pharmacodynamic activity at the progesterone receptor (see also Hampson et al., 2022). Pharmacodynamic activity values were derived from *in vivo* bioassay and receptor-binding studies and are compiled in Dickey and Seymour (2021). Progestogenic activity reflects not only the pharmacodynamic activity and dose of the steroids within the formulation, but also any amplified or antagonistic effects. In other words, progestogenic activity represents the combined biological effect of progestin (and estradiol, if present) across a complete 28-day cycle. These values are formulation-specific, allowing for between-formulation comparisons of receptor-specific activity. The seven most common formulations account for nearly 70% of OC use in this sample, with Loestrin 1/20 being the most common (see Table 2.1 for progestogenic activity values for the most commonly prescribed OC brands in this sample). A range of phasic types, progestin types, and progestogenic activities are represented.

Procedures

All procedures were completed using Qualtrics, an online survey host platform, and participants were compensated with course credit for completing the study. Participants were pre-screened to ensure they met eligibility requirements. Those who consented provided information about their demographics and their OC use, and then completed 3 questionnaires, in a random order, to assess trait disgust sensitivity and PI: the DPSS-R (van Overveld et al., 2010), DS-R (Haidt et al., 1994; modified by Olatunji et al., 2007b), and PVD (Duncan et al., 2009). Next, participants completed the C-DIS pathogen disgust manipulation and were randomly assigned to rate images of either (1) pathogen-salient stimuli or (2) matched pathogen-free stimuli for disgust. After the manipulation, participants completed two measures to assess BIS reactivity: (1) a single-

Table 2.1

Most Common Oral Contraceptives in the Sample (≥ 5% of Participants) by Brand, Formulation, and Progestogenic Activity.

Brand	*n*	%	Formulation details	Progestogenic activity
Loestrin 1/20	58	19	21 active (1 mg NEA / 20 µg EE), 7 reminder (inert)	1.2
Lo Loestrin FE	36	11.8	24 active (1 mg NEA / 10 µg EE), 2 estrogen-only (10 µg EE), 2 reminder (75 mg FE)	1.4
Micronor	25	8.2	28 active (0.35 mg NE)	0.4
Loestrin 1.5/30	22	7.2	21 active (1.5 mg NEA / 30 µg EE), 7 reminder (inert)	1.7
Sprintec	20	6.6	21 active (0.25 mg NG / 35 µg EE), 7 reminder (inert)	0.4
Tri-Norinyl 28	19	6.2	7 active (0.5 mg NE / 35 µg EE), 7 active (1 mg NE / 35 µg EE), 7 active (0.5 mg NE / 35 µg EE) 7 reminder (inert)	0.7
Loestrin 24 FE	18	5.9	24 active (1 mg NEA / 20 µg EE) 4 reminder (75 mg FE)	1.4
Alesse	15	4.9	21 active (0.10 mg LNG / 20 µg EE) 7 reminder (inert)	0.5
Remaining brands	95	30.2		

Note: OC = oral contraceptive; EE = ethinyl estradiol; FE = ferrous fumarate; LNG = levonorgestrel; NE = norethindrone; NEA = norethindrone acetate; NG = norgestimate. Dosages are given as mg progestin/µg EE per tablet unless otherwise indicated. “FE” brands include ferrous fumarate in the nonhormonal reminder tablets. Dosages are given as mg progestin/µg EE per active tablet. Progestogenic activity values reflect a relative potency (higher values indicate greater progestogenic activity) derived from Dickey and Seymour (2021).

item question, “How disgusted do you feel right now?”, rated on a 0-100 sliding scale, and (2) the Situational Pathogen Avoidance scale (Makhanova et al., 2021).

Results

Analyses were conducted in SPSS (Version 31), with statistical significance set at $\alpha = .05$. All variables were screened for statistical assumptions (e.g., normality, homogeneity of variance) and met criteria prior to analyses. The planned analyses were designed to evaluate whether the experimental manipulation effectively elicited pathogen-based BIS reactivity, both emotionally (via state disgust) and motivationally (via SPA), and to test whether individual differences in trait vulnerability (PI) and hormonal modulation (progestogenic activity of OCs) predicted these BIS responses across participants. Although the planned analyses explored the basic effects of the manipulation and trait/hormonal predictors when collapsed across conditions, they did not test whether these same predictors have unique effects depending on experimental context. It is likely that the influence of PI and hormonal activity may vary based on exposure to pathogen cues. To further explore these relationships, a series of mediation, moderation, and moderated mediation models were conducted. The findings at each step inform the development of the subsequent model.

A power analysis conducted in G*Power (Version 3.1.9.7) indicated that a minimum total sample of $N = 128$ ($n = 64$ per subgroup) was needed to detect a medium effect (Cohen’s $d = .50$) for the planned group comparisons. The present sample ($N = 308$) exceeds this requirement for experimental conditions, indicating adequate power for experimental (i.e., manipulation-based) hypotheses. However, phase-based comparisons were limited by the small reminder OC phase subgroup ($n = 27$), and several exploratory

interaction models involved small cells (minimum $n = 17$). Sensitivity analyses indicated these subgroups were not powered to detect large effects. As such, nonsignificant findings in those models should be interpreted cautiously, as limited sensitivity may obscure smaller but potentially meaningful effects.

All figures were created using the Scientific Colour Map *batlow* (Crameri, 2018) to ensure accessibility for readers with color-vision deficiencies (Crameri et al., 2020).

Descriptive Statistics and Preliminary Analyses

Table 2.2 presents descriptive statistics for demographic variables, OC-related characteristics, baseline BIS traits, and BIS reactivity variables by OC phase (active, reminder) and manipulation condition (experimental, control). No statistically significant group differences were found, with the exception of state disgust, which varied systematically by experimental condition as expected.

Manipulation Check

To evaluate the basic effectiveness of the manipulation, two independent-samples *t*-tests compared participants in the experimental and control conditions on state disgust and SPA. As expected, participants exposed to the pathogen-salient manipulation reported significantly greater state disgust ($M = 75.56$, $SD = 25.36$) compared to those in the control group ($M = 24.98$, $SD = 23.54$; $t(306) = -18.14$, $p < .001$). However, SPA did not differ between these groups ($t(306) = .542$, $p = .542$; $M_{control} = 4.71$, $SD_{control} = 1.01$; $M_{experimental} = 4.62$, $SD_{experimental} = 1.21$; see Figure 2.2), suggesting the manipulation increased emotional reactivity, but did not directly increase pathogen avoidance motivations.

Table 2.2

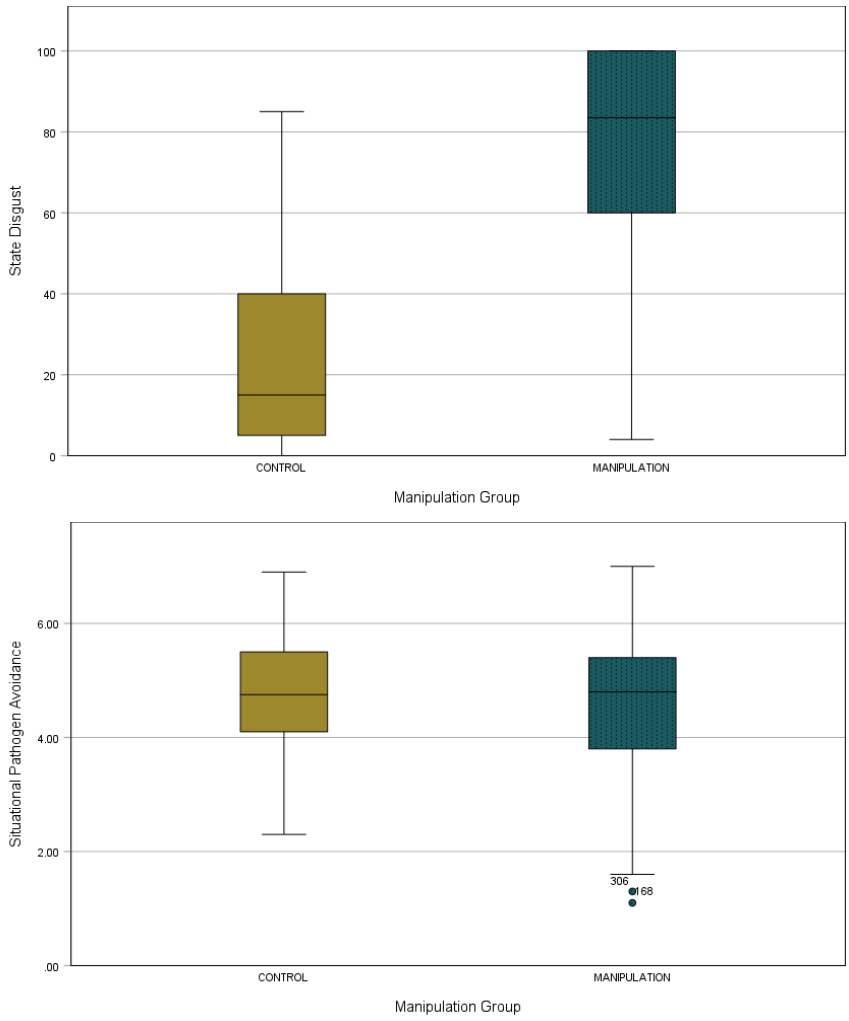
Participant Characteristics by Oral Contraceptive Phase and Experimental Condition.

Variable	Total (*N* = 308)	Active (*n* = 281)	Reminder (*n* = 27)	Control (*n* = 154)	Experimental (*n* = 154)
Demographic Variables, *M* (*SD*) or *n* (%)					
Age (years)	19.96 (3.08)	20.05 (3.19)	18.92 (.10)	19.79 (2.79)	20.13 (3.35)
Sexual orientation					
Heterosexual	258 (83.8)	235 (83.6)	23 (85.2)	133 (86.4)	125 (81.2)
Homosexual	7 (2.3)	7 (2.5)	0 (0.0)	2 (1.3)	5 (3.2)
Bisexual	37 (12.0)	34 (12.1)	3 (11.1)	16 (10.4)	21 (13.6)
Other	6 (1.9)	5 (1.8)	1 (3.7)	3 (1.9)	3 (1.9)
Relationship status					
Exclusive relationship	183 (59.4)	166 (59.1)	17 (63)	100 (64.9)	83 (53.8)
Single	123 (39.9)	114 (40.6)	9 (33.3)	54 (35.1)	69 (44.8)
Nonmonogamous	2 (.6)	1 (.4)	1 (3.7)	0 (0.0)	2 (1.3)
Race					
White	251 (81.5)	230 (81.9)	21 (77.8)	124 (80.5)	127 (82.5)
Black	28 (9.1)	26 (9.3)	2 (7.4)	15 (9.7)	13 (8.4)
Multiracial	15 (4.9)	14 (5)	1 (3.7)	6 (3.8)	9 (5.7)
Other race	12 (3.9)	9 (3.4)	3 (11.1)	8 (5.1)	4 (2.6)
Prefer not to say	2 (.6)	2 (.7)	0 (0.0)	1 (0.6)	1 (0.6)
Oral contraceptive characteristics, *M* (*SD*)					
Duration of OC use (months)	25.72 (19.38)	26.65 (18.84)	20.43 (21.77)	25.64 (21.65)	25.80 (17.59)
Progestogenic activity	1.00 (.48)	1.00 (.49)	1.04 (.48)	1.04 (.47)	.95 (.49)
Behavioral immune system traits, *M* (*SD*)					
PI	3.79 (1.28)	3.77 (1.29)	3.92 (1.19)	3.74 (1.28)	3.84 (1.27)
Disgust propensity	19.36 (4.09)	19.29 (4.14)	20.10 (3.55)	19.19 (4.15)	19.54 (4.04)
Disgust sensitivity	16.88 (4.80)	16.84 (4.84)	17.28 (4.37)	16.73 (4.89)	17.03 (4.70)
DS-R	2.49 (.59)	2.47 (.59)	2.68 (.53)	2.48 (.58)	2.49 (.60)
Dependent variables, *M* (*SD*)					
State disgust	50.27 (35.19)	49.36 (35.03)	59.78 (36.10)	24.98 (23.54)	75.56 (25.36)
SPA	4.68 (1.11)	4.67 (1.11)	4.60 (1.19)	4.70 (1.01)	4.63 (1.21)

Note: Values are presented as *M* (*SD*) or *n* (%). Group differences between OC phase and experimental condition were evaluated using chi-square tests of independence or independent-samples *t*- tests. Abbreviations: OC = oral contraceptive, PI = perceived infectability, DS-R = Disgust Scale-Revised, SPA = situational pathogen avoidance.

Figure 2.2

State Disgust and Situational Pathogen Avoidance by Experimental Condition



Note. Top panel displays state disgust scores and bottom panel displays situational pathogen avoidance scores by condition. Bars reflect mean values; error bars reflect standard error.

Hormonal Associations with BIS Reactivity

To examine hormonal contributions to BIS reactivity, independent-samples *t*-tests compared women in the experimental condition who were using either their active or

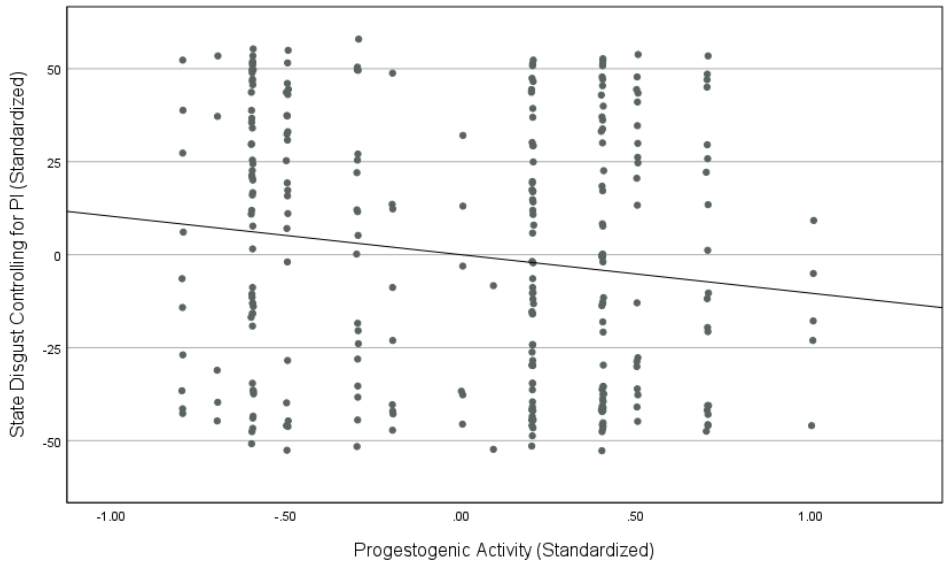
reminder pill. There were no differences between phases in state disgust ($t(152) = .88$, $p = .382$; $M_{active} = 74.93$, $SD_{active} = 25.54$; $M_{reminder} = 80.65$, $SD_{reminder} = 23.98$) or SPA ($t(152) = -0.124$, $p = .902$; $M_{active} = 4.63$, $SD_{active} = 1.20$; $M_{reminder} = 4.59$, $SD_{reminder} = 1.37$).

Two multiple regressions tested whether PI and the progestogenic activity of participants' OCs predicted BIS reactivity across all conditions. Because these models investigate the progestogenic activity of an OC as a function of its receptor activity, these models are restricted to participants in their active pill phase only who have circulating progestin. In the first model predicting state disgust, PI and progestogenic activity accounted for a small but significant proportion of the variance ($R^2 = .03$, $F(2, 275) = 4.25$, $p = .015$). Although PI did not significantly predict state disgust ($p = .103$), progestogenic activity was *negatively* associated with disgust ($\beta = -.14$, $t = -2.41$, $p = .016$), contrary to predictions (see Figure 2.3). This suggests that higher progestogenic activity in OCs may be linked to reduced emotional BIS sensitivity.

In the second model predicting SPA, PI and progestogenic activity did not jointly account for significant variance ($R^2 = .01$, $F(2, 275) = 1.34$, $p = .265$). Neither PI ($p = .136$) nor progestogenic activity ($p = .507$) significantly contributed to SPA. Thus, neither trait PI nor progestogenic activity independently predicted motivational BIS responses in this model, suggesting that any role for these factors in SPA may depend on interactions with other variables or specific contexts, rather than by exerting direct effects.

Figure 2.3

Partial Regression Plot Depicting the Association Between Progestogenic Activity and State Disgust



Note. Values represent the association between progestogenic activity and state disgust after controlling for PI. The line reflects the fitted regression slope.

Abbreviations: PI = perceived infectability

Exploratory Analyses

Because the experimental manipulation was designed to elicit pathogen-relevant emotional (state disgust) and motivational (SPA) responses, follow-up moderated regression analyses were conducted to test whether the effects of trait PI and progestogenic activity differed between experimental conditions. Before examining potential interaction effects, participant distributions across all predictor combinations were checked to ensure adequate cell sizes (all subgroups $\geq n = 17$). Interaction effects for this analysis were probed using PROCESS for SPSS (Hayes, 2022). However, due to

small subgroup sample sizes and potential corresponding Type II error, interaction effects should be interpreted with caution.

Additional exploratory analyses estimated mediation and moderated mediation models via bootstrapping using PROCESS for SPSS (Hayes, 2022) with 5,000 bias-corrected percentile bootstrap resamples. First, the experimental manipulation was examined in a mediation model (PROCESS Model 4) to evaluate whether state disgust mediated its effect on SPA. Based on those results, a moderated mediation model (PROCESS Model 72) was then tested to assess whether the indirect effect was moderated by PI and progestogenic activity, providing a direct test of the CPH.

For all mediation and moderated mediation models, PROCESS estimated total (*c*), direct (*c'*), and indirect (*ab*) effects, as well as interaction terms, using heteroskedasticity-consistent (HC4) *F*-values and standard errors. 95% Confidence intervals (CIs) were considered statistically significant if they did not include zero. A significant indirect effect supports the presence of a mediation effect. Following Hayes' (2022) recommendations, predictors were entered uncentered and PROCESS defaults were used to mean-center continuous and categorical predictors involved in interaction terms. Unstandardized regression coefficients and standard errors are reported for models conducted using PROCESS, with pathway nomenclature (e.g., *a, c'*) used to indicate each effect: *a* (indirect effect of X on M), *b* (indirect effect of M on Y), *ab* (total indirect effect of the mediation), *c* (total effect of X on Y without considering M), and *c'* (direct effect of X on Y when controlling for M).

Conditional Effects of PI and Progestogenic Activity on BIS Reactivity

Moderated regressions examined whether PI, progestogenic activity, and

manipulation condition jointly predicted state disgust and SPA among OC users. Analyses indicated a comparable three-way interaction pattern for both state disgust and SPA.

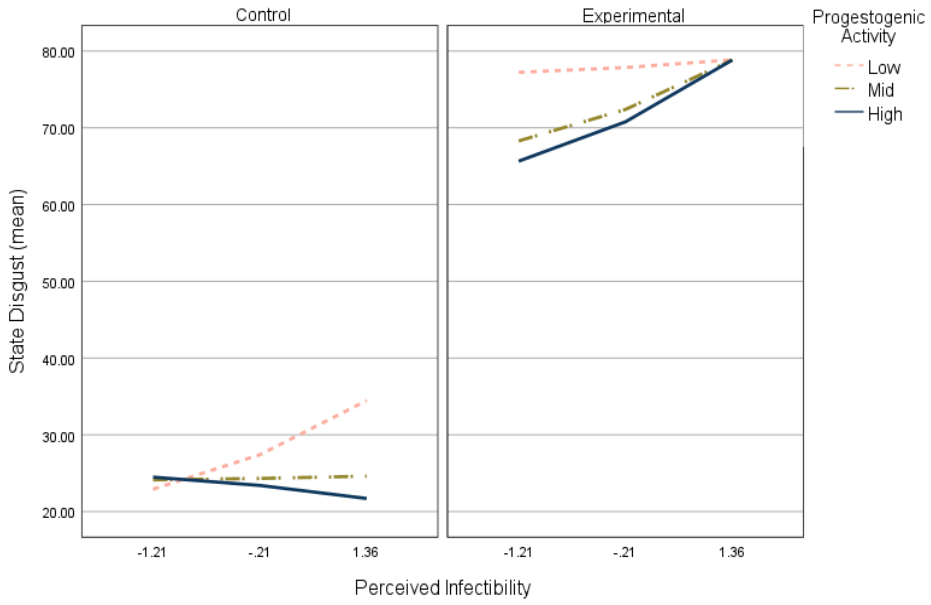
In predicting state disgust, the three main effects accounted for a significant proportion of variance (R^2 = .52, F(3, 274) = 98.63, p < .001). As expected, the manipulation robustly increased state disgust (β = .70, t = 16.70, p < .001). Neither PI (β = .07, t = 1.78, p = .077) nor progestogenic activity (β = -.07, t = -1.63, p = .105) were significant independent predictors. Adding the two-way interactions did not improve model fit (ΔR^2 = .002, ΔF = .34, p = .799), although the model itself remained significant (F(6, 271) = 49.12, p < .001). Neither the condition × PI (β = .04, t = .90, p = .371), condition × progestogenic activity (β = -0.01, t = -0.16, p = .874), nor PI × progestogenic activity (β = -0.02, t = -0.35, p = .730) interactions were significant. Adding the three-way interaction significantly improved model fit (ΔR^2 = .01, ΔF = 4.68, p = .031; F(7, 270) = 43.35, p < .001), and the condition × PI × progestogenic activity interaction was significant (β = .09, t = 2.16, p = .031), indicating that the moderating effect of progestogenic activity on the relationship between PI and disgust differed by condition.

To better understand the nature of this interaction, simple slopes analyses examined the association between PI and state disgust within each condition at low (-1 *SD*), mean, and high (+ 1 *SD*) levels of progestogenic activity using PROCESS (Model 3). Multicollinearity was acceptable (all variance inflation factors [VIFs] < 2.06). Consistent with the regression model, the three-way interaction was significant (F(1, 270) = 4.65, p = .032). In the experimental condition only, greater PI predicted greater state disgust at mean (b = 4.10, *SE* = 1.99, t = 2.06 p = .041, 95% CI [.18, 8.03]) and high (b =

5.13, *SE* = 2.52, *t* = 2.04, *p* = .042, 95% CI [.18, 10.08]) levels of progestogenic activity, but not at low activity (*b* = .64, *SE* = 2.45, *t* = .23, *p* = .796, 95% CI [-4.19, 5.46]; see Figure 2.4). In the control condition, PI did not significantly predict state disgust at any level of progestogenic activity in the control condition (all *p*s ≥ .09), although a marginal effect of PI in this same direction was observed at low progestogenic activity (*b* = 4.49, *SE* = 2.63, *t* = 1.71, *p* = .089, 95% CI [-0.68, 9.67]).

Figure 2.4

Three-Way Interaction Between Perceived Infectability, Progestogenic Activity, and Manipulation Condition on State Disgust.



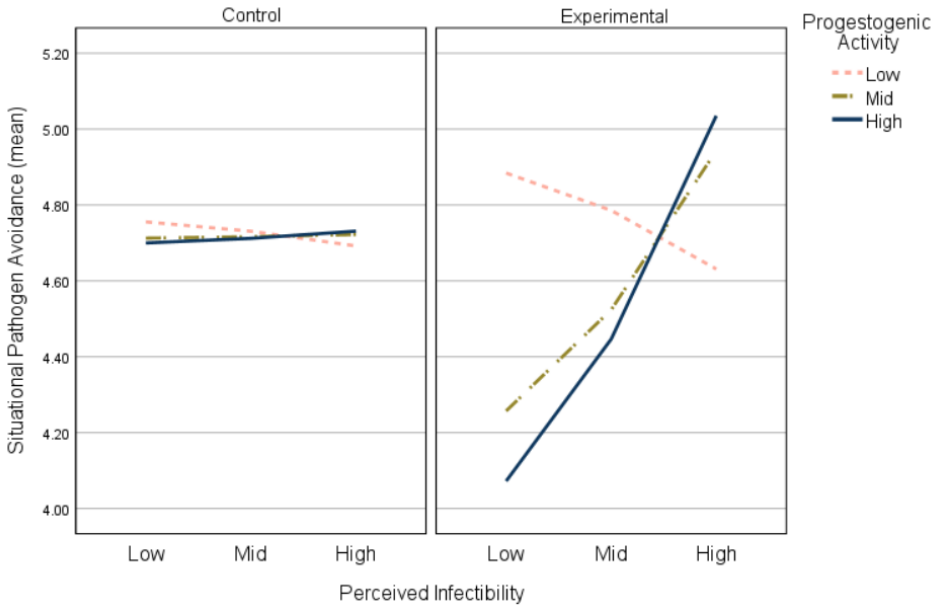
Note. Lines represent the association between perceived infectability and state disgust at low (-1 *SD*; dashed pink line), mean (dashed and dotted brown line), and high (+1 *SD*; solid blue line) levels of progestogenic activity. Panels display effects separately by experimental condition.

In predicting SPA, the main-effects model was nonsignificant (R^2 = .01, F(3, 274) = 1.00, p = .329). SPA was not predicted by condition (β = -0.04, t = -0.58, p = .559), PI (β = .09, t = 1.51, p = .132), nor progestogenic activity (β = -0.04, t = -0.72, p = .471). Adding the two-way interactions significantly improved model fit (ΔR^2 = .03, ΔF = 2.81, p = .040), although the model remained nonsignificant (F(6, 271) = 1.92, p = .078). Neither the condition × PI (β = .11, t = 1.78, p = .076) nor condition × progestogenic activity (β = -0.05, t = -0.82, p = .415) interactions were significant. However, the PI × progestogenic activity interaction was significant (β = .140, t = 2.33, p = .020), suggesting a potential interactive role of trait and hormonal vulnerability in shaping pathogen avoidance motivations. Adding the three-way interaction further improved model fit (ΔR^2 = .03, ΔF = 4.42, p = .036; F(7, 270) = 2.30, p = .027), and the condition × PI × progestogenic activity interaction was significant (β = .13, t = 2.10, p = .036).

To better understand the nature of these interactions, simple slopes analyses were conducted using the same specification as the above described state disgust model. Multicollinearity was acceptable (all VIFs < 2.07). The interaction between PI and progestogenic activity was significant only in the experimental condition (F(1, 270) = 6.99, p = .009). As with state disgust, greater PI predicted greater SPA at mean (b = .27, SE = .09, t = 3.07, p = .002, 95% CI [.10, .44]) and high (b = .37, SE = .11, t = 3.41, p = .001, 95% CI [.16, .59]) levels of progestogenic activity, but not low activity (b = -0.10, SE = .131, t = -0.75, p = .452, 95% CI [-0.32, .27]). See Figure 2.5 for this plotted interaction.

Figure 2.5

Three-Way Interaction Between Perceived Infectability, Progestogenic Activity, and Manipulation Condition on Situational Pathogen Avoidance.



Note. Lines reflect the association between perceived infectability and situational pathogen avoidance at low (-1 *SD*; dashed pink line), mean (dashed and dotted brown line), and high (+1 *SD*; solid blue line) levels of progestogenic activity. Panels display effects separately by experimental condition.

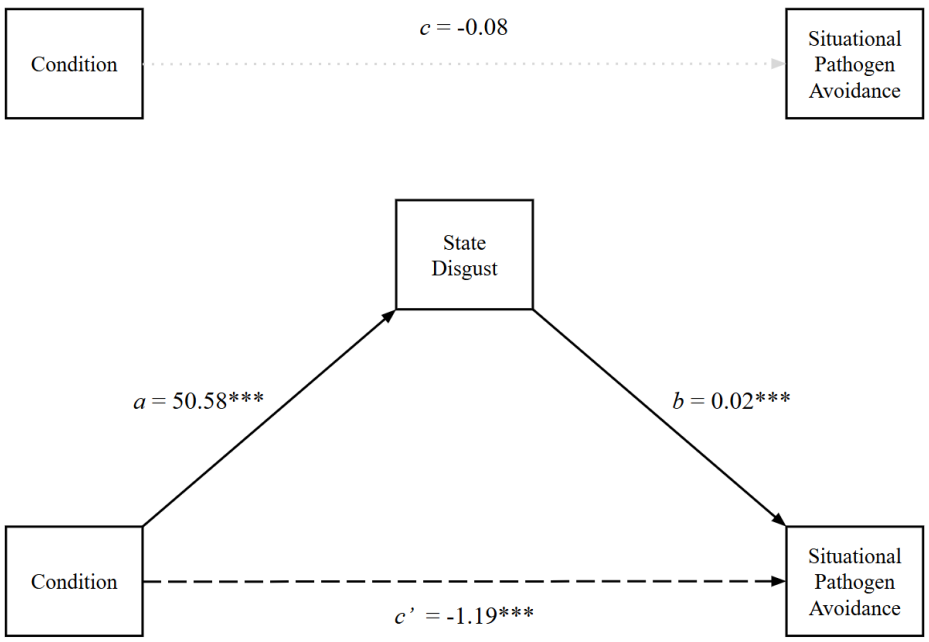
Indirect Effects of Pathogen Cues on SPA

Because the manipulation increased state disgust, but did not directly increase SPA, a mediation analysis was conducted using PROCESS Model 4 (Hayes, 2022) to test whether the experimental manipulation indirectly influenced SPA through state disgust. The experimental condition significantly predicted state disgust (R^2 = .52, F(1, 306) = 329.04, p < .001), indicating that 52% of the variance in state disgust was explained by

the condition. The overall model, with condition and state disgust entered as predictors of SPA, explained 18% of the variance in SPA (R^2 = .18, F(2, 305), = 29.81, p < .001). See Figure 2.6 for the conceptual model with associated pathways (unstandardized regression coefficients and their statistical significance) denoted.

Figure 2.6

Conceptual Mediation Model of the Effect of Condition on Situational Pathogen Avoidance via State Disgust.



Note. Values reflect unstandardized regression coefficient for the total effect (*c*; top) and the direct (*c'*) and indirect (*a*, *b*) paths in the mediation model (bottom). Solid black lines indicate statistically significant positive paths, dashed black lines indicate statistically significant negative paths, and dotted grey lines indicate nonsignificant paths.

***p < .001

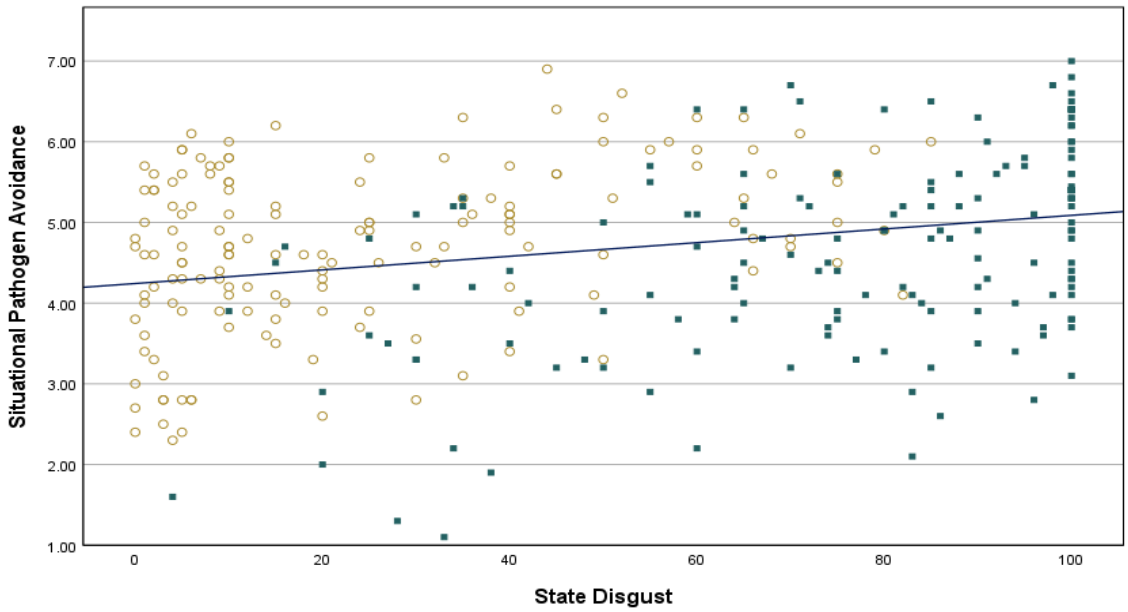
The manipulation did not have a significant total effect on SPA (*c* = -0.08, *SE* = 0.13, *t* = -0.61, *p* = .542, 95% CI [-0.33, 0.17]). The indirect pathways were significant; the manipulation robustly evoked disgust (*a* = 50.58, *SE* = 2.79, *t* = 18.14, *p* < .001, 95% CI [45,09, 56.07]), which in turn substantially increased SPA (*b* = 0.02, *SE* = 0.003, *t* = 7.70, *p* < .001, 95% CI [0.01, 0.02]). For each 1-point increase in disgust (measured on a 100-point scale), SPA (measured on a 7-point scale) increased by .02 points, indicating a robust effect of disgust on pathogen avoidance motivations.

The significant indirect effect (*ab* = .97, *BootSE* = 0.14, 95% CI [0.72, 1.25]) suggests that the manipulation's 50-point increase in state disgust translated to nearly a 1-point increase in SPA on its 7-point scale, representing a substantial 14% increase in pathogen avoidance motivation (see Figure 2.7).

Unexpectedly, the direct effect of the manipulation on SPA was negative (*c'* = -1.05, *SE* = 0.17, *t* = -6.07, *p* < .001, 95% CI [-1.39, -0.71]), indicating that participants in the experimental group reported 1 point *lower* SPA compared to the control group after controlling for disgust. Thus, the manipulation increased SPA only indirectly by elevating disgust, while simultaneously suppressing SPA through a direct pathway unrelated to state disgust.

Figure 2.7

The Association Between State Disgust and Situational Pathogen Avoidance Across Experimental Conditions.



Note. Dot shape and shading reflect condition (light circles = control, dark squares = experimental). The fitted regression line represents the overall association between disgust and situational pathogen avoidance.

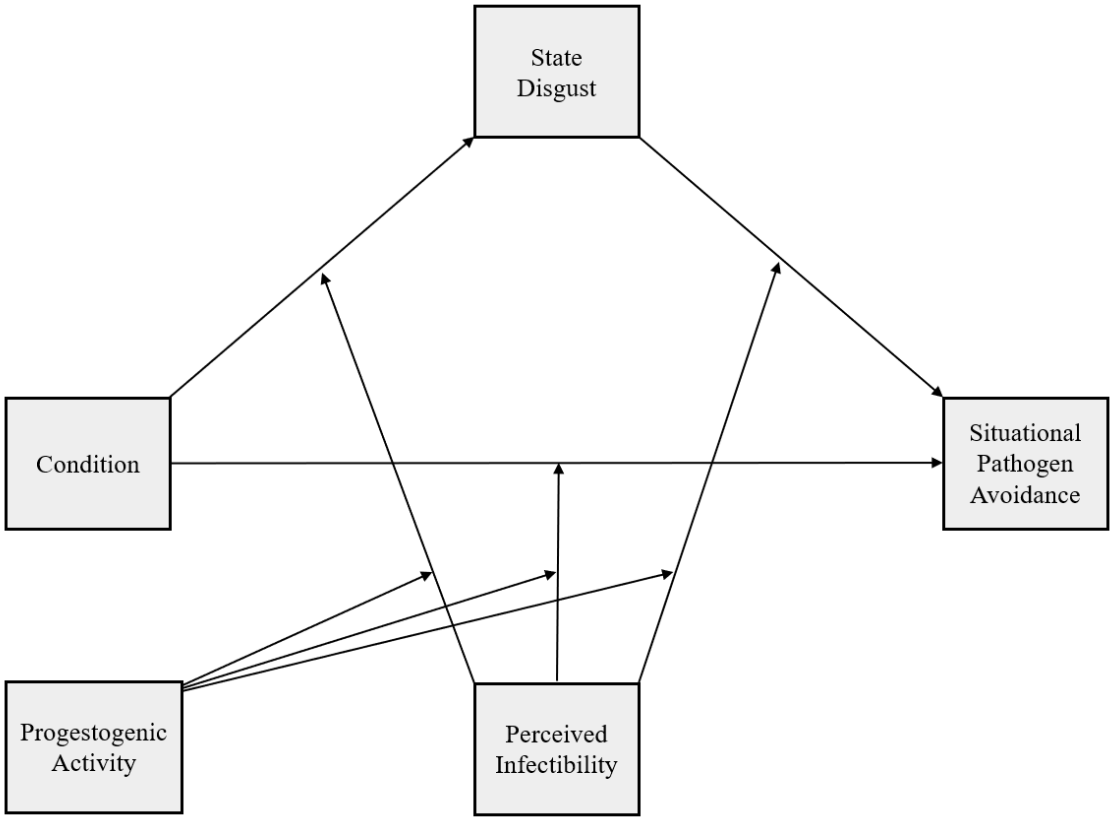
Conditional Indirect Effects on SPA

To investigate whether the indirect effect of experimental condition (*x*) on SPA (*y*) through state disgust (*m*) varied as a function of PI (*w*) and progestogenic activity (*z*), a moderated mediation analysis was conducted using PROCESS Model 73 (Hayes, 2022). This model was informed by earlier findings showing significant condition × PI × progestogenic activity interactions for both state disgust and SPA, as well as a negative direct effect of condition on SPA. As in prior analyses using progestogenic activity, only

participants in the active pill phase were included. Multicollinearity was acceptable (all VIFs < 4.68). See Figure 2.8 for a conceptual diagram.

Figure 2.8

Conceptual Moderated Mediation Model of the Effect of Condition on Situational Pathogen Avoidance via State Disgust, With Perceived Infectability and Progestogenic Activity as Moderators.



Note. The indirect effect of experimental condition on situational pathogen avoidance via state disgust is hypothesized to be moderated by perceived infectability, progestogenic activity, and their interaction.

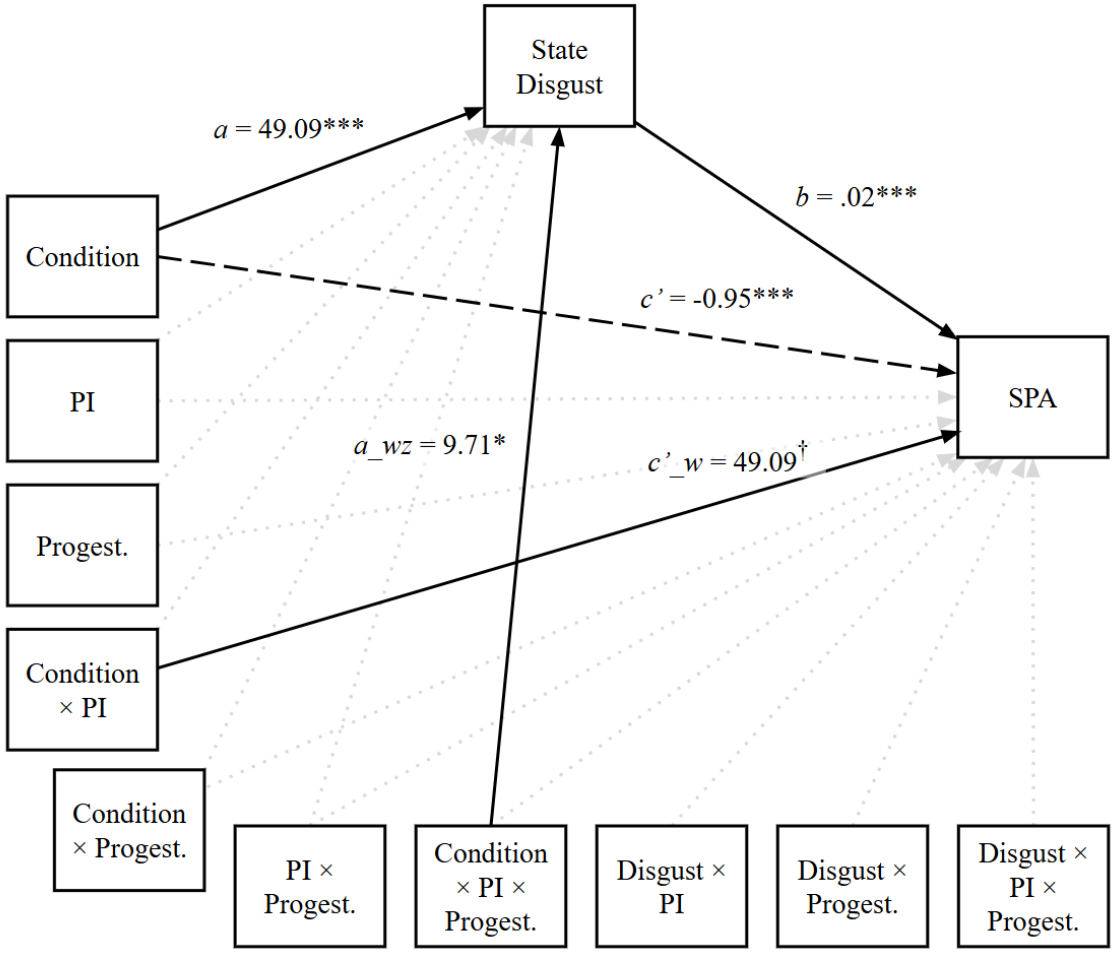
The overall model was significant (R^2 = .23, F(11, 266) = 5.20, p < .001), and including the additional predictors (PI, progestogenic activity) provided more explanatory power than the mediation model alone. As expected, the manipulation significantly increased state disgust (a = 49.09, SE = 2.99, t = 16.43, p < .001, 95% CI [43.21, 54.97]), and, in turn, state disgust predicted greater SPA (b = .02, SE = .003, t = 6.44, p < .001, 95% CI [.01, .02]). This mediating pathway (see Figure 2.9) reinforces the importance of emotional reactivity (i.e., state disgust) in shaping pathogen avoidance motivations.

A significant three-way interaction emerged on the a path (F(1, 270) = 4.65, p = .032), suggesting the effect of the manipulation on state disgust was dependent on both PI and progestogenic activity. Follow-up tests of conditional effects revealed that PI significantly moderated the manipulation's effect on disgust, but only among users of highly progestogenic OCs (+1 *SD*; F(1, 270) = 4.27, p = .040). In this high progestogenic group, disgust reactivity to the manipulation was strongest among participants with high PI. Although this interaction was nonsignificant in the moderately progestogenic (mean) OC group (F(1, 270) = 3.91, p = .113), the effect of the manipulation does begin to strengthen at higher PI. However, low progestogenic (-1 *SD*) OC users responded strongly to the manipulation regardless of PI (F(1, 270) = 1.15, p = .284). Thus, among highly progestogenic OC users only, PI moderated the effect of the manipulation on disgust reactivity.

Although earlier moderated regressions identified conditional effects on SPA, these effects did not extend to the b path from state disgust to SPA after shared variance with disgust was directly modeled. Although state disgust remained a significant predictor of SPA (b = .02, SE = .002, t = 6.44, p < .001, 95% CI [.01, .02]), this

Figure 2.9

Statistical Moderated Mediation Model of the Effect of Condition on Situational Pathogen Avoidance via State Disgust, With Perceived Infectability and Progestogenic Activity as Moderators.



Note: Solid black lines indicate significant positive paths, dashed black lines indicate significant negative paths, and dotted grey lines indicate nonsignificant paths. Values represent unstandardized regression coefficients.

Abbreviations: PI = perceived infectability, Progest. = progestogenic activity.

*$p < .05$, ***$p < .001$, $^{\dagger}p = .052$

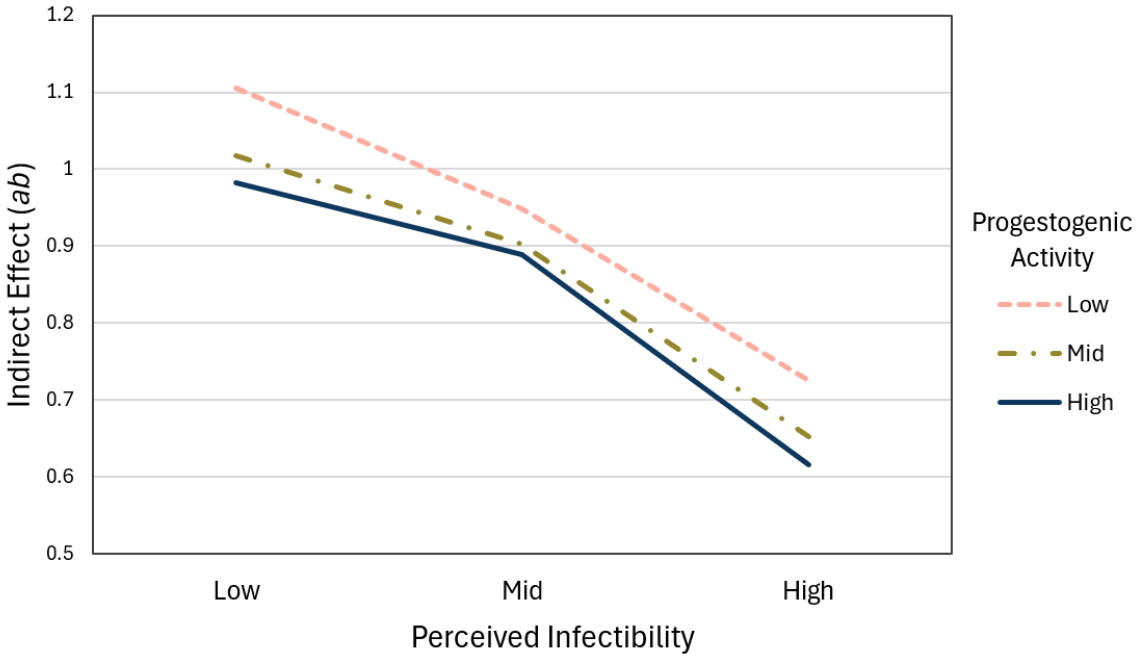
association was not moderated by PI or progestogenic activity. Neither the three-way interaction between state disgust, PI, and progestogenic activity (b_{wz} = -0.003, *SE* = .01, *t* = -0.58, *p* = .560, 95% CI [-0.02, .01]), nor the state disgust × PI interaction (b_w = -0.004, *SE* = .002, *t* = -1.44, *p* = .152, 95% CI [-0.01, .001]) was significant. Thus, once state disgust was included in this model, its positive association with SPA was comparable across levels of PI and progestogenic activity.

The conditional indirect effect (*ab* path; manipulation → state disgust → SPA) was significant across nearly all combinations of PI and progestogenic activity (see Figure 2.10). It was strongest at low PI and low progestogenic activity (ab_{wz} = 1.11, *BootSE* = .32, 95% CI [.53, 1.82]), and weakened as PI and progestogenic activity increased, becoming nonsignificant at high PI and high progestogenic activity (ab_{wz} = .62, *BootSE* = .34, 95% CI [-0.12, 1.26]).

In line with the mediation model, the direct effect of the manipulation on SPA was significantly negative (*c*' = -0.95, *SE* = .19, *t* = -5.03, *p* < .001, 95% CI [-1.32, -0.58]), indicating that, after accounting for state disgust, the condition retained a significant negative direct association with SPA (see Figure 2.11). There was a trend-level moderation of this negative relationship by PI (c'_w = .33, *SE* = .17, *t* = 1.95, *p* = .052, 95% CI [-0.003, 0.67], such that those higher in PI *may* be buffered against the SPA-inhibiting effect of the manipulation (all *p*s > .121). No other predictors or interactions were significant (all *p*s > .152).

Figure 2.10

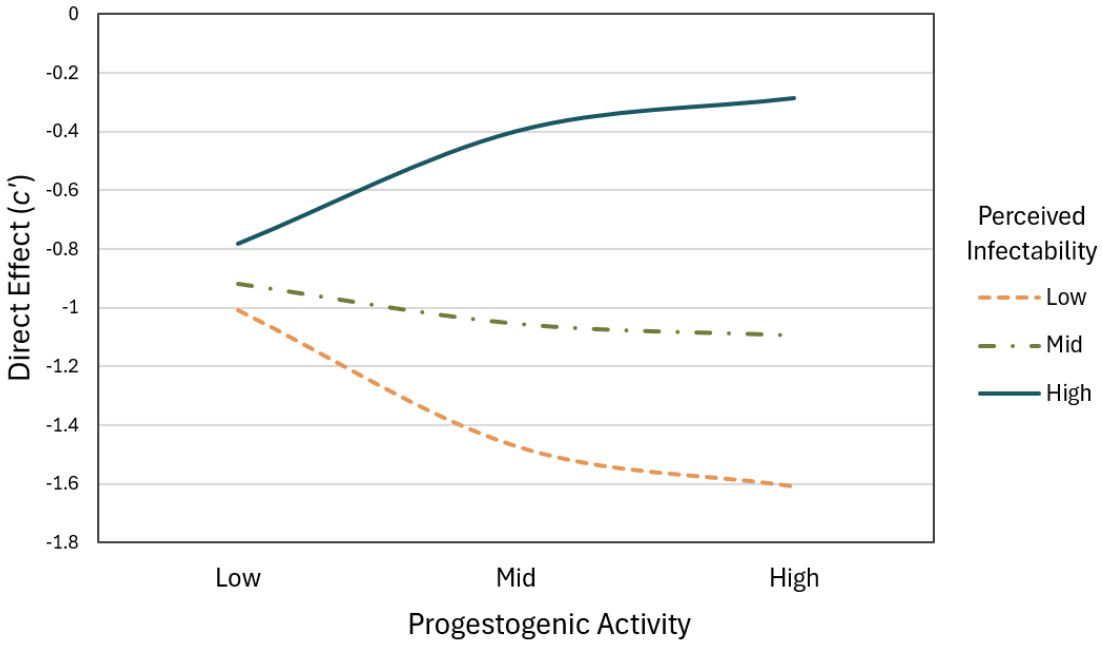
Indirect Effect of Experimental Condition on Situational Pathogen Avoidance via State Disgust, Moderated by Perceived Infectability and Progestogenic Activity.



Note. Lines represent conditional indirect effects (*ab*) of the pathogen-salient condition on situational pathogen avoidance via state disgust at low (-1 *SD*; dashed pink line), mean (dashed and dotted brown line), and high (+1 *SD*; solid blue line) levels of perceived infectability and progestogenic activity. Indirect effects decrease as both moderators increase.

Figure 2.11

Direct Effect of Experimental Condition on Situational Pathogen Avoidance, Moderated by Perceived Infectability and Progestogenic Activity.



Note. Lines represent conditional direct effects (*c'*) of the pathogen-salient condition on situational pathogen avoidance after controlling for state disgust, at low (-1 *SD*; dashed orange line), mean (dashed and dotted green line), and high (+1 *SD*; solid navy line) levels of perceived infectability and progestogenic activity.

Discussion

The present study examined whether synthetic progestins found in OCs influence behavioral immune responses to pathogen cues, and whether these responses differ by pill phase, progestogenic activity, and PI. It was hypothesized that participants exposed to a pathogen-salient manipulation would report increased state disgust and SPA, with these effects further amplified among women in their active pill phase and among those using

OCs with higher progestogenic activity. Additionally, PI was expected to increase BIS responses, particularly under conditions of pathogen salience.

BIS reactivity was not uniform across OC users, but rather depended on the combined influence of pathogen cues, perceived vulnerability, and contraceptive formulation. Most notably, the effect of the pathogen manipulation on state disgust was dependent on both PI and progestogenic activity. Among users of low-progestogenic OCs, pathogen exposure elicited strong disgust regardless of PI, suggesting that disgust reactivity in this group was relatively consistent across levels of perceived vulnerability. In contrast, among users of highly progestogenic OCs, pathogen cues elicited stronger disgust primarily among those higher in PI, whereas those lower in PI showed comparatively less disgust. Thus, under higher progestogenic exposure, behavioral immune reactivity appeared more dependent on individual differences in perceptions of infection vulnerability.

This pattern is consistent with a compensatory prophylactic interpretation like the CPH (Fessler, 2001). When progestogenic activity is higher and immunomodulatory effects are hypothesized to be stronger, BIS reactions may become more selectively engaged when multiple simultaneous cues signal an elevated risk. In the present study, those cues included both an external pathogen signal (the manipulation) and an internal vulnerability signal (high PI). More broadly, these findings suggest that OC formulation may shape the conditions under which this pathogen avoidance system is activated.

One speculative explanation for the observed negative association between progestogenic activity and disgust is that certain progestins may dampen emotional reactivity through neuroendocrine pathways. Endogenous progesterone is metabolized

into allopregnanolone (ALLO), a potent $GABA_A$ receptor agonist with anxiolytic effects, particularly during the luteal phase (Bitran et al., 1995; Porcu et al., 2019). Not all progestins appear to share this pathway, however. Levonorgestrel, which comprised 6.5% of the OCs in the present sample, cannot be converted to ALLO and has been shown to reduce ALLO levels (Follesa et al., 2002), although these reductions are not consistently linked to worsened mood (Rapkin et al., 2006). In contrast, other progestins common in this sample, including norethindrone acetate (45.9%; e.g., Loestrin 1/20) and drospirenone (7.2%; e.g., Yaz, Yasmin), have been associated with increases in ALLO (e.g., Fruzzetti & Fidecicchi, 2020; Lenzi et al., 2008; Memi et al., 2024; Pluchino et al., 2009) and with reduced anxiety-related symptoms or mood instability (e.g., Huber et al., 2008; Paoletti et al., 2004), although the extent to which ALLO directly accounts for these effects remains unclear (Pluchino et al., 2013). Disgust is closely related to threat sensitivity (Karg et al., 2019), anxiety (Knowles et al., 2018), and affective intensity (Stark et al., 2005). Variation in ALLO-related GABAergic signaling may therefore shape how strongly pathogen cues are interpreted as emotionally salient. Because ALLO has anxiolytic properties, lower progestogenic activity may be associated with heightened anxiety-related responding to pathogen cues, leading to stronger disgust reactivity even among individuals lower in PI. In contrast, higher progestogenic activity may dampen generalized affective reactivity to pathogen cues, such that heightened disgust responses emerge primarily among those who also perceive themselves as vulnerable to infection. Thus, two OCs with similar progestogenic activities may still differ in their effects on ALLO or broader GABAergic signaling, which could contribute to individual differences in BIS reactivity.

The mediation results are consistent with functional accounts of the BIS, which posit that disgust serves as a proximal mechanism motivating pathogen avoidance (reviewed in Oaten et al., 2009). In the present study, pathogen avoidance motivations were predicted by subjective disgust, not by the objective presence of pathogen cues alone, underscoring the central role of affective processes in guiding avoidance behavior. Although cognitive appraisals likely also contribute to avoidance, the current findings suggest that emotional reactivity may be especially important in responses to pathogen cues.

At the same time, the wide range of self-reported disgust observed in both conditions is notable. Although pathogen-salient imagery reliably evoked disgust in prior work (e.g., Curtis & Biran, 2001; Makhanova et al., 2021), and the present manipulation has been previously validated (Culpepper et al., 2018), participants varied substantially in their emotional responses to the same stimuli. This pattern suggests that subjective interpretation of the pathogen cues may shape emotional reactivity alongside the experimental manipulation itself. In other words, exposure to a pathogen cue may be insufficient if that cue is not experienced as being personally relevant, aversive, or threatening.

An unexpected finding was the significant negative direct effect of the manipulation on SPA after accounting for disgust. Although speculative, this pattern suggests that pathogen cues may not uniformly increase avoidance motivations when they fail to elicit disgust. Instead, exposure to pathogen cues in the absence of disgust may lower perceived personal risk or divide attentional resources, thereby limiting participants' ability to plan or report avoidance intentions. More broadly, the

manipulation increased disgust without directly increasing SPA, suggesting that the motivation components of the BIS may require more prolonged, immersive, or ecologically valid cues to activate. It is also possible that SPA reflects downstream behavioral intentions that depend on both emotional reactivity and additional cognitive processes. This possibility warrants further investigation with designs that manipulate the signal duration, intensity, and ecological validity to clarify when pathogen cues translate into avoidance motivations.

Together, PI and progestogenic activity shaped *who* became emotionally reactive to pathogen cues, but once disgust was experienced, it reliably motivated avoidance across participants regardless of trait or hormonal factors. This finding suggests that internal vulnerability cues may determine who reacts emotionally to pathogen threats, whereas disgust itself functions as the more immediate motivational component of the BIS. The current findings therefore extend functional flexibility models (see discussion in Schaller & Park, 2011) by suggesting that the BIS is not uniformly activated by threat cues, but instead is calibrated by an interaction between internal (hormonal, dispositional) and external (environmental) factors. Although previous work has often treated disgust as a relatively stable, trait-level predictor (e.g., see Tybur & Karinen, 2018 for a review), the present findings suggest that BIS reactivity may emerge most strongly when perceived infectability, progestogenic activity, and environmental threat aligned. These findings support a more flexible state-trait framework in which disgust varies in its activation, but remains relatively consistent in its motivational output once elicited.

Contrary to predictions, there were no significant differences in BIS responses between women in their active versus reminder OC phases. This broadly aligns with prior

work finding limited or null differences between OC phases on disgust-related outcomes (e.g., Radke & Derntl, 2016). However, these null effects should be interpreted cautiously, as only 27 participants were tested during their reminder pill phase, compared to 281 in their active pill phase, creating substantial asymmetry in statistical power. An imbalance was expected given the shorter reminder phase (4-7 days) relative to the active phase (21-24 days), as well as the requirement that participants be at least three days into their reminder phase to allow for greater clearance of synthetic hormones before participation. Nonetheless, the asymmetry was larger than anticipated and represents an important limitation of the current work. These phase-based comparisons were retained because they were pre-planned and address a theoretically meaningful question about whether BIS sensitivity changes when synthetic hormone exposure is temporarily halted. As such, they should be viewed as informative but underpowered tests that can guide future, more balanced, designs.

Limitations and Future Directions

Although Study 1 identified important trait-hormone interactions in BIS reactivity, several limitations do restrict these interpretations. Participants were recruited from the undergraduate Psychology Subject Pool and were, on average, approximately 20 years old. This may limit generalizability, as patterns of OC use (e.g., formulation type) in this sample may differ from those observed in older or more diverse populations. Nevertheless, women aged 20-29 represent the largest age group of OC users (21.6%; compared with 19.5% aged 15-19, 10.9% aged 30-39, and 6.5% aged 40-49; Daniels & Abma, 2020), which mitigates, but does not eliminate, concerns regarding age-related representativeness.

This study also used a between-subjects design, which increased the diversity of OC formulations across participants, but could not inform whether BIS reactivity shifts within individuals across their own pill phases. In addition, the sample was limited to OC users. This improved estimation of the formulation-specific progestogenic activity, but excluded users of non-oral HCs (e.g., implant, injection), whose pharmacodynamics are more difficult to quantify (Kuhl, 2005; reviewed in Bick et al., 2021). Unlike OCs, several non-oral methods deliver hormone levels that lessen over time following insertion or injection, producing variable exposure that is difficult to estimate for individual participants (Apter et al., 2014; Jain et al., 2004; Timmer & Mulders, 2000). The progestogenic activity values used here therefore reflect formulation-level estimates derived from receptor-binding and bioassay data (Dickey & Seymour, 2021), rather than participant-specific pharmacodynamic measurements. Individual differences in metabolism, adherence, and timing since their last pill were not measured and may have influenced these associations. Future research should incorporate adherence-related covariates and pharmacokinetic indicators (e.g., BMI) to improve estimates of formulation-specific effects.

Critically, although the observed pattern is consistent with a compensatory prophylaxis framework (see Fessler, 2001), the present study cannot determine whether behavioral activation co-occurred with suppressed inflammatory activity or instead reflected variation in emotional reactivity alone. Without direct immune measures, conclusions regarding hormonal modulation of disease-avoidance behavior remain indirect. Direct assessment of immune biomarkers is therefore necessary to determine whether the behavioral patterns observed here correspond more closely to compensatory

or complementary models of BIS–physiological immune coordination. Study 2 addresses this gap by assaying an immune marker to test whether state-level behavioral immune responses are associated with changes in physiological immune activity within and across hormonal contexts.

Finally, Study 1 assessed BIS reactivity exclusively through overt disgust and pathogen avoidance motivations. Although these represent foundational outputs of the BIS, they capture only part of its broader functional scope. The BIS has also been theorized to influence social judgments and group-level attitudes (e.g., Faulkner et al., 2004), including prejudice toward outgroups, xenophobia, and ethnocentrism. If progestogenic activity alters the sensitivity and activation of the BIS, such shifts may extend beyond immediate disgust responses to shape downstream social judgements. Study 3 tests this possibility by examining whether hormonal context and pathogen salience predict intergroup attitudes linked to pathogen avoidance.

CHAPTER THREE

STUDY 2

Although research on the interplay between inflammation and the BIS is still emerging, findings are often contradictory (reviewed in Murray et al., 2019). Two primary frameworks have been proposed to explain these interactions: complementary and compensatory models. The complementary framework suggests that activation of one system coincides with activation of the other, such that both increase or decrease in tandem. Within this model, detection of pathogen cues should prompt pathogen avoidance behaviors alongside an inflammatory response, with both systems working together to reduce infection risk. Empirical support comes from studies showing that exposure to pathogen-related stimuli increases both inflammatory and behavioral immune responses (Miller & Maner, 2011; Schaller et al., 2010; Stevenson et al., 2011, 2012, 2015). For example, participants exposed to images of pathogen threats report greater disgust and exhibit concurrent physiological immune activation, including elevated body temperature (Stevenson et al., 2012) and increased levels of cytokines such as IL-6 (Ersche et al., 2014; Gassen et al., 2019; Schaller et al., 2010) and TNF-α (Stevenson et al., 2011, 2012, 2015). These findings suggest that co-activation of physiological and behavioral defenses may enhance protection when pathogen threats are immediate or highly salient.

By contrast, the compensatory framework proposes that increases in one system correspond with decreases in the other, reflecting an adaptive tradeoff between the costs

and benefits of immune activation (e.g., Bradshaw et al., 2022; Ersche et al., 2014; Fleischman & Fessler, 2011; Gassen et al., 2018; Oaten et al., 2017; Stevenson et al., 2009). In this model, pathogen avoidance behaviors compensate for the costs of initiating and sustaining inflammation, including tissue damage and metabolic demand. For example, individuals with compromised immune function, such as those with rheumatoid arthritis (Oaten et al., 2017), exhibit higher trait behavioral immune activity, including greater self-reported germ aversion (see also Bradshaw et al., 2022; Dlouhá et al., 2023; but see de Barra et al., 2014). Similarly, people living in high pathogen-risk environments (e.g., regions with high infection-related mortality) demonstrate greater pathogen disgust sensitivity (Hlay et al., 2021; Prokop et al., 2010; Skolnick & Dzokoto, 2013) and lower baseline levels of inflammatory markers such as IL-6 and CRP (Cepon-Robins et al., 2021). These findings suggest that the BIS may adaptively reduce reliance on the physiological immune system while maintaining pathogen defense.

The direction of the relationship between inflammation and the BIS is therefore likely context-dependent. When pathogen threats are experimentally induced, complementary activation of both systems may provide more robust protection, as the immediate benefits of an immune response may outweigh its physiological costs (e.g., Miller & Maner, 2011; Schaller et al., 2010; Stevenson et al., 2011, 2012, 2015). In contrast, nonexperimental studies assessing trait-level disgust in the absence of acute pathogen cues more often identify compensatory patterns, with heightened behavioral responses corresponding to lower physiological immune activation (e.g., Bradshaw et al., 2022; Dlouhá et al., 2023; Ersche et al., 2014; Fleischman & Fessler, 2011; Gassen et al., 2018; Hlay et al., 2021; Oaten et al., 2017; Prokop et al., 2010; Skolnick & Dzokoto,

2013; Stevenson et al., 2009). Studies supporting compensatory dynamics tend to focus on long-term pathogen exposure or stable trait-level responses, whereas studies supporting complementary dynamics more often examine experimentally induced threats in novel settings. This adaptive balance may be especially relevant when mounting a physiological response is costly or difficult, such as during immunosuppression or chronic high pathogen exposure.

Other work suggests that complementary and compensatory dynamics may coexist. For example, Keller and colleagues (2022) found that viewing disease- and disgust-related videos increased salivary secretory immunoglobulin A (sIgA) relative to a neutral control condition, consistent with complementary activation. However, participants with higher trait contamination disgust exhibited dampened increases in sIgA, suggesting that chronic behavioral vigilance may reduce the need for reactive physiological upregulation. Thus, even within experimentally induced pathogen contexts, complementary and compensatory processes may operate simultaneously.

Progesterone is an immunomodulating hormone that suppresses inflammatory activity (reviewed in Raghupathy & Szekeres-Bartho, 2022; Zwahlen & Stute, 2023; see Chapter 1). Considering that pregnancy and, to a lesser extent, the luteal phase of the menstrual cycle, are both characterized by reduced pro-inflammatory and increased anti-inflammatory signaling (e.g., Denney et al., 2011; Vetrano et al., 2020), a compensatory framework would predict increased behavioral defenses to reduce infection risk. Given that pathogen threat manipulations, but not necessarily women's trait levels of disgust (e.g., Zelaźniewicz et al., 2016), are associated with the upregulation of key inflammatory biomarkers (e.g., IL-6, TNF-α; Gassen et al., 2019; Stevenson et al., 2015), whereas

progesterone suppresses inflammatory reactivity (reviewed in Raghupathy & Szekeres-Bartho, 2022; Zwahlen & Stute, 2023), it would be informative to identify the shifts in inflammatory biomarkers that underpin compensatory prophylaxis. Although progesterone-induced immunomodulation may not increase trait disgust within non-pathogenic contexts (e.g., Kaňková et al., 2022), it may increase state pathogen avoidance motivations when pathogen threat is salient – a context in which the risk to the immune system is much greater. Under these conditions, progesterone-induced immunomodulation may be offset by compensatory increases in pathogen avoidance motivations. However, most studies examining this relationship fail to consider the underlying immunological mechanisms contributing to behavioral prophylaxis, leaving significant gaps in understanding.

Much of this literature relies on methodologies that presume, rather than directly measure, immunosuppressed states. For instance, ovulatory cycle timing is often used to estimate immune function, with women in the luteal phase (e.g., Fessler & Navarrete, 2003) or those with high levels of progesterone (e.g., Fleischman & Fessler, 2011; Jones et al., 2018; Stern & Shiramizu, 2022) treated as immunosuppressed. Findings using these methodologies have been inconsistent, however. Some studies have not observed a relationship between trait disgust sensitivity and either estimated progesterone (i.e., calculated from self-reported menstrual timing; Rafiee et al., 2023) nor assayed progesterone levels (Jones et al., 2018). Other studies have reported greater trait disgust sensitivity among women in the luteal phase than the follicular phase (Miłkowska et al., 2021).

To more closely, though still indirectly, assess immune function, some studies

have asked participants to self-report short-term illnesses (e.g., a cold, the flu, inflammation; Hlay et al., 2024; Makhanova et al., 2022a; Miłkowska et al., 2019). Interestingly, compensatory increases in disgust sensitivity during the luteal phase were observed only among participants reporting a current illness, suggesting that progesterone-induced immunomodulation may heighten BIS sensitivity when the physiological immune system is already taxed. Similarly, Kaňková and colleagues (2022) found that among pregnant women in their first trimester, greater trait disgust sensitivity was associated with lower levels of several cytokines, including Th1 cytokines, cytokines that mediate innate immunity, and cytokines from the Th2 and Th17 families. Taken together, these findings suggest that compensatory prophylaxis may not broadly offset progesterone-induced immunomodulation, but instead may emerge most clearly when immune functioning is already compromised. Importantly, Kaňková and colleagues (2022) is one of the few studies to directly assess immunological markers within the CPH framework. Nonetheless, among studies that have assessed immunological biomarkers in relation to the CPH (e.g., white blood cell count, IL-1β, TNF-α, IFN-γ; Kaňková et al., 2022, 2023; Zelaźniewicz et al., 2016), all have measured the BIS at the trait level only. This approach may overlook situational shifts in pathogen avoidance motivations, which may be better suited to testing the adaptive tradeoff between physiological and behavioral immune responses. Understanding these situational dynamics is important for identifying the contexts under which compensatory or complementary relationships emerge, and assessing cytokines involved in innate and adaptive immunity may clarify when compensatory prophylactic behaviors are upregulated.

IL-6 and Immune Signaling

IL-6 is a cytokine with both pro- and anti-inflammatory functions (reviewed in Murakami et al., 2019), but its pivotal role in initiating local, systemic, and central immune responses, as well as shaping the adaptive immune response, makes it a key marker for exploring the interplay between behavioral and physiological immune responses (see "Cytokine Signaling Shapes the Immune Response" in Chapter 1 for a discussion). While these immune processes are essential for combating infection, inflammatory responses involving IL-6 are energetically costly and, if left unchecked, can contribute to muscle breakdown (reviewed in Chang & Bistrian, 1998) or the pathogenesis of autoimmune and inflammatory conditions (reviewed in Aliyu et al., 2022).

Notably, changes in IL-6 secretion can be detected in saliva as soon as 10 minutes after exposure to acute psychological stressors (e.g., the Trier Social Stress Test; Izawa et al., 2013; Kirschbaum et al., 1993), with levels typically peaking around 36 minutes after stressor onset (Szabo et al., 2020; reviewed in Slavish et al., 2015). These temporal dynamics make IL-6 a practical and sensitive marker for capturing short-term physiological immune reactivity. To be sure, IL-6 has been used as a biological marker in studies exploring the immunological underpinnings of the BIS, but, as noted above, much of this work relies on trait-level measures of disgust rather than state-level reactions to pathogen cues (see Murray et al., 2019 for a review). In the absence of a relevant pathogen cue, greater self-reported germ aversion has been associated with lower inflammatory cytokine levels (Gassen et al., 2018), aligning with the view that behavioral prophylaxis may compensate for reduced inflammatory responses. However, the extent of IL-6 reactivity in women appears to be modulated by reproductive hormones.

Meta-analytic work shows reduced IL-6 in cervicovaginal fluid during the luteal phase compared with the follicular phase (Hughes et al., 2022), aligning with progesterone's immunosuppressive role. Other immune effectors, such as beta-defensins (i.e., antimicrobial cells that line mucosal surfaces), also increase during the luteal phase, potentially buffering this suppression. HC use may similarly affect cytokines, with lower IL-6 when progestins are supplied (e.g., the active pill phase) and higher IL-6 during the inactive or reminder pill phase. If this is the case, HC users may display heightened prophylactic behaviors to compensate for reduced immune function associated with progestin exposure. However, progestins differ in their pharmacological properties, and these differences may influence mechanisms relevant to compensatory prophylactic behavior.

Biological Activities of Progestin

Although synthetic progestins were designed to mimic the physiological effects of progesterone on the endometrium (Mitchell & Welling, 2020), they vary substantially in their pharmacological properties and subsequent immunological effects. Some, such as MPA (reviewed in Hapgood et al., 2018; Heffron et al., 2019), bind strongly to glucocorticoid receptors (GRs), producing pronounced anti-inflammatory effects (e.g., Polis et al., 2016; reviewed in Hapgood et al., 2014). Because the GR is widely expressed throughout the body (Lu et al., 2006) and is a primary regulator of immune activity, strong GR agonism can suppress inflammatory signaling, including proinflammatory cytokine production (De Bosscher et al., 2000; reviewed in Africander et al., 2011). Other progestins exhibit weaker GR activity but may still influence cytokine signaling through progesterone receptors (e.g., Ichioka et al., 2015; Terakawa et al., 1987; reviewed in Reis

et al., 2020). Thus, progestins should not be treated as biologically interchangeable.

Progestin-induced immunomodulation may also have downstream implications for infection vulnerability. HC users show elevated risk for certain infections, including sexually transmitted infections (reviewed in Brabin, 2004; Hapgood et al., 2018), with GR-mediated mechanisms implicated in the association between MPA and HIV acquisition (Polis et al., 2016). Other progestins, such as norethindrone enanthate (Quispe Calla et al., 2020) and levonorgestrel (Quispe Calla et al., 2016a), alter mucosal barriers and increase susceptibility to pathogens, including Herpes Simplex Virus 2 and chlamydia (Miguel et al., 2018; Quispe Calla et al., 2016b). Correspondingly, combined OC users, relative to NC women, report more symptoms of illness at other mucosal barriers, including more frequent sneezing and greater gastrointestinal distress (Scanlan et al., 1995). Together, these findings suggest that variation in progestin pharmacology may meaningfully shape immune functioning and, by extension, behavioral defenses against disease.

The Current Study

The present study directly tested whether hormonal context and formulation-specific progestogenic activity help explain when behavioral and physiological immune responses operate in compensatory versus complementary ways. Building on Study 1, which identified hormone- and trait-dependent variation in behavioral immune reactivity, the current study incorporated a physiological immune marker (salivary IL-6) to examine whether changes in disgust and pathogen avoidance correspond to shifts in inflammatory activity. Prior work suggests that the behavioral and physiological immune systems may either co-activate under threat (complementary model) or exhibit tradeoffs when

inflammatory activation is costly (compensatory model). The present design was intended to test these competing possibilities across NC and OC hormonal contexts.

This study investigated these relationships among NC women (1 session) and OC users (2 sessions: active pill phase, reminder pill phase). All participants completed a pathogen threat manipulation, provided salivary samples before and after the manipulation (assayed for IL-6), and completed self-report measures assessing both trait and state behavioral immune responses, including PI (a subscale of the PVD scale; Duncan et al., 2009), disgust sensitivity (measured by the DS-R; Haidt et al., 1994; modified by Olatunji et al., 2007b), state disgust, and SPA (Makhanova et al., 2021).

In the absence of a pathogen threat (control condition), both IL-6 reactivity and state-level BIS activation (state disgust and SPA) were expected to be low. However, among individuals high in trait BIS sensitivity (PI and disgust sensitivity), attenuated IL-6 reactivity was expected, consistent with a compensatory model in which elevated behavioral vigilance reduces the need for inflammatory activation. In the experimental condition, complementary co-activation of IL-6 and BIS responses was expected, particularly among individuals high in trait BIS sensitivity.

Consistent with the CPH, hormonal context was expected to shift these dynamics. In high progesterone contexts, whether endogenous (luteal phase) or exogenous (active OC phase), blunted IL-6 reactivity paired with heightened BIS reactivity was expected, particularly among participants higher in trait BIS sensitivity, reflecting compensatory activation. Conversely, in low progesterone contexts (follicular phase or reminder OC phase), complementary increases in both IL-6 and BIS reactivity were expected. These predictions were tested both between groups and, among OC users, within the same

participants across pill phases.

Among OC users, higher progestogenic activity was further expected to predict greater IL-6 suppression accompanied by stronger compensatory BIS reactivity, reflecting a dose-dependent moderation of the compensatory pattern.

Method

Design

This study employed a mixed-combined design. Participants were randomly assigned to either a pathogen-salient (experimental) or pathogen-free (control) condition. NC participants completed one session, whereas OC users completed two sessions (active pill phase and reminder pill phase).

Within each session, participants provided two saliva samples (baseline and post-manipulation). Physiological immune reactivity was examined using two complementary analytic approaches. First, IL-6 change scores (post-manipulation minus baseline) were computed to create a continuous index of within-session IL-6 reactivity for regression, mediation, and moderation analyses. Second, repeated-measures analyses modeled baseline and post-manipulation IL-6 concentrations directly, with timepoint treated as a within-session factor. Behavioral immune reactivity was assessed via state disgust and SPA. Hormonal context was assessed categorically (NC follicular vs. luteal; OC active vs. reminder) and continuously among OC users via progestogenic activity. Trait PI and hormonal context were examined as individual difference moderators of behavioral and physiological immune reactivity. The primary outcome variables were state disgust, SPA, and IL-6 reactivity.

Participants

Cisgender women (N = 287) between the ages of 18 and 35 years old (M = 19.75, SD = 2.24) were recruited from Oakland University's Psychology Student Subject pool, including 213 NC women and 74 women using an OC. NC participants were defined as those with regular menstrual cycles who were not using HCs. Eligible OC users were required to use a formulation that included both active and reminder pills, could not skip their reminder pill phase (e.g., to prevent withdrawal bleeding), must have taken at least 2 complete days of their current pill phase, and were not using another form of hormonal contraception in addition to their OC. Prior to analyses, 33 participants were excluded for the following reasons: requesting removal of their data (n = 25; NC = 21, OC = 4), having incomplete surveys that were missing both BIS reactivity measures (n = 4, all NC), submitting surveys with no variability in responses (n = 1 OC user), reporting a current pregnancy (n = 1 NC participant), or completing the survey multiple times with inconsistent hormone information indicating poor data quality (n = 2, both OC users).

This left 254 participants (M_{age} = 19.78, SD_{age} = 2.31) for analyses (n = 188 NC [99 follicular phase, 85 luteal phase, 4 missing NC phase data], n = 66 OC [42 active pill phase, 23 reminder pill phase, 1 missing OC phase data) who were not pregnant or breastfeeding. Participants were predominantly White (62.2%; 13.4% Black, 8.6% multiracial, 15.7% "other"), heterosexual (77.2%; 16.9% bisexual, 2.8% homosexual, 3.1% "other"), and single (55.5%; 43.3% in an exclusive romantic relationship, 1.2% nonmonogamous).

Materials

Demographics Questionnaire

Participants provided demographic information (i.e., age, biological sex, sexual

orientation, relationship status, ethnicity, recent illness) as per Study 1.

Menstrual Cycle Questionnaire

Naturally cycling participants completed a menstrual cycle questionnaire to estimate their ovulatory cycle phase at the time of their study participation. Participants reported the first day of menstrual flow from their two most recent periods, the expected start date of their next period, and their typical cycle length (i.e., the number of days from the first day of one menstrual period to the day before the next begins). For each response, participants estimated their confidence on a 1 ("Not at all certain") to 9 ("Completely certain") scale.

NC participants' cycle phase at the time of participation was estimated using their self-reported menstrual cycle dates and confidence ratings from the menstrual cycle questionnaire. For each participant, estimates of typical cycle length were calculated using a weighted average of provided cycle lengths, with greater weight given to responses with higher self-reported certainty. These estimates were then included in a fertility phase calculation that uses a combined approach of forward- (i.e., estimating the next menstrual onset by counting forward the estimated cycle length from the most recent menstrual onset; e.g., Ecochard & Gougeon, 2000) then backward- (i.e., estimating day of ovulation by counting backward 15 days from the estimated next menstrual onset; e.g., Dixon et al., 1980) counting to categorize participants into high- or low- fertility groups at the time of their participation, corresponding to low versus high progesterone, respectively. Sessions that occurred between the most recent menstrual onset and the predicted day of ovulation were classified as follicular phase (n = 99), whereas sessions after the predicted day of ovulation but before the next estimated menstrual onset were

classified as luteal phase (n = 85). This combined method is commonly used in behavioral endocrinology research to estimate participants' day of ovulation and to determine high- and low-fertility windows based on self-report (for more details on these methods for ascertaining cycle phase, see Gangestad et al., 2016; Welling & Burriss, 2019).

Oral Contraceptive Questionnaire

For OC users, the OC questionnaire was nearly identical to that used in Study 1 (see Chapter 2 materials for details), except that the drop-down menu for selecting OC brand included 36 commonly prescribed formulations, rather than the full list of currently available formulations included in Study 1. Participants who did not see their OC brand listed selected "Other" and were asked to manually enter the medication name and dosage information as listed on their pill pack.

Perceived Vulnerability to Disease Questionnaire

The PVD (Duncan et al., 2009) was given as per Study 1 (α = .797); PI: α = .904, GA: α = .701). Only the PI subscale was included in analyses.

Disgust Scale–Revised.

The DS-R (Haidt et al., 1994; modified by Olatunji et al., 2007b) was given as per Study 1 (α = .861; Core Disgust: α = .730, Animal Reminder Disgust: α = .797, Contamination Disgust: α = .667). Given its stronger internal consistency relative to the subscales, the DS-R total score (i.e., DS-R) was again retained for analysis.

Culpepper Disgust Image Set

The C-DIS (Culpepper et al., 2018) was used as per Study 1 (Experimental Condition: α = .911; Control Condition: α = .922).

Behavioral Immune System Reactivity

BIS reactivity was assessed in two ways, as described in Study 1: (1) a single state disgust item, “How disgusted do you feel right now?”, and (2) the SPA (α = .813; Makhanova et al., 2021).

Progestogenic Activity of OCs

For OC users, progestogenic activity was determined as per Study 1. See Table 3.1 for progestogenic activity values for the most commonly prescribed OC brands in this sample: The five most common formulations account for 80% of OC use in this sample, with Loestrin 1/20 being the most common (as in Study 1). A range of phasic types, progestin types, and progestogenic activities are represented.

Physiological Data

IL-6 Collection and Quantification

Each participant provided two saliva samples per session: one before and one after the experimental manipulation. This resulted in two total samples for NC participants and four for OC users. Salivary IL-6 concentrations were quantified using a commercially available sandwich enzyme-linked immunosorbent assay (ELISA; Salimetrics, PA, USA) and analyzed using Gen5 data reduction software, which converts optical density (OD) readings into concentration estimates based on a standard curve. The assay has a lower limit of detection (LOD) of 0.07 pg/mL and a working range of 1.56-100 pg/mL.

All standards, controls, and saliva samples were run in duplicate. Samples were pipetted into microtiter plate wells pre-coated with IL-6-specific antibodies, allowing IL-6 present in the samples to bind to the plate. After incubation and 4x washing, biotin-

Table 3.1

Most Common Oral Contraceptives in the Sample (≥ 5% of Participants) by Brand, Formulation, and Progestogenic Activity

Brand	*n*	%	Formulation details	Progestogenic activity
Loestrin 1/20	17	25.8	21 active (1 mg NEA / 20 µg EE), 7 reminder (inert)	1.2
Alesse	11	16.7	21 active (0.10 mg LNG / 20 µg EE), 7 reminder (inert)	0.5
Desogen	7	10.6	21 active (0.15 mg DES / 30 µg EE), 7 reminder (inert)	1.5
Ortho-Cyclen 28	7	10.6	21 active (0.25 mg NG / 35 µg EE), 7 reminder (inert)	0.4
Ortho Tri-Cyclen Lo	7	10.6	7 active (0.18 mg NG / 25 µg EE), 7 active (0.215 mg NG / 25 µg EE), 7 active (0.25 mg NG / 25 µg EE), 7 reminder (inert)	0.3
Ortho Tri-Cyclen	4	6.1	7 active (0.18 mg NG / 35 µg EE), 7 active (0.215 mg NG / 35 µg EE), 7 active (0.25 mg NG / 35 µg EE), 7 reminder (inert)	0.2
Remaining brands	13	19.7		

Note: Formulation details list active pills followed by reminder (placebo) pills. Dosages are given as mg progestin/µg EE per active tablet. Progestogenic activity values reflect a relative potency (higher values indicate greater progestogenic activity) derived from Dickey and Seymour (2021).

Abbreviations: OC = oral contraceptive; DES = desogestrel; EE = ethinyl estradiol; LNG = levonorgestrel; NEA = norethindrone acetate; NG = norgestimate.

conjugated, goat-derived anti-IL-6 antibodies were added to tag the captured IL-6. Following a second incubation and 4x wash, streptavidin conjugated to horseradish peroxidase (HRP) was added to bind to the biotin tags. Following a third incubation and 4x wash, tetramethylbenzidine (TMB) solution was added, which reacted with the HRP enzyme to produce a measurable color change in each well proportional to the amount of bound IL-6. The reaction was stopped with acidic Stop Solution, changing the color from blue to yellow. OD was measured at 450 nanometers (nm) with a secondary wavelength correction at 630 nm.

To assess intra-assay precision of duplicates, the coefficient of variation (CV) was calculated for each sample pair by dividing the standard deviation of the sample by the mean of the set. If a set exceeded 20% CV, a third measurement was conducted in single, and the two readings with the lowest CV were retained. The average intra-assay CV was 5.82% across 590 sample pairs, indicating acceptable intra-assay reliability and falling within conventional thresholds (<10%) for cytokine assays (Salimetrics, PA, USA).

Samples with IL-6 concentrations exceeding the assay's upper LOD (100 pg/mL) were diluted 5:1, re-assayed, and the measured values multiplied by 5 to estimate the original concentrations (range = .037 - 104.17 pg/mL). If these back-calculated concentrations still exceeded 100 pg/mL (n = 51 [8.6%] samples), they were treated as out-of-range and truncated at this upper limit (100 pg/mL). Samples with concentrations below the lower LOD (n = 24 [4.1%] samples) were imputed at one-half the LOD (0.035 pg/mL), following established practice in cytokine biomarker research (e.g., Goovaerts et al., 2013; Hornung & Reed, 1990; US Environmental Protection Agency, 2000). Because IL-6 distributions are typically non-normal in salivary immunoassay research (e.g., Miller

et al., 2013), IL-6 values were transformed prior to inferential analyses. Mean, minimum, and maximum raw IL-6 levels are reported in Table 3.2.

Procedure

NC participants completed a single session of the following procedures, whereas OC users completed two near-identical (see details below) sessions: one during their active pill phase and one during their reminder pill phase. Participants who completed their first session during their active pill phase completed their second during the reminder pill phase, and vice versa. OC users participated in their sessions only after completing at least 2 consecutive doses of their active or reminder pill. Because OC users participated in a second session, they were also asked to generate a unique ID code at the

Table 3.2

Raw IL-6 Concentrations at Baseline and Post-Test by Session.

Session		Baseline		Post-Test	
	n	**Mean (*SD*)**	**Range**	**Mean (*SD*)**	**Range**
Session 1 (all)	238	16.98 (29.80)	.04-100	19.14 (31.64)	.04-100
NC	177	16.86 (30.06)	.04-100	19.48 (32.34)	.04-100
OC	61	17.34 (29.25)	.04-100	18.16 (29.74)	.04-100
Session 2	57	21.08 (32.07)	.13-100	20.36 (28.91)	.27-100

Note: Concentrations are reported in pg/mL. Ranges reflect observed minimum and maximum values. Session 2 sample sizes reflect oral contraceptive users only. Abbreviations: IL-6 = interleukin-6.

end of Session 1 to link their data across sessions. This code was composed of: (1) the first letter of their middle name, (2) the first letter of their mother's first name, (3) the last two digits of their phone number, (4) their month of birth (as a number), and (5) the first letters of their self-identified ethnicity.

Participants enrolled in this study through the Psychology Subject Pool and selected a time to visit the lab for an introductory session. After providing informed consent, participants received their study kit and scheduled a time to return it. Each study kit contained a copy of the consent form, a unique participant ID number, two cryovials labeled with the participant ID number and marked at the 2 mL line, two short straws, two clear plastic specimen bags labeled with the participant ID number, disinfectant wipes, a Dixie cup, and a scheduling reminder card (for OC users). At the conclusion of each participant's introductory session, an appointment was scheduled for their kit drop-off. For NC participants, this occurred within 1 week of pick-up. For OC users, a window for completing their second session and their second kit drop-off were either scheduled at that same time or arranged later during their Session 1 drop-off, depending on the participant's preferences. OC users' drop-offs were scheduled to correspond with their pill pack timing (e.g., no sooner than three days into their pill phase for users that just began a new phase) to ensure proper participation during each phase.

All other procedures were completed remotely via Qualtrics. After the introductory session (and again following session 1 kit drop-off for OC users), participants received the survey link at the email associated with their Subject Pool account. Participants were instructed to complete the study in a private, comfortable location and to refrain from eating or drinking for at least 10 minutes before beginning.

Upon opening the survey, they were prompted to swish and swallow at least 3 ounces of water (about the size of the provided Dixie cup) and indicate when they were finished. A 10-minute Qualtrics timer then began, during which participants entered their ID number and completed the following baseline measures: demographics (e.g., age, sexual orientation, relationship status, recent illness), menstrual cycle questionnaire (for NC participants) or oral contraceptive questionnaire (for OC users), and the DPSS-R.

After the 10-minute timer elapsed, participants were instructed to provide their first saliva sample (i.e., Time 1). The following instructions were provided:

> "It is now time to provide your TIME #1 saliva sample. Use the vial labeled "TIME 1" for this first sample. To reduce bubbling, place your straw into the provided TIME 1 vial and passively drool (you may want to tilt your head forward and to one side) through the straw into the vial without spitting your sample. If you are having trouble producing saliva, it is often helpful to rub your lower jaw or think about biting into a sour lemon or your favorite food. We need at least 2 mL of fluid saliva, not including bubbles, to properly analyze your sample. 2 mL is indicated on your vial, but we have emphasized this with a sharpie mark. When your fluid saliva reaches at least the 2 mL line, please remove your straw and tightly cap your TIME #1 vial."

Participants were instructed to proceed to the next screen only after their sample was completed and their vial was capped. Participants then received the following instructions:

> "IMPORTANT REQUIREMENT: Use the provided disinfectant wipes to wipe and disinfect the sides of your vial after it is capped and place your disinfected

vial into the provided plastic specimen bag labeled ‘TIME #1’. Move your bagged sample to your freezer until it can be returned to the research team. It is important to keep your sample frozen until it can be returned to Pryale Hall Room 103. To return the saliva sample, please transport the sample in a cooler with ice or gel packs and bring it directly to the researcher in Pryale Hall Room 103 at the agreed upon time.”

Participants confirmed they disinfected their vial, placed the vial in the plastic specimen bag, and placed the bagged sample into their freezer before continuing. These procedures were in line with the CDC COVID-19 guidelines for saliva collection at the time of Institutional Review Board (IRB) approval.

Next, participants completed the PVD and the DS-R. Participants were then randomly assigned to view and rate either pathogen-salient (experimental condition) or pathogen-free (control condition) images from the C-DIS, as in Study 1. Immediately after viewing the images, participants answered a single-item state disgust rating (“How disgusted do you feel right now?”; rated on a 0-100 slider). Following this manipulation, participants provided a second saliva sample (i.e., Time 2) following identical procedures to the first provided saliva sample. Again, instructions for vial disinfection and storage (bagging and freezing), and participants’ confirmation, followed on the next screen. Finally, participants completed the SPA scale followed by an "Honesty Verification" item to confirm data validity and exclude those who opted out of data analysis. Participants returned their completed kits to the lab at their scheduled time. Samples were immediately placed in a non-cycling freezer at -20°C until analysis.

For OC users, Session 2 procedures were identical to Session 1 with two

exceptions: (1) participants entered their unique ID code at the beginning of the Qualtrics survey, and (2) participants only answered the recent illness item from the demographics questionnaire (i.e., participants did not complete the entire demographics questionnaire for a second time) to capture any acute illness since Session 1. After returning their Session 2 kit, OC users' participation in the study was completed.

Results

Data Preparation

Of the total sample of 254 participants, 4 NC participants were missing sufficient data to determine their cycle phase and 1 OC participant was missing sufficient data to determine OC pill phase. All 5 were retained for analyses not requiring phase-based categorizations (NC follicular vs. luteal, OC active vs. reminder). Nineteen participants were missing the state disgust measure, and 18 were missing data for at least one IL-6 timepoint in Session 1 (including one who was also missing state disgust data). As a result, analytic sample sizes vary depending on the measures included. Analytic *n* is reported with each analysis.

Distributions of IL-6 concentrations were positively skewed and non-normal at all time points (skewness = 1.63-1.89; kurtosis = 1.40-2.29). This pattern is typical for salivary immunoassay data (see Miller et al., 2013) and data transformation is standard practice to meet assumptions of general linear model analyses. To improve normality and maintain homoscedasticity across all conditions, Box-Cox family of power transformations (Box & Cox, 1964) were applied following the approach of Miller and Plessow (2013). Box-Cox transformations test a range of power coefficients (λ) to identify the value that best reduces skewness and kurtosis while preserving equal

variance across groups. The Box-Cox transformation is defined as $X(\lambda) = (X^{\lambda} - 1)/\lambda$, where X is the original data value, λ is the power coefficient, and $X(\lambda)$ is the transformed value. For each IL-6 timepoint (Session 1 and Session 2 at baseline and post-test), λ was tested from 0 (natural log transformation) to 0.30 in increments of 0.05. For each transformation, skewness, kurtosis, Shapiro-Wilk tests of normality, and Levene's tests for equality of variance were evaluated across condition (experimental, control) and hormone groups (NC follicular, NC luteal, OC active, OC reminder).

The optimal balance between skewness reduction and variance equality was achieved at $\lambda = 0.05$. This transformation reduced skewness from 1.63-1.89 (untransformed) to -0.34-0.18 (transformed) and kurtosis from 1.40-2.29 (untransformed) to -1.01-.001 (transformed), while Levene's tests remained non-significant across groups. All subsequent analyses used IL-6 values transformed at $\lambda = 0.05$.

Data Analysis

Following data preparation, analyses were conducted in SPSS (Version 31), with statistical significance set at $\alpha = .05$. Unless otherwise noted, all models met assumptions of normality and homogeneity of variance. Analyses were designed to test whether physiological immune activity (IL-6) and behavioral immune responses (state disgust, SPA) operated in complementary or compensatory ways across hormonal contexts. Descriptive statistics were computed and compared by condition (experimental vs. control) and hormonal context (NC follicular, NC luteal, OC active, OC reminder). A manipulation check compared experimental and control groups on state disgust and SPA using independent-samples *t*-tests. A mediation model then tested whether the manipulation indirectly influenced SPA through disgust, extending the mediation pattern

observed in Study 1 to the current sample.

Two complementary approaches were used to examine IL-6 reactivity: IL-6 as a change score (ΔIL-6, post-test minus baseline) and IL-6 as a repeated measure. Mediation and moderated mediation models (PROCESS Models 4, 6, 59; Hayes, 2022) tested whether the manipulation influenced ΔIL-6 indirectly through state disgust and SPA, and whether these pathways varied by PI or hormonal context. Mixed analyses of covariance (ANCOVAs) and linear mixed-effects models (LMMs) treated baseline and post-test IL-6 as repeated measures, allowing comparisons both between hormonal groups and within participants across active and reminder OC phases.

Between-subjects models compared NC follicular versus luteal participants and OC active versus reminder users to test whether hormonal context shaped BIS–IL-6 associations across individuals. Within-subject models examined whether the same woman exhibited different BIS–IL-6 patterns across her active and reminder pill phases. PI and formulation-specific progestogenic activity were included as continuous moderators to test whether individual differences influenced the strength of the hypothesized compensatory pathway.

For PROCESS models, 5,000 bias-corrected bootstrap resamples were used, with HC4 *F*-values and standard errors. Predictors were entered uncentered, and PROCESS defaults were used to mean-center variables included in interaction terms. Unstandardized regression coefficients are reported, with pathways labeled according to standard notation (e.g., *a*: X → M, *b*: M → Y, *c'*: X → Y).

Descriptive Statistics and Preliminary Analyses

Descriptive statistics for participant demographics, trait-level BIS measures, and

state-level BIS responses are presented in Table 3.3. Chi-square tests of independence, independent samples *t*-tests, and one-way analyses of variance (ANOVAs) were used to compare descriptive variables across hormone groups (NC, OC) and conditions (experimental, control). Across most variables, no significant group differences emerged. However, relationship status differed by hormone group ($p = .008$), reflecting disproportionately more single participants in the NC group and more participants in exclusive relationships in the OC group. Race also differed by hormone group ($p = .005$), reflecting a greater proportion of White participants among OC users and "Other" race participants among the NC group. As expected, state disgust varied systematically by experimental condition ($p < .001$).

Replication of BIS Reactivity

Manipulation Check

To examine the effect of the pathogen manipulation on BIS reactivity, two independent-samples *t*-tests compared participants in the experimental and control conditions on state disgust and SPA (analytic $n = 235$ due to missing data). Replicating Study 1, participants in the experimental condition reported significantly greater state disgust than those in the control condition ($t(233) = -14.10$, $p < .001$; $M_{control} = 22.27$, $SD_{control} = 23.65$; $M_{experimental} = 68.42$, $SD_{experimental} = 26.10$). As in Study 1, SPA did not differ significantly between conditions ($t(252) = 0.90$, $p = .185$; $M_{control} = 4.58$, $SD_{control} = .95$; $M_{experimental} = 4.47$, $SD_{experimental} = 1.02$).

Table 3.3

Participant Characteristics by Hormonal Context and Experimental Condition

Variable	Total (*N* = 254)	NC (*n* = 188)	OC (*n* = 66)	Control (*n* = 127)	Experimental (*n* = 127)
Demographic variables, M (*SD*) or *n* (%)					
Age (years)	19.78 (2.31)	19.70 (2.46)	20.00 (1.77)	19.85 (2.62)	19.71 (1.95)
Sexual orientation					
Heterosexual	196 (77.2)	147 (78.2)	49 (74.2)	101 (79.5)	95 (74.8)
Homosexual	7 (2.8)	4 (2.1)	3 (4.5)	3 (2.4)	4 (3.1)
Bisexual	43 (16.9)	34 (18.1)	9 (13.6)	17 (13.4)	26 (20.5)
Other	8 (3.1)	8 (1.6)	5 (7.6)	6 (4.7)	2 (1.6)
Relationship status					
Exclusive relationship	110 (43.3)	71 (37.8)	39 (59.1)	54 (42.5)	69 (54.3)
Single	141 (55.5)	114 (60.6)	27 (40.9)	72 (56.7)	56 (44.1)
Nonmonogamous	3 (1.2)	3 (1.6)	0 (0)	1 (0.8)	2 (1.6)
Race					
White	158 (62.2)	108 (57.4)	50 (75.8)	79 (62.2)	79 (62.2)
Black	34 (13.4)	27 (14.4)	7 (10.6)	19 (15.0)	15 (11.8)
Multiracial	22 (8.7)	15 (8.0)	7 (10.6)	8 (6.3)	14 (11.0)
Other race	40 (15.7)	38 (20.2)	2 (3)	21 (16.5)	19 (15.0)
Behavioral immune system traits, M (*SD*)					
PI	3.53 (1.27)	3.48 (1.25)	3.68 (1.30)	3.41 (1.29)	3.65 (1.24)
DS-R	2.42 (.62)	2.46 (.65)	2.30 (.53)	2.49 (.62)	2.36 (.62)
Dependent variables, M (*SD*)					
State disgust	47.21 (33.97)	47.08 (34.32)	47.46 (33.25)	22.27 (23.65)	68.42 (26.10)
SPA	4.53 (.99)	4.53 (1.01)	4.53 (.93)	4.58 (.95)	4.47 (1.02)

Note: Values are presented as M (*SD*) or *n* (%). Group differences between naturally cycling and oral contraceptive hormonal contexts and between experimental conditions were evaluated using chi-square tests of independence or independent-samples *t*-tests. Abbreviations: PI = perceived infectability, DS-R = Disgust Scale–Revised, SPA = situational pathogen avoidance.

Indirect Effects of the Manipulation of Situational Pathogen Avoidance

Because SPA was not directly influenced by condition, a mediation analysis (PROCESS Model 4; Hayes, 2022) tested whether state disgust (M) accounted for variance in SPA (Y) associated with the manipulation (X), consistent with the pattern observed in Study 1. PI and self-reported recent illness were included as covariates to reduce potential confounding with trait BIS sensitivity and current health status. The model predicting state disgust was significant (R^2 = .48, F(3, 231) = 77.32, p < .001). As expected, the manipulation significantly increased state disgust (a = 45.09, SE = 3.28, t = 13.75, p < .001, 95% CI [38.63, 51.55]), which in turn predicted higher SPA (b = .01, SE = .003, t = 3.81, p < .001, 95% CI [.01, .02]). The model predicting SPA was also significant (R^2 = .10, F(4, 230) = 6.10, p < .001). The indirect effect was significant (ab = .44, *BootSE* = .12, 95% CI [.21, .70]), reflecting a .44-point increase in SPA (on a 1-7 scale) attributable to increases in state disgust elicited by the manipulation. As in Study 1, the direct effect of the condition on SPA remained significant and negative (c' = -0.61, SE = .16, t = -3.72, p < .001, 95% CI [-0.93, -0.29]).

Together, these findings replicate the Study 1 pattern, indicating that state disgust, rather than pathogen cues alone, was the proximal predictor of SPA. Subsequent analyses tested whether heightened disgust responses were also associated with downstream IL-6 reactivity, consistent with the proposed coordination between the behavioral and physiological immune systems.

Evidence for a Compensatory BIS–IL-6 Pathway

To test whether BIS reactivity compensates for inhibited IL-6 reactivity, the mediation model was expanded to include physiological immune reactivity as the

outcome. IL-6 reactivity was operationalized as a continuous change variable rather than as concentrations at two discrete timepoints. Following recommendations by Jennings & Cribbie (2016) for comparing pre-post change when baseline differences are present, IL-6 reactivity was calculated as the change in log-transformed IL-6 from baseline to post-test (ΔIL-6 = IL-6$_{post}$ – IL-6$_{baseline}$). In addition to excluding participants missing data at one or both IL-6 timepoints (n = 18), participants whose IL-6 concentrations were coded as extreme (both values at floor [n = 5] or ceiling [n = 18] for baseline and post-test) were excluded from change-score analyses because values outside the detectable range could not yield interpretable change estimates.

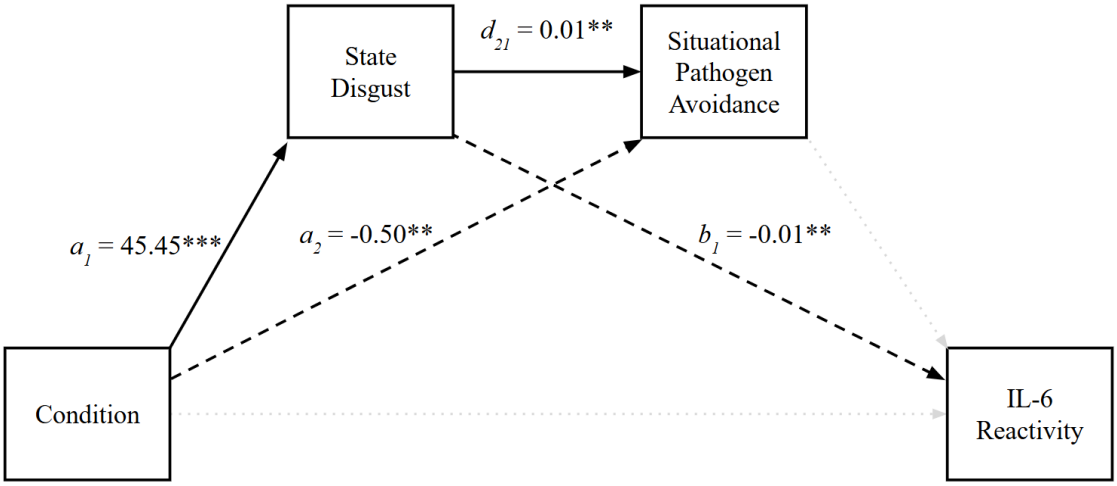
PROCESS Model 6 (Hayes, 2022) tested whether BIS reactivity to the manipulation predicted reduced physiological reactivity. Condition (control, experimental) was entered as the predictor (X), state disgust as the first mediator (M_1), SPA as the second mediator (M_2), and IL-6 reactivity as the outcome (Y). PI and recent illness were included as covariates. The analytic sample was limited to participants with valid state disgust data and both IL-6 timepoints, with at least one IL-6 value falling within the detectable range (n = 197). Multicollinearity was acceptable (all VIFs < 2.05). Findings are presented in Figure 3.1.

The manipulation strongly predicted state disgust (R^2 = .49, F(3, 193) = 69.30, p < .001), with participants in the experimental condition reporting approximately 45 points higher disgust than controls (a_1 = 45.45, SE = 3.57, t = 12.74, p < .001, 95% CI [38.42, 52.49]). The subsequent model predicting SPA from condition and disgust was also significant (R^2 = .10, F(4, 192) = 4.66, p = .001). As expected, higher disgust was associated with greater SPA (d_{21} = .01, SE = .003, t = 3.05, p = .003, 95% CI [.003, .01]),

whereas condition retained a negative direct effect on SPA (a_2 = -0.50, *SE* = .18, *t* = -2.74, *p* = .007, 95% CI [-0.86, -0.14]). Finally, the model predicting IL-6 reactivity from condition, disgust, and SPA explained a significant portion of variance (R^2 = .06, *F*(5, 191) = 2.67, *p* = .023). Within this model, state disgust significantly predicted reduced IL-6 reactivity (b_1 = -0.01, *SE* = .003, *t* = -2.98, *p* = .003, 95% CI [-0.02, -0.003]), whereas SPA (*p* = .524) and the manipulation (*p* = .421) did not.

Figure 3.1

Serial Mediation Model of the Effect of Experimental Condition on IL-6 Reactivity via State Disgust and Situational Pathogen Avoidance.



Note: Values represent unstandardized regression coefficients for the serial mediation model, with state disgust and situational pathogen avoidance entered as sequential mediators. Solid black lines indicate significant positive paths, dashed black lines indicate significant negative paths, and dotted gray lines indicate nonsignificant paths. **p < .01. ***p < .001.

Bootstrapped indirect effects revealed a significant negative pathway from condition to ΔIL-6 through state disgust alone (a_1b_1 = -0.45, *BootSE* = .16, 95% CI [-0.81, -0.18]). Indirect effects through SPA alone (a_2b_2 = .03, *BootSE* = .06, 95% CI [-0.06, .17]) and through the serial pathway ($a_1d_{21}b_2$ = -0.03, *BootSE* = .04, 95% CI [-0.14, .05]) were not significant. Thus, pathogen cues were associated with reduced IL-6 reactivity indirectly through increases in state disgust, consistent with a compensatory trade-off between behavioral and physiological immune responses. Building on this effect, subsequent analyses tested whether individual differences in PI altered the strength of this indirect pathway.

Moderation by Perceived Infectability

Because SPA did not significantly predict ΔIL-6 in the preceding serial mediation model, subsequent analyses focused on the significant indirect pathway through state disgust. Accordingly, a simplified moderated mediation model (PROCESS Model 59; see Figure 3.2) tested whether PI moderated the indirect effect of the manipulation on ΔIL-6 through state disgust. The analytic sample was limited to participants with valid data for both IL-6 timepoints and state disgust (analytic n = 197). Condition (control, experimental) was entered as the predictor (X), state disgust as the mediator (M), ΔIL-6 as the outcome (Y), and PI as the moderator (W), with recent illness included as a covariate. Multicollinearity was acceptable (all VIFs < 4.24).

Figure 3.2

Conceptual and Statistical Moderated Mediation Models of the Effect of Experimental Condition on IL-6 Reactivity via State Disgust, With Perceived Infectability as a Moderator.

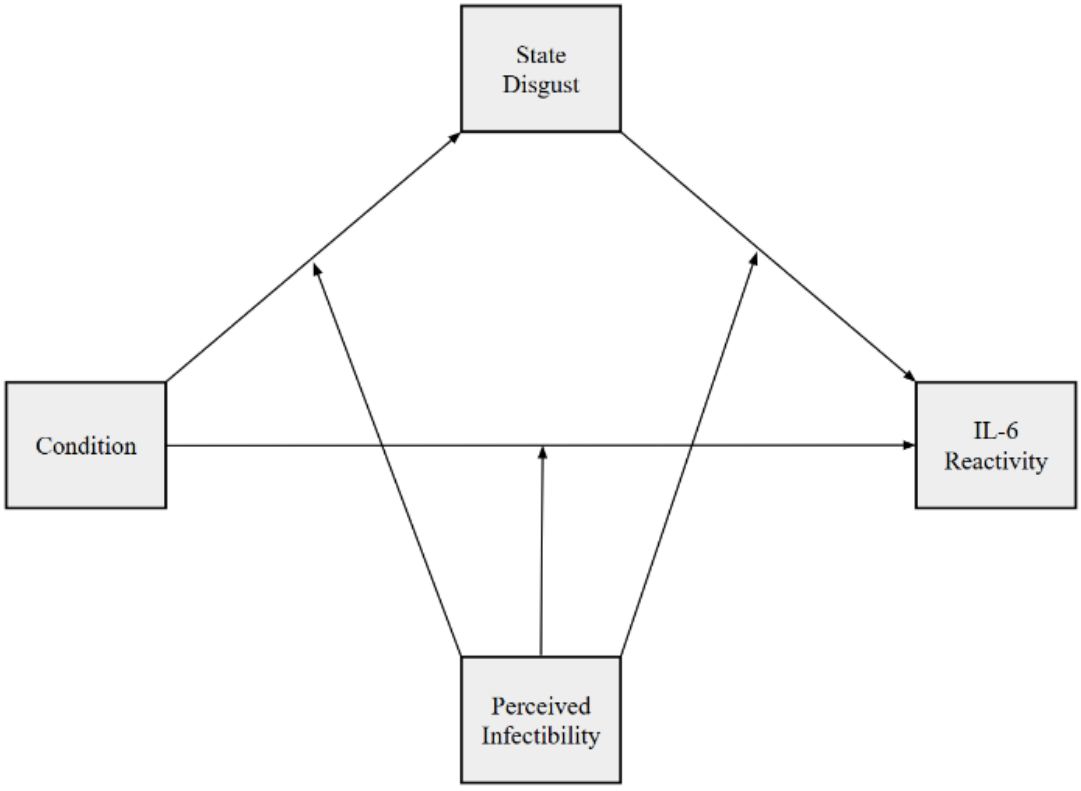
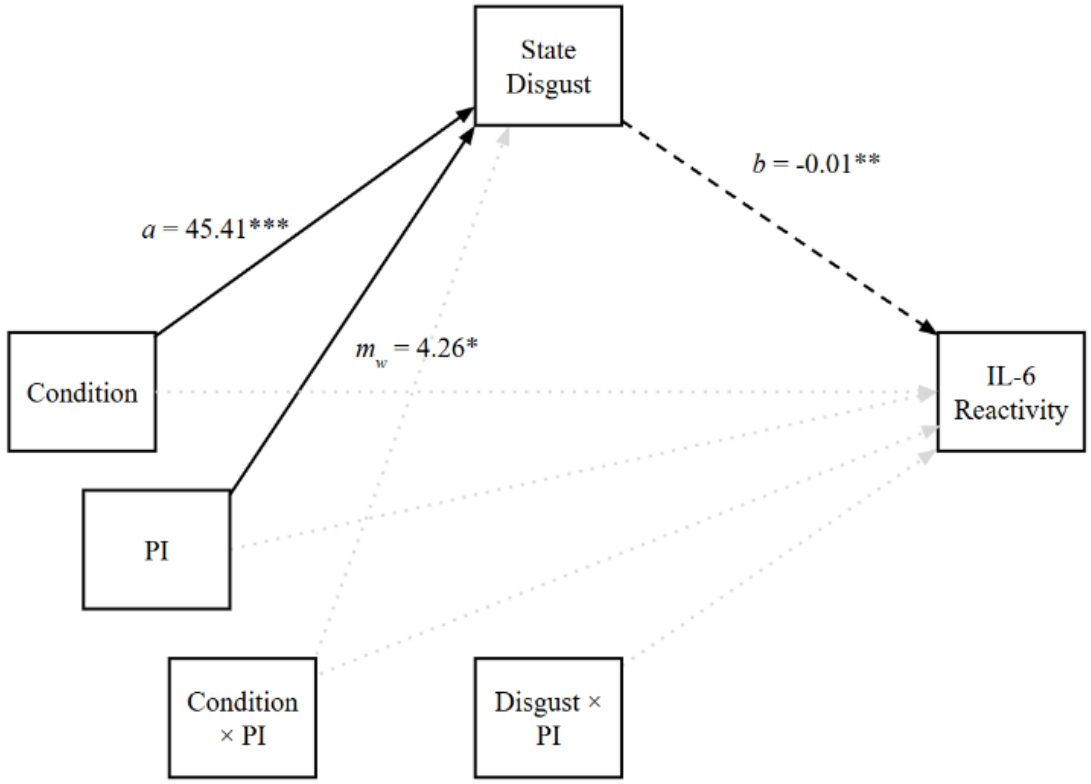


Figure 3.2 – Continued



Note: The top panel displays the conceptual moderated mediation model proposed theoretically, whereas the bottom panel depicts the statistical model estimated in PROCESS, including the observed regression paths and coefficients. Solid black lines indicate significant positive paths, dashed black lines indicate significant negative paths, and dotted grey lines indicate nonsignificant paths. Values represent unstandardized regression coefficients.

Abbreviations: PI = perceived infectability.

****p* < .001, ***p*< .01, **p* < .05*

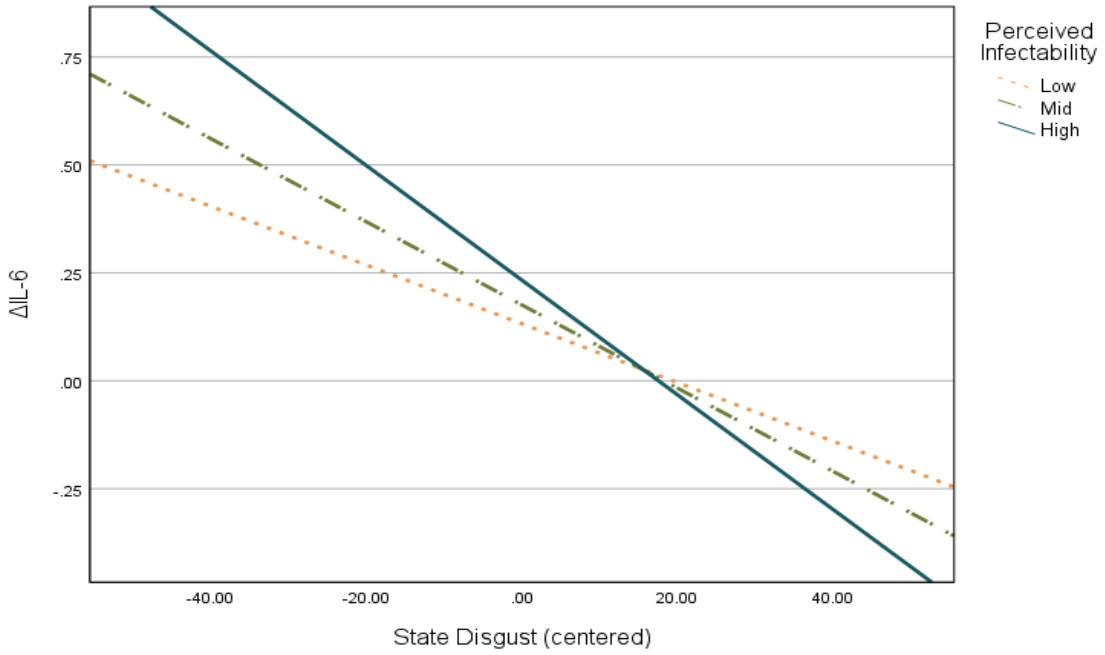
The model predicting state disgust was significant (R^2 = .49, F(4, 192) = 54.92, p < .001). The manipulation predicted significantly greater state disgust (a = 45.41, SE = 3.56, t = 12.67, p < .001, 95% CI [38.34, 52.49]). Higher PI also predicted greater overall disgust (m_w = 4.26, SE = 2.02, t = 2.11, p = .036, 95% CI [.28, 8.24]), but PI did not significantly moderate the effect of the manipulation on disgust (a_w = -3.22, SE = 2.96, t = -1.09, p = .277, 95% CI [-9.06, 2.61]). The model predicting ΔIL-6 was also significant (R^2 = .06, F(6, 190) = 2.35, p = .032). State disgust predicted significantly lower ΔIL-6 (b = -0.01, SE = .003, t = -2.83, p = .005, 95% CI [-0.2, -0.003]). PI did not predict IL-6 reactivity (y_w = -0.04, SE = .11, t = -0.40, p = .692, 95% CI [-0.26, .17]) and did not significantly moderate the association between state disgust and ΔIL-6 (b_w = -0.002, SE = .003, t = -0.84, p = .403, 95% CI [-0.01, .003]). Conditional effects indicated that state disgust predicted significantly lower IL-6 reactivity at average (b_w = -0.01, SE = .004, t = -2.71, p = .007, 95% CI [-0.02, -0.003]) and high (b_w = -0.01, SE = .01, t = -2.77, p = .006, 95% CI [-0.02, -0.004]) levels of PI, but not at low PI (p = .221; see Figure 3.3).

The direct effect of condition on ΔIL-6 was not significant (c' = .18, SE = .24, t = .73, p = .464, 95% CI [-0.30, .65]), nor was it moderated by PI (c'_w = .15, SE = .18, t = .83, p = .407, 95% CI [-0.21, .51]). However, the indirect effect of condition on ΔIL-6 through state disgust was significant and negative at mean (ab_w = -0.44, $BootSE$ = .17, 95% CI [-0.79, -0.14]) and high (ab_w = -0.54, $BootSE$ = .20, 95% CI [-1.03, -0.22]) levels of PI, but not at low PI (ab_w = -0.34, $BootSE$ = .27, 95% CI [-0.92, .17]; see Figure 3.4). These findings indicate that the indirect pathway was most evident at average-to-higher levels of PI, although the formal interaction terms were not significant. A Johnson-Neyman analysis further examined where the indirect effect was statistically reliable

along the observed range of PI. The indirect effect was significantly negative across most of the PI distribution, becoming non-significant at lower PI (-0.63) and again at the upper end of the observed range (3.16). The compensatory pathway was most consistent across typical levels of PI, but was less reliable at the lowest and highest observed PI values.

Figure 3.3

Interaction Between State Disgust and Perceived Infectability on IL-6 Reactivity.

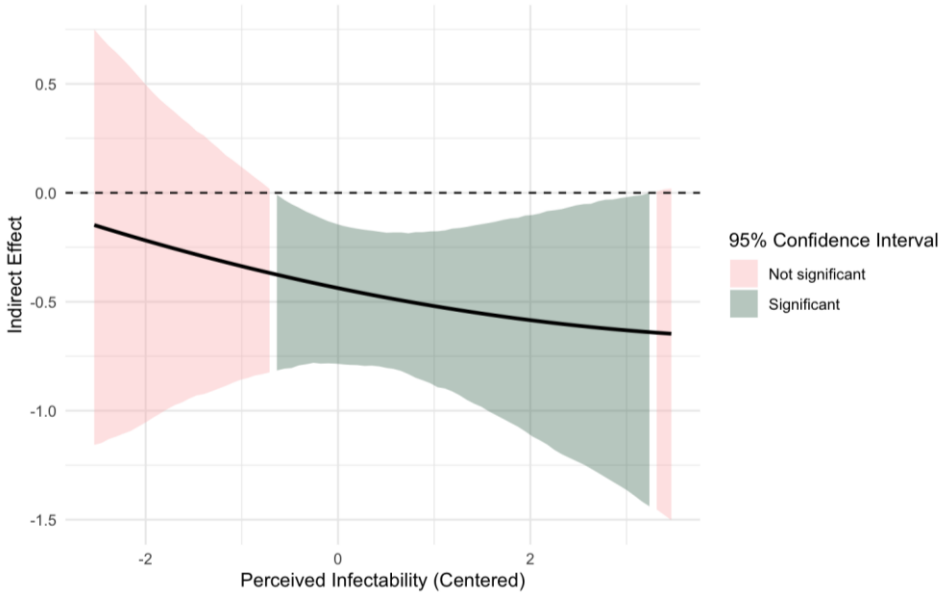


Note. Lines represent conditional effects of state disgust on ΔIL-6 at low (-1 *SD*; dotted orange line), mean (dotted and dashed green line), and high (+1 *SD*; solid navy line) levels of perceived infectability. Values reflect predicted ΔIL-6 across the observed range of mean-centered state disgust.

Abbreviations: ΔIL-6 = IL-6 reactivity.

Figure 3.4

Conditional Indirect Effect of the Experimental Condition on IL-6 Reactivity via State Disgust, Moderated by Perceived Infectability.



Note. The black line represents the estimated indirect effect (*ab*) across levels of perceived infectability. Shaded bands reflect 95% confidence intervals (CIs) derived from bootstrap resampling. Green regions indicate values of perceived infectability where the indirect effect is statistically significant, and pink regions indicate nonsignificant values.

Hormonal Context and Between-Subject Differences

Baseline IL-6 Differences Across Hormone Groups

To test whether baseline IL-6 concentrations differed across hormone groups (NC follicular, NC luteal, OC active, OC reminder), a one-way ANCOVA was conducted with recent illness included as a covariate. Analyses were limited to participants with baseline IL-6 data who could be categorized into a hormone group (analytic $n = 234$).

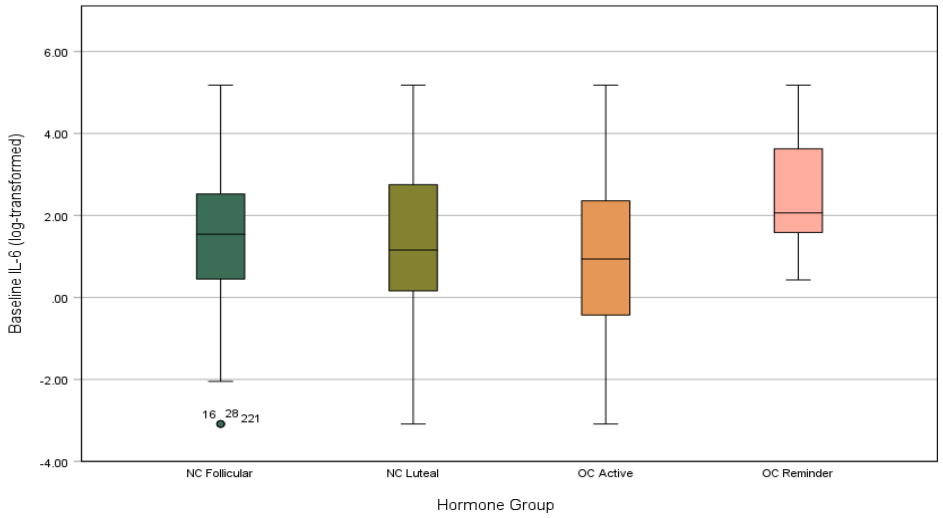
A significant effect of hormone group emerged ($F(3, 229) = 3.06$, $p = .029$; $\eta^2 =$

.04). Pairwise comparisons revealed that OC users in the reminder pill phase had significantly higher baseline IL-6 (*M* = 2.55, *SE* = .44, 95% CI [1.68, 3.43]) than OC users in the active pill phase (*M* = .93, *SE* = .32, 95% CI [.30, 1.56]; mean difference = 1.63, *SE* = .55, *p* = .020). No other pairwise comparisons were significant (all *p*s > .148).

These findings indicate that baseline IL-6 differed across hormonal contexts, with the clearest difference observed between OC users in the active versus reminder pill phases. Specifically, baseline IL-6 was lower during active pill use and higher during the reminder phase (see Figure 3.5).

Figure 3.5

Group Differences in Baseline IL-6 Concentrations Across Hormone Groups.



Note. Boxplots display the distribution of log-transformed baseline IL-6 concentrations across hormone groups (NC follicular, NC luteal, OC active, OC reminder). Boxes represent interquartile ranges, center lines indicate means, and whiskers represent the standard error.

Abbreviations: NC = naturally cycling, OC = oral contraceptive

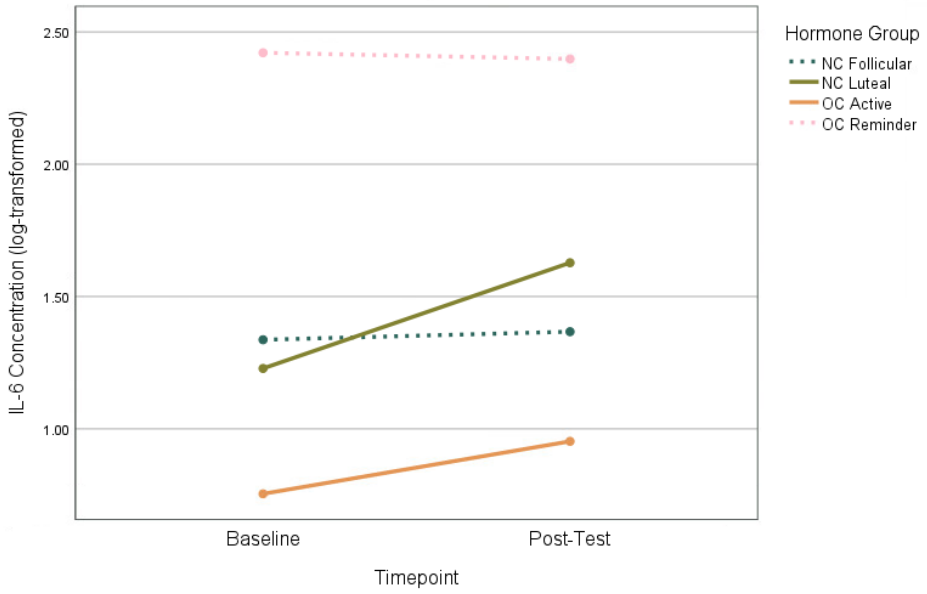
Differences in IL-6 Reactivity Across Hormone Groups.

A repeated-measures ANCOVA tested whether IL-6 concentrations from baseline to post-test varied by hormone group and condition (control, experimental), while controlling for self-reported recent illness. The analytic sample included 209 participants who were classified into a hormone group (NC Follicular, NC Luteal, OC Active, OC Reminder) and had valid data at both IL-6 timepoints. No main effects of condition ($F(1, 200) = .001$, $p = .975$) or timepoint ($F(1, 200) = 1.63$, $p = .204$) emerged. There were also no significant interactions involving timepoint, condition, or hormone group (all ps > .171). However, a significant main effect of hormone group was observed ($F(3, 200) = 4.00$, $p = .009$, $\eta^2 = .06$). Bonferroni-corrected pairwise comparisons indicated that OC active users had significantly lower IL-6 ($M = .854$, $SE = .27$, 95% CI [.32, 1.39]) than OC reminder users ($M = 2.41$, $SE = .36$, 95% CI [1.70, 3.12], $p = .004$). No other pairwise comparisons were significant (all ps > .053).

Although the hormone group × timepoint interaction was not significant ($p = .251$), planned comparisons were conducted to examine specific group differences across timepoints. At baseline, OC reminder participants ($M = 2.42$, $SE = .37$, 95% CI [1.69, 3.16]) had higher IL-6 than OC active participants ($M = .75$, $SE = .28$, 95% CI [.20, 1.31]; $p = .003$) and NC luteal participants ($M = 1.23$, $SE = .20$, 95% CI [1.21, 2.05]; $p = .031$). At post-test, the difference between OC active and OC reminder participants remained significant ($M_{active} = .95$, $SE_{active} = .30$, 95% CI_{active} [.36, 1.55]; $M_{reminder} = 2.40$, $SE_{reminder} = .40$, 95% $CI_{reminder}$ [1.62, 3.18]; $p = .024$). This pattern is visualized in Figure 3.6.

Figure 3.6

IL-6 Concentrations Across Timepoints by Hormone Group.



Note. Points represent estimated marginal means of log-transformed IL-6 at baseline and post-test. Lines connect values within each hormone group (NC follicular [dotted teal], NC luteal [solid green], OC active [solid orange], OC reminder [dotted pink]). Abbreviations: NC = naturally cycling, OC = oral contraceptive.

Among the NC groups, only the luteal-phase participants in the control condition showed a significant within-group increase in IL-6 from baseline (M = 1.15, SE = .30, 95% CI [.56, 1.74]) to post-test (M = 1.88, SE = .32, 95% CI [1.26, 2.50]); $p < .001$). No other within-group increases were observed (all ps > .251).

Moderation by Hormonal Contexts

Having established differences in baseline IL-6 concentrations across hormone groups, subsequent analyses tested whether hormonal context also moderated the compensatory BIS–IL-6 pathway. Moderated mediation models (PROCESS Model 59;

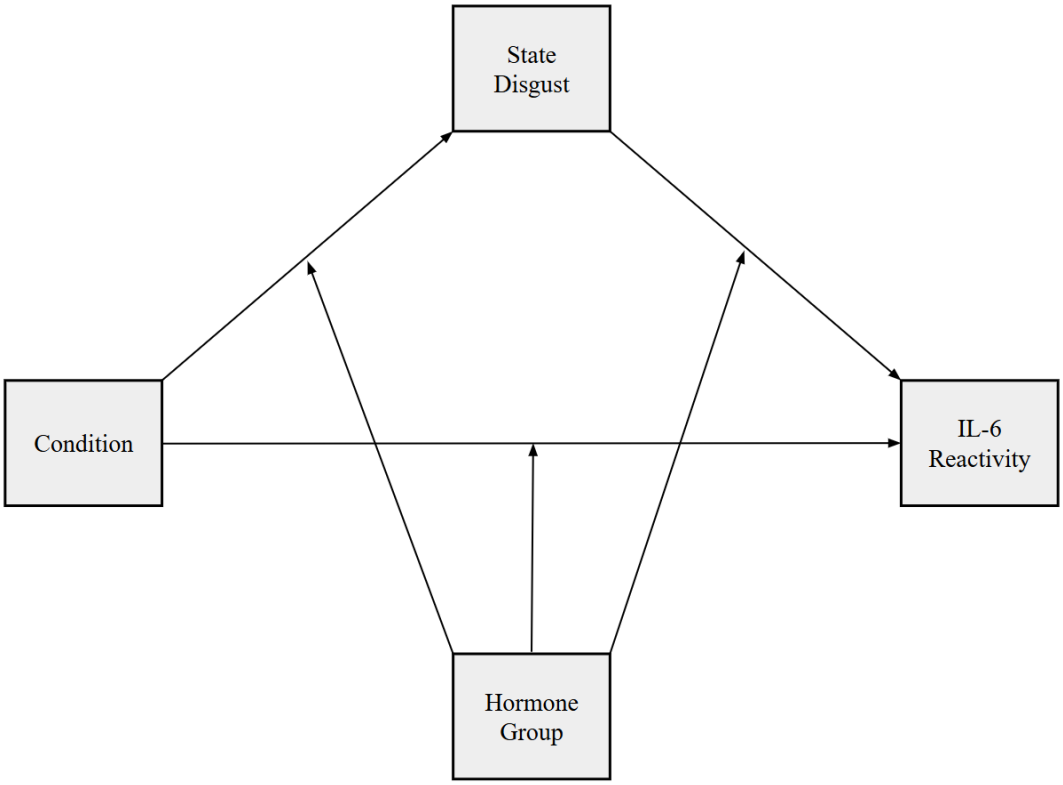
Hayes, 2022) were used to test whether the indirect effect of condition (X) on ΔIL-6 (Y) through state disgust (M) varied as a function of hormone group (w). To improve interpretability and preserve statistical power, participants were grouped by endogenous versus exogenous hormonal context, with separate analyses conducted for NC participants (follicular vs. luteal phases) and OC participants (active pill vs. reminder pill phases). Because PI did not significantly moderate the BIS–IL-6 pathway in the preceding analysis, but was associated with overall levels of state disgust, PI was retained as a covariate alongside recent illness. Analyses were limited to participants with valid data for both IL-6 timepoints and state disgust (NC analytic sample n = 140; OC analytic sample n = 53; see Figure 3.7).

Naturally Cycling Participants (Follicular vs. Luteal Phase)

Multicollinearity was acceptable (all VIFs < 1.97). To avoid redundancy, reported results below focus on tests of moderation by cycle phase and the conditional indirect effects. Main effects were consistent with prior models (see "Moderation of the BIS–IL-6 Pathway by Trait Perceived Infectability"), and PI significantly predicted greater disgust (M_c = 4.13, SE = 2.01, t = 2.06, p = .042, 95% CI [.16, 8.10]). Cycle phase did not moderate the effect of the manipulation on state disgust (a_w = 1.34, SE = 8.21, t = .16, p = .870, 95% CI [-14.89, 17.58]), indicating that pathogen cues increased disgust similarly in follicular and luteal phase participants. The direct effect of condition on ΔIL-6 was also not significant (c' = .22, SE = .85, t = .26, p = .796, 95% CI [-1.45, 1.89]). However, cycle phase significantly moderated the association between state disgust and ΔIL-6 (b_w = -0.02, SE = .01, t = -2.44, p = .016, 95% CI [-0.04, -0.004]; see Figure 3.8).

Figure 3.7

Conceptual Moderated Mediation Model of the Effect of Experimental Condition on IL-6 Reactivity via State Disgust, With Natural Cycle Phase as a Moderator.

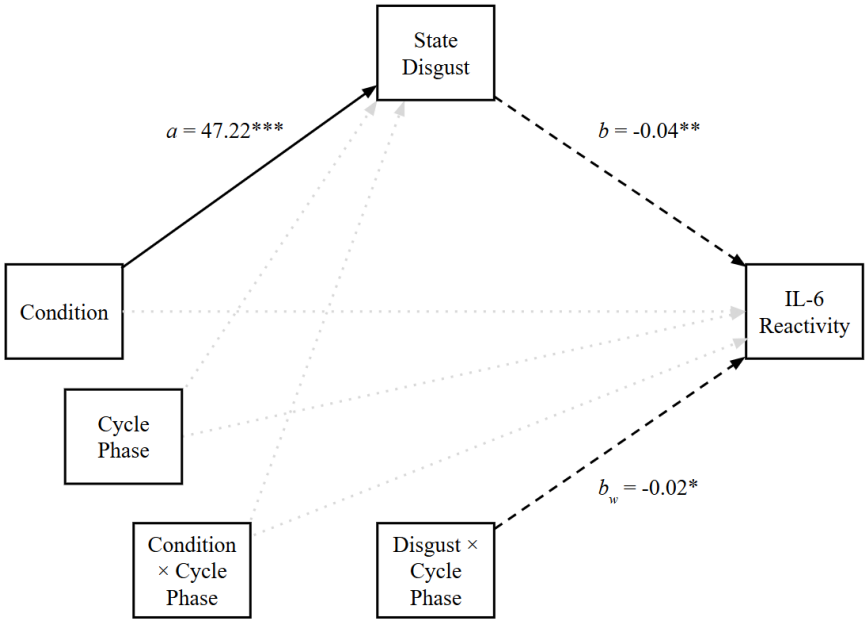


Note. The conceptual model illustrates hormone group (NC follicular vs. NC luteal, OC active vs. OC reminder) as a moderator of the indirect effect of experimental condition on IL-6 reactivity via state disgust.

Abbreviations: NC = naturally cycling, OC = oral contraceptive.

Figure 3.8

Statistical Moderated Mediation Model of the Effect of Experimental Condition on IL-6 Reactivity via State Disgust, With Natural Cycle Phase as a Moderator.



Note: Values represent unstandardized regression coefficients from the moderated mediation model. NC cycle phase includes follicular and luteal groups. Solid black lines indicate significant positive paths, dashed black lines indicate significant negative paths, and dotted grey lines indicate nonsignificant paths.

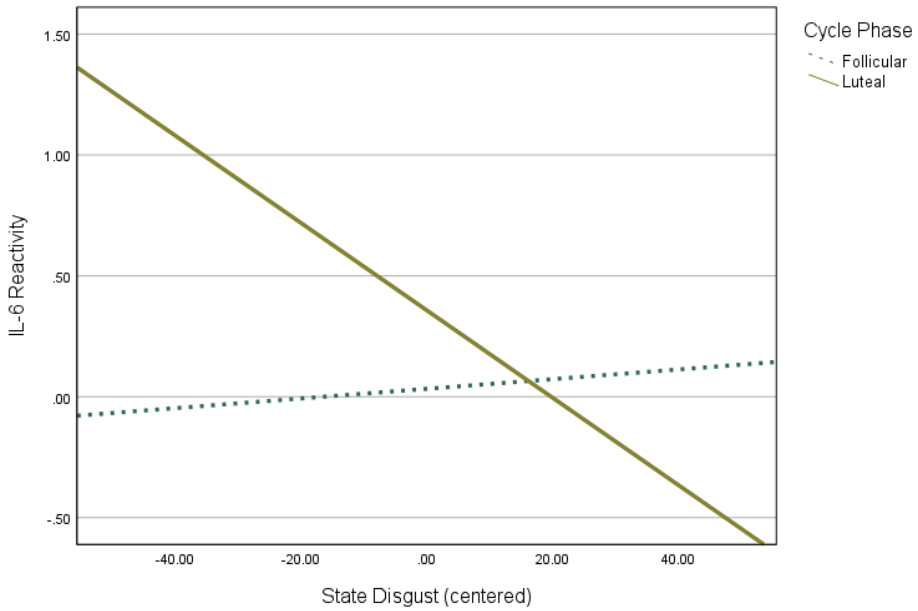
Abbreviations: NC = naturally cycling.

****p* < .001, ***p*< .01, **p* < .05*

Simple slopes analyses indicated that state disgust was unrelated to ΔIL-6 among follicular phase participants ($b_{w\_follicular}$ = .002, *SE* = .01, *t* = .38, *p* = .704, 95% CI [-0.01, .01]), but predicted lower ΔIL-6 among luteal phase participants (b_{w_luteal} = -0.02, *SE* = .01, *t* = -2.90, *p* = .004, 95% CI [-0.03, -0.01]). This is visualized in Figure 3.9.

Figure 3.9

Interaction Between State Disgust and Cycle Phase on IL-6 Reactivity in Naturally Cycling Participants.

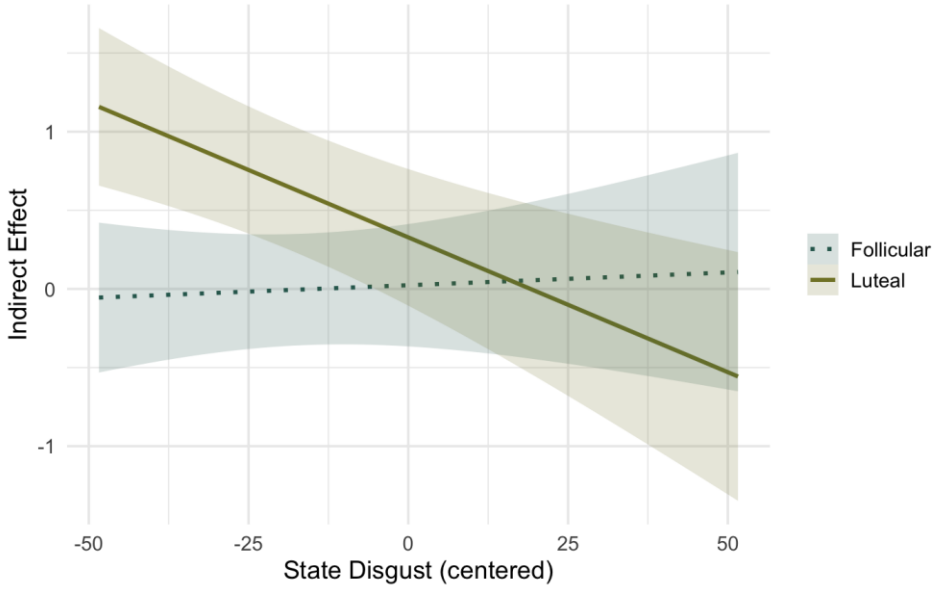


Note. Lines represent simple slopes of the association between state disgust and IL-6 reactivity for follicular (dashed blue line) and luteal (solid green line) NCs. Values reflect predicted IL-6 reactivity across the observed range of mean-centered state disgust. Abbreviations: NC = naturally cycling.

Consistent with this pattern, the indirect effect of condition on ΔIL-6 through state disgust was nonsignificant for follicular phase participants ($ab_{follicular}$ = .09, *BootSE* = .23, 95% CI [-0.37, .53]), but was significant and negative for luteal phase participants (ab_{luteal} = -0.83, *BootSE* = .31, 95% CI [-1.59, -0.33]). The index of moderated mediation was significant (ab_w = -0.92, *BootSE* = .39, 95% CI [-1.78, -0.25]). The indirect effect became increasingly negative at higher levels of state disgust in the luteal phase, whereas it remained near zero across levels of state disgust in the follicular phase (see Figure 3.10).

Figure 3.10

Conditional Indirect Effect of Experimental Condition on IL-6 Reactivity via State Disgust, Moderated by Natural Cycle Phase.



Note. Lines represent conditional indirect effects (*ab*) of the pathogen-salient condition on IL-6 reactivity via state disgust for follicular (dotted blue line) and luteal (solid green line) NC participants. Shaded bands represent 95% confidence intervals derived from bootstrap resampling.

Abbreviations: NC = naturally cycling.

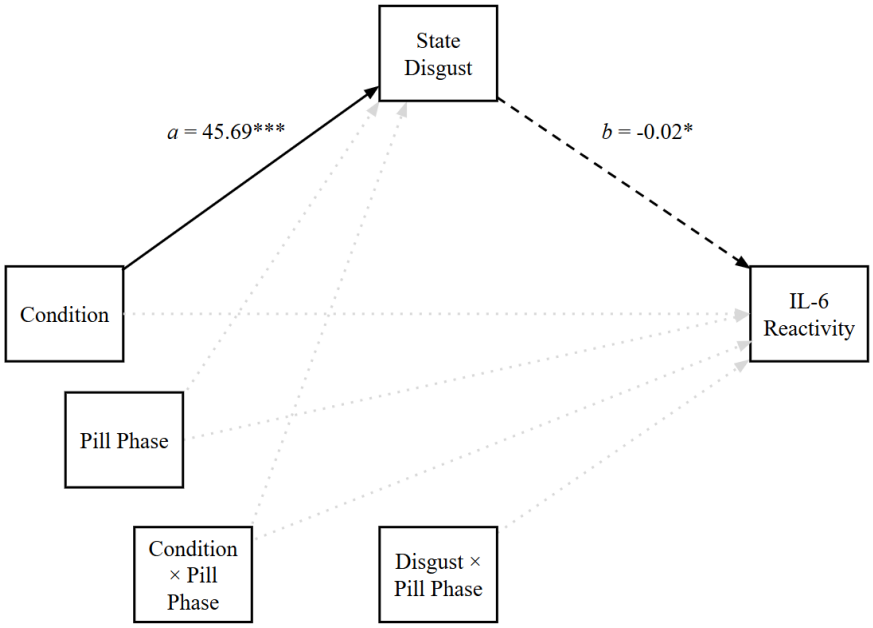
Together, these findings indicate that among NC participants, the compensatory BIS–IL-6 pathway was present in the luteal phase, but not the follicular phase.

Oral Contraceptive Users (Active vs. Reminder Pill Phase)

To avoid redundancy, reported results below focus on tests of moderation by pill phase and the conditional indirect effects (see Figure 3.11). Main effects were consistent with prior models. Multicollinearity was acceptable (all VIFs < 2.91).

Figure 3.11

Statistical Moderated Mediation Model of the Effect of Experimental Condition on IL-6 Reactivity via State Disgust, With Pill Phase as a Moderator.



Note. Values represent unstandardized regression coefficients. Pill phase includes OC active and OC reminder groups. Solid black lines indicate significant positive paths, dashed black lines indicate significant negative paths, and dotted grey lines indicate nonsignificant paths.

Abbreviations: OC = oral contraceptive

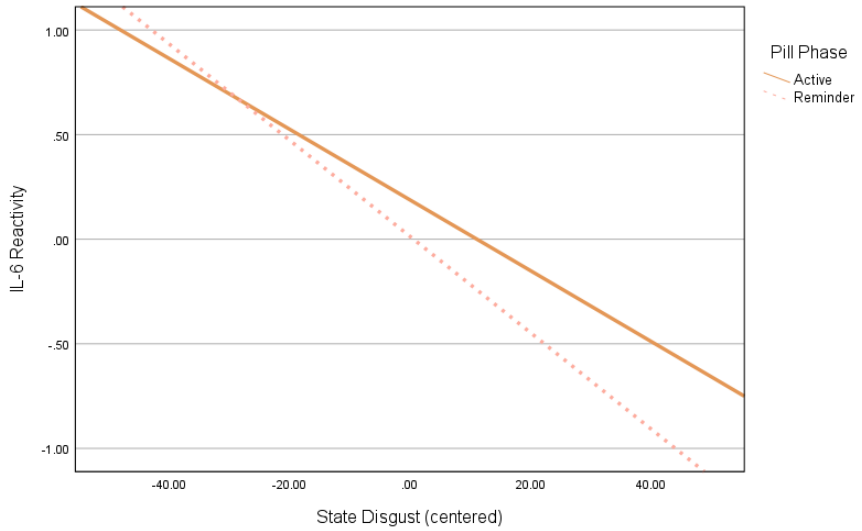
****p* < .001, **p* < .05

In contrast to NC participants, PI was not associated with state disgust among OC users (p = .751). Pill phase did not moderate the effect of the manipulation on disgust (a_w = -15.01, SE = 13.54, t = -1.11, p = .273, 95% CI [-42.24, 12.22]), indicating that pathogen cues increased disgust similarly among active and reminder phase OC users.

The direct effect of condition on ΔIL-6 was also not moderated by pill phase (c'_w = -0.60, *SE* = 1.11, *t* = -0.54, *p* = .590, 95% CI [-2.83, 1.63]). Contrary to NC participants, hormone group (i.e., pill phase) did not moderate the effect of state disgust on ΔIL-6 (b_w = .01, *SE* = .02, *t* = .34, *p* = .737, 95% CI [-0.03, .04]). Simple slopes analyses indicated a nonsignificant trend among reminder pill phase users such that greater state disgust was descriptively associated with lower IL-6 reactivity (b_w = -0.02, *SE* = .01, *t* = -1.69, *p* = .056, 95% CI [-0.04, .001]). There was no corresponding significant association or trend among active pill phase participants (b_w = -0.02, *SE* = .01, *t* = -1.49, *p* = .144, 95% CI [-0.04, .01]; see Figure 3.12).

Figure 3.12

Interaction Between State Disgust and Pill Phase on IL-6 Reactivity.



Note. Lines represent simple slopes of the association between state disgust and IL-6 reactivity for active (solid orange line) and reminder pill (dashed pink line) phase participants. Values reflect predicted IL-6 reactivity across the observed range of mean-centered state disgust.

The indirect effect of condition on ΔIL-6 through state disgust was significant and negative for active phase OC users (ab_{active} = -0.68, *BootSE* = .57, 95% CI [-2.29, -0.06]), and showed a similar negative pattern for reminder phase users ($ab_{reminder}$ = -1.27, *BootSE* = .87, 95% CI [-4.36, 0.020). The index of moderated mediation was not significant (ab_{w} = .59, *BootSE* = 1.04, 95% CI [-1.11, 3.53]), indicating that the strength of the indirect pathway did not differ significantly by pill phase. The indirect effect became increasingly negative at higher levels of state disgust in both pill phases (see Figure 3.13).

Thus, unlike the phase-specific pattern observed among NC participants, OC users as a group showed a comparable negative indirect pathway across pill phases, indicating that OC pill phase did not significantly alter the compensatory BIS–IL-6 pathway.

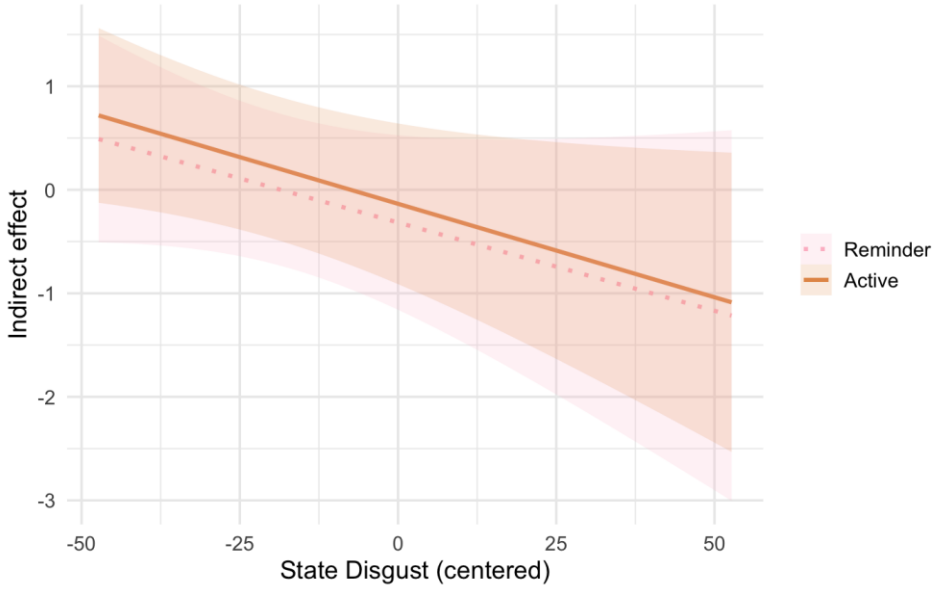
Oral Contraceptive Users (Progestogenic Activity as a Continuous Predictor)

To more precisely capture variability in OC formulation beyond categorical pill phase, formulation-specific progestogenic activity (M) was included as a continuous moderator of the same BIS–IL-6 pathway. Reminder phase participants were coded as 0 progestogenic activity, reflecting the absence of exogenous progestin during that phase. Analyses controlled for PI and recent illness and were limited to OC users with valid data for both IL-6 timepoints (n = 53)[1]. Multicollinearity was acceptable (all VIFs < 2.96).

[1] A parallel moderated mediation model restricted to active phase OC users only (n = 35) was also estimated to examine whether including reminder phase participants coded as 0 progestogenic activity altered the findings. The same general pattern emerged. However, within this smaller active phase subsample, the state disgust → ΔIL-6 path was no longer significant, and confidence intervals around the conditional indirect effects were wider, although estimates remained in the same direction. These differences are likely due to the smaller analytic sample size.

Figure 3.13

Conditional Indirect Effect of Experimental Condition on IL-6 Reactivity via State Disgust, Moderated by Pill Phase.

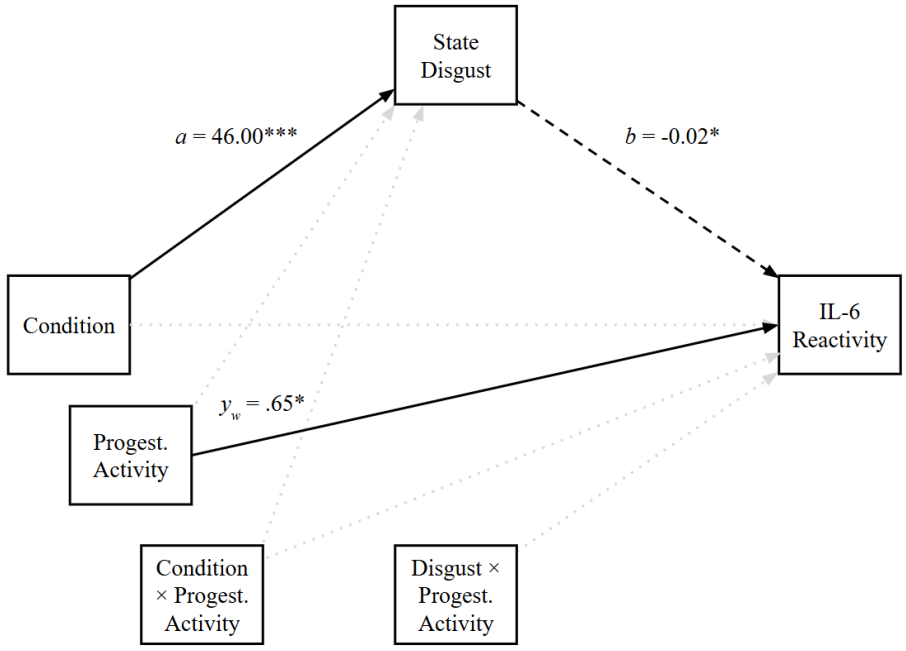


Note. Lines represent conditional indirect effects (*ab*) of the pathogen-salient condition on IL-6 reactivity via state disgust for active (solid orange line) and reminder (dotted pink line) pill phase participants. Shaded bands represent 95% confidence intervals (CIs) derived from bootstrap resampling.

The manipulation significantly increased disgust (a = 46.00, SE = 7.06, t = 6.52, p < .001, 95% CI [31.80, 60.20]). Greater disgust, in turn, significantly predicted lower ΔIL-6 (b = -0.02, SE = .01, t = -2.25, p = .029, 95% CI [-0.04, -0.002]). Although progestogenic activity did not significantly moderate the disgust → ΔIL-6 association, it was directly associated with ΔIL-6 (y_w = .65, SE = .31, t = 2.10, p = .041, 95% CI [.03, 1.27]), such that higher progestogenic activity predicted greater IL-6 reactivity. This pattern is visualized in Figure 3.14.

Figure 3.14

Statistical Moderated Mediation Model of the Effect of Experimental Condition on IL-6 Reactivity via State Disgust, With Progestogenic Activity as a Moderator.

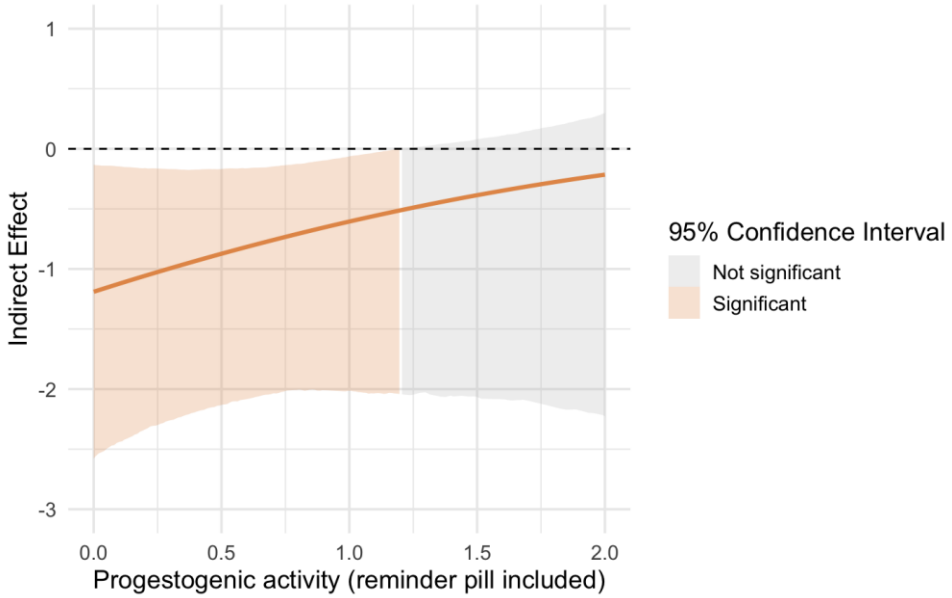


Note: Values represent unstandardized regression coefficients from the moderated mediation model. Solid black lines indicate significant positive paths, dashed black lines indicate significant negative paths, and dotted grey lines indicate nonsignificant paths. **p < .05, ***p < .001*

Conditional indirect effects indicated that the indirect effect of condition on ΔIL-6 through state disgust was significant at low (ab_w = -1.62, *BootSE* = .95, 95% CI [-4.48, -0.42]) and moderate (ab_w = -1.17, *BootSE* = .65, 95% CI [-2.89, -0.33]) levels of progestogenic activity, but not at high levels (ab_w = -0.48, *BootSE* = .63, 95% CI [-2.71, .05]). Thus, the negative indirect pathway was strongest at lower levels of progestogenic activity and diminished as progestogenic activity increased (Figure 3.15).

Figure 3.15

Conditional Indirect Effect of Experimental Condition on IL-6 Reactivity via State Disgust, Moderated by Progestogenic Activity.



Note. Line represents the estimated indirect effect (*ab*) across levels of progestogenic activity. Shaded regions reflect 95% confidence intervals (CIs) derived from bootstrap resampling. Orange shading indicates values where the indirect effect is statistically significant, and gray shading indicates nonsignificant values.

Within-Subject Changes Across OC Phases

Mean-Level Differences in BIS and IL-6 Outcomes Across Pill Phases

To examine whether BIS–IL-6 patterns varied across women's own shifting pill phases, within-subjects analyses were conducted among OC users who completed both active and reminder phase sessions. Of the 62 OC participants who completed both sessions, 4 were missing data for both IL-6 timepoints in Session 2, 5 had invalid IL-6 data (i.e., extreme at both timepoints) for at least one session (3 from Session 1, 2 from

Session 2), 1 was missing data for Session 2 baseline IL-6 only, and 1 was missing data for Session 2 posttest only. Two additional participants were missing state disgust data, one of whom was also missing IL-6 data at both Session 2 timepoints. Finally, one participant was missing sufficient data to determine the progestogenic activity of her OC formulation. As a result, analytic sample sizes vary depending on the variables included.

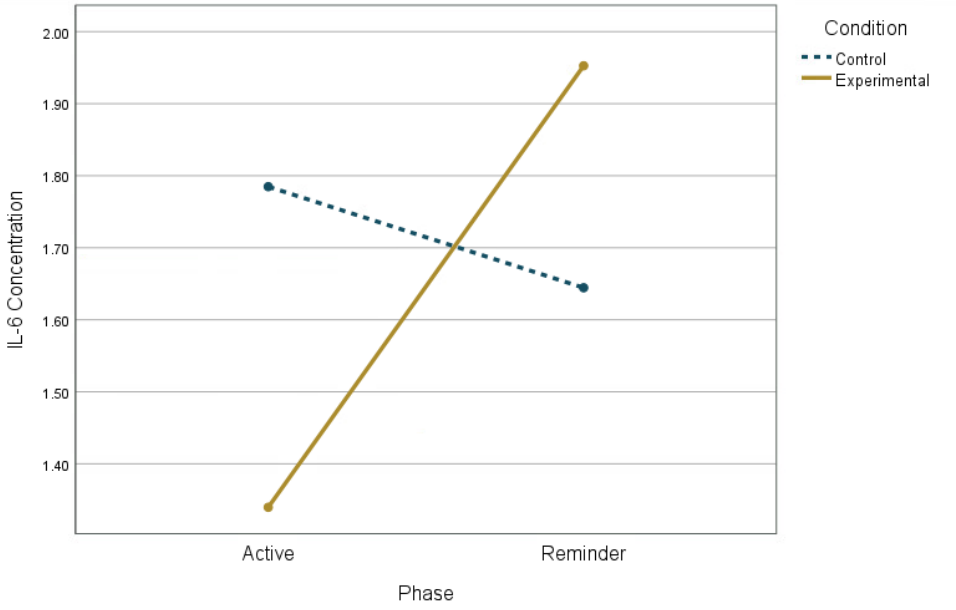
Paired-samples *t*-tests first examined within-person differences across pill phases. No significant phase differences were observed for baseline IL-6 ($t(56) = -1.36$, $p = .179$), IL-6 reactivity ($t(50) = -0.47$, $p = .644$), state disgust ($t(56) = -1.83$, $p = .072$), or SPA ($t(60) = -0.26$, $p = .795$). Thus, simple mean-level comparisons did not indicate reliable shifts in BIS or IL-6 outcomes across women's own pill phases.

Within-Subject Differences in IL-6 Across Pill Phases

Next, a repeated-measures ANCOVA tested whether IL-6 concentrations from baseline to post-test varied across pill phase as a function of experimental condition. Condition (control, experimental) was entered as a between-subjects factor, with IL-6 timepoint (baseline, post-test) and pill phase (active, reminder) entered as within-subject factors. Session 1 PI and recent illness at Sessions 1 and 2 were included as covariates. Analyses were limited to participants with valid IL-6 data at both timepoints during both sessions ($n = 51$). A significant phase × condition interaction emerged ($F(1, 46) = 4.12$, $p = .048$, $\eta^2 = .08$; Figure 3.16). No other main effects (all ps > .528) or interactions (all ps > .496) were significant. Pairwise comparisons indicated that among participants in the experimental condition, IL-6 levels were significantly lower during the active pill phase ($M = 1.34$, $SE = .35$, 95% CI [.63, 2.05]) than during the reminder pill phase ($M = 1.95$, $SE = .32$, 95% CI [1.31, 2.60]; $p = .016$).

Figure 3.16

Interaction Between Experimental Condition and Pill Phase on IL-6 Concentration.

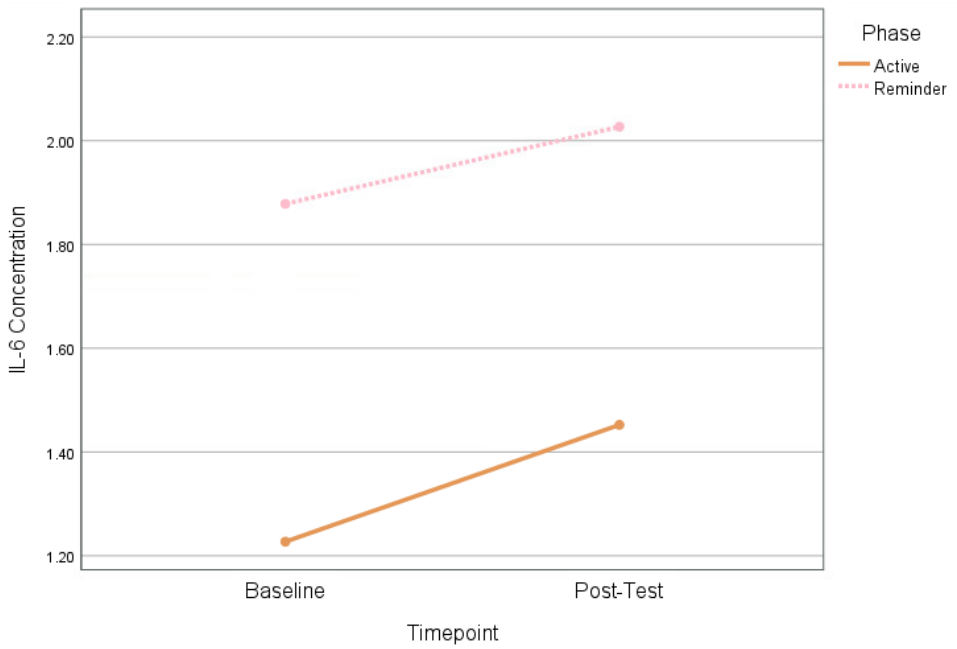


Note. Points represent estimated marginal means of IL-6 concentrations for active and reminder oral contraceptive pill phases within each condition. Lines connect values within the control (dotted blue) and experimental (solid gold) conditions.

Planned comparisons indicated that this phase difference was significant at baseline (mean difference = .65; p = .035) and near-significant at post-test (mean difference = .58, p = .051), but only among participants in the experimental condition (control condition: ps > .371). Thus, consistent with the between-subject findings, IL-6 tended to be higher during the reminder pill phase than the active pill phase, with the clearest within-person differences emerging following exposure to pathogen cues (Figure 3.17).

Figure 3.17

IL-6 Concentrations From Baseline to Post-Test by Pill Phase in the Experimental Condition.



Note. Points represent estimated marginal means of IL-6 concentrations at baseline and post-test for active (solid orange) and reminder (dotted pink) pill phases within the experimental condition. Lines connect values across timepoints within each pill phase.

To validate these findings and structure a modeling framework that could be extended to BIS outcomes, a LMM was estimated. LMMs allow repeated observations to be modeled directly while accommodating correlated data and missing data more flexibly than the repeated-measures ANCOVA. Because the sample of OC users with complete IL-6 data across all four timepoints was relatively small (n = 51), more complex random-effects structures (e.g., with random slopes for phase and/or timepoint) frequently failed to converge and yielded invalid solutions, suggesting insufficient power to support these

complex estimations. Accordingly, the final LMM model reported here balances capturing the within-subject correlation with achieving stable estimates.

This final model specification included: (1) fixed effects for pill phase, timepoint, and their interaction, which tested average differences in IL-6 across these groupings; (2) random intercepts for participant, which accounted for between-subject variance in baseline IL-6; and (3) a repeated-measures structure specified at the participant × phase level, which modeled the correlation between baseline and post-test IL-6 within each phase. Thus, the model nested timepoints (baseline, post-test) within OC phases (active, reminder) within participants. The model was estimated using the restricted maximum likelihood (REML) method to ascertain less biased estimates of variance components. The model fit indices indicated acceptable overall fit (AIC = 757.39, BIC = 767.62).

Results were consistent with the repeated-measure ANCOVA. Neither the main effect of timepoint ($F(1, 107.71) = 1.27$, $p = .263$) nor the phase × timepoint interaction ($F(1, 107.71) = 1.05$, $p = .307$) was significant. However, a significant main effect of phase emerged ($F(1, 53.85) = 4.20$, $p = .045$). Pairwise comparisons indicated higher baseline IL-6 during the reminder pill phase ($M = 1.78$, $SE = .25$) than during the active pill phase ($M = 1.27$, $SE = .25$, $p = .024$).

BIS Predictors of Within-Subject IL-6 Reactivity Across Pill Phases

To test whether BIS responses predicted IL-6 reactivity within individuals across their OC phases, and whether these associations differed by experimental condition, an LMM was estimated. Fixed effects included pill phase (active, reminder), timepoint (baseline, post-test), condition (control, experimental), and the time-varying BIS predictors: state disgust, SPA, and PI. All two-way and higher-order interactions among

phase, timepoint, condition, and the BIS predictors were included. All other model parameters were identical to the previous LMM. Model fit was acceptable (AIC = 763.24, BIC = 772.99).

No significant main effects emerged for phase (p = .469), timepoint (p = .086), condition (p = .567), or the BIS predictors individually (all ps > .355). A significant timepoint × SPA interaction was observed (F(1, 97.49) = 5.37, p = .023), indicating that the association between SPA and IL-6 differed across baseline and post-test timepoints. However, the timepoint × SPA × condition interaction was nonsignificant (F(1, 97.44) = 2.44, p = .121), suggesting that this pattern did not differ between the control and experimental conditions. No other interactions were significant (all ps > .121).

Estimated marginal means indicate that when SPA was low (-1 *SD*), IL-6 increased significantly from baseline (M = 1.38, SE = .37) to post-test (M = 1.81, SE = .37; p = .016). In contrast, when SPA was high (+1 *SD*), IL-6 did not significantly change across timepoints ($M_{baseline}$ = 1.47, $SE_{baseline}$ = .37; M_{post} = 1.33, SE_{post} = .37; p = .383).

Discussion

Study 2 extends the investigation of compensatory prophylaxis by directly assessing both behavioral and physiological immune responses to pathogen cues across NC and OC hormonal contexts. Building on Study 1, which showed that hormonal context and PI shape who becomes emotionally reactive to pathogen cues, the present study tested whether those affective responses also correspond with measurable inflammatory change. By integrating a pathogen threat manipulation with pre- and post-manipulation IL-6 measurements and both state and trait measures of BIS sensitivity, Study 2 examined whether the behavioral and physiological immune systems operate in

complementary or compensatory ways, and whether this coordination varies across endogenous and exogenous hormonal contexts. It was hypothesized that pathogen exposure would increase disgust, pathogen avoidance motivations, and IL-6 reactivity (i.e., complementary activation), but that during hormonally immunomodulated states (i.e., NC luteal and OC active phases), behavioral reactivity would compensate for reduced inflammatory reactivity. Greater progestogenic activity was further expected to amplify this pattern.

Contrary to expectations, complementary activation was not supported. Neither direct pathogen exposure nor behavioral immune activation predicted heightened IL-6 reactivity. Instead, this pattern aligns most closely with compensatory frameworks of BIS–immune coordination (e.g., Bradshaw et al., 2022; Gassen et al., 2018; Oaten et al., 2017). Across participants, pathogen cues increased state disgust, and greater disgust was associated with two simultaneous outcomes: stronger pathogen avoidance motivations and reduced IL-6 reactivity. Thus, the same emotional response that promoted behavioral avoidance also corresponded with attenuated inflammatory change. Disgust appeared to link pathogen detection with both behavioral avoidance and reduced inflammatory reactivity. These findings suggest that disgust may function as a coordinating mechanism through which pathogen cues motivate low-cost behavioral defenses while simultaneously reducing reliance on–or arguably the need for–inflammatory activation.

Although prior experimental work has reported complementary co-activation under pathogen threat (e.g., Stevenson et al., 2011, 2015), complementary and compensatory dynamics may depend on the strength and meaning of the pathogen cue. In the current paradigm, there was substantial variability in disgust within each condition,

which suggests that participants differed in how strongly they interpreted the stimuli as pathogen-relevant (see Chapter 2 Discussion for a consideration of the experimental paradigm used here). Under moderately salient pathogen cues, behavioral avoidance may be sufficient without complementary inflammatory upregulation. In this context, the absence of complementary activation does not necessarily contradict prior findings (e.g., Ersche et al., 2014; Gassen et al., 2019; Miller & Maner, 2011; Schaller et al., 2010; Stevenson et al., 2011, 2012, 2015), but instead suggests that BIS–immune coordination may shift as a function of stimulus intensity, stimulus interpretation, and/or internal vulnerability signals.

The compensatory relationship between disgust and IL-6 was broadly evident in the current study, but individual differences shaped when this pattern emerged most clearly. Consistent with Study 1, participants higher in PI reported greater disgust in response to pathogen cues. Although PI did not significantly moderate the disgust–IL-6 pathway, conditional effects indicated that the negative indirect effect was most robust at average-to-higher levels of PI. Thus, perceived vulnerability may be especially relevant for determining when pathogen cues elicit affective responses that correspond with reduced inflammatory reactivity. This interpretation is consistent with functional flexibility accounts (reviewed in Schaller at al., 2007; Schaller & Park, 2011), which propose that pathogen avoidance responses are calibrated by internal vulnerability cues and changing environmental demands. It also aligns with evidence that complementary and compensatory dynamics may not be mutually exclusive systems. For example, Keller and colleagues (2022) found that watching pathogen-related videos increased salivary sIgA, suggesting rapid immune preparation. However, individuals higher in

contamination disgust showed smaller increases in sIgA. Together with the present findings, this suggests that BIS–immune coordination may depend on multiple factors, including salience of the pathogen cue, trait individual vulnerabilities, situational responses, and the specific immune pathway assessed.

Hormonal context further shaped compensatory responding. Among NC participants, cycle phase did not influence baseline IL-6 levels. However, cycle phase did significantly moderate the relationship between disgust and inflammatory reactivity. A compensatory BIS–IL-6 relationship emerged during the luteal phase, when endogenous progesterone is elevated, but not during the follicular phase, when endogenous progesterone is low. Importantly, the manipulation evoked comparable levels of disgust across cycle phases, suggesting that these effects reflected differences in how disgust translated into inflammatory change, not differences in emotional reactivity overall. During the luteal phase, greater disgust predicted lower IL-6 reactivity, whereas no such association emerged during the follicular phase. This pattern is consistent with the CPH, in that compensatory dynamics were most evident in the luteal phase when physiological immunity is presumed to be hormonally constrained.

Among OC users, baseline inflammation showed a different pattern. IL-6 was lowest during the active pill phase and highest during the reminder phase, both between participants and within the same women across sessions. This finding is consistent with the anti-inflammatory properties of progestin (e.g., Hapgood et al., 2018; Reis et al., 2020), suggesting that synthetic hormones meaningfully alter inflammatory set points. However, unlike NC participants, pill phase did not significantly moderate the compensatory disgust–IL-6 association. Across both active and reminder phases, stronger

disgust corresponded with lower IL-6 reactivity. Thus, while exogenous progestins appeared to shift baseline inflammatory activity, they did not meaningfully alter the compensatory disgust–IL-6 pattern.

On the surface, the OC participant results may seem inconsistent with the NC participant results. Among NC participants, compensatory BIS–IL-6 coordination emerged specifically during the luteal phase when endogenous progesterone is elevated, whereas among OC users the same compensatory pattern persisted even during the low-hormone reminder phase. However, it is worth noting that the reminder phase in OC users is not physiologically equivalent to the low-progesterone follicular phase in NC women. One interpretation is that once synthetic progestins alter inflammatory set points, the BIS continues to calibrate to this immune environment, allowing compensatory coordination to persist even during the brief hormone withdrawal phase. The BIS, under this view, may be less sensitive to short-term pill-phase changes than to endogenous hormonal fluctuations across the natural ovulatory cycle. Residual receptor-level effects may also prolong immunomodulatory consequences beyond the active pill phase. Progestins can bind to progesterone and glucocorticoid receptors, and downstream transcriptional effects may persist into the reminder pill phase such that the inflammatory environment may not fully return to baseline during synthetic hormone withdrawal. More broadly, synthetic progestins differ from endogenous progesterone in receptor affinity and pharmacokinetics (e.g., Grøntvedt et al., 2017; reviewed in Africander et al., 2011), and prolonged OC use may produce a more stable endocrine and inflammatory environment across pill phases. In other words, whereas NC women experience dynamic within-cycle hormone fluctuations that may alter compensatory BIS-immune

coordination, OC users may instead exhibit a more chronically recalibrated immune environment in which compensatory coordination remains relatively stable across pill phases. Although these interpretations are speculative, exogenous hormonal modulation may shift baseline inflammation without necessarily altering how disgust relates to IL-6 reactivity. However, these possibilities should be investigated in future work using larger samples and directly measured hormone levels across multiple sessions among naturally cycling participants.

Considering progestins and their doses uniquely may provide researchers with more opportunity to identify formulation-specific effects. When progestogenic activity was modeled continuously, potency did not significantly moderate the compensatory pathway. However, the indirect effect was strongest at low-to-moderate levels of progestogenic activity and weakened at higher levels. These findings should be interpreted cautiously given the modest OC subsample size. Nevertheless, it is possible that stronger progestogenic formulations may dampen the disgust-induced compensatory pathway, perhaps by further constraining inflammatory reactivity or by influencing immune processes that are not fully captured by salivary IL-6. Future work with larger samples, pharmacologically diverse OCs, and additional inflammatory markers is needed to clarify whether progestin potency shapes BIS–immune calibration in a dose-dependent manner.

Limitations and Future Directions

Study 2 relied on IL-6 as the sole marker of immunological reactivity. Although IL-6 is widely used in psychological research examining the interaction between behavioral and physiological immune responses (e.g., Cepon-Robins et al., 2021; Ersche

et al., 2014; Gassen et al., 2018; Juran et al., 2023; Schaller et al., 2010; reviewed in Murray et al., 2019), its pleiotropic nature complicates interpretation. IL-6 contributes to inflammatory signaling during infection (reviewed in Gruys et al., 2005), but it is also involved in tissue repair and the resolution of inflammation (reviewed in Fuster & Walsh, 2014). Consequently, increases or decreases in IL-6 cannot be interpreted as simple indicators of "more" or "less" immune activation without additional context. Indeed, IL-6 dynamics reflect complex regulatory processes, and elevated IL-6 levels do not necessarily correspond to chronic low-grade inflammation (see Del Giudice & Gangestad, 2018).

The inclusion of baseline and post-test IL-6 measurements strengthens interpretation by focusing on within-person change rather than absolute levels. However, between-session differences in IL-6 may also reflect broader physiological processes beyond acute responses to pathogen cues. Although within-person comparisons are informative for testing compensatory prophylaxis, future research incorporating multiple inflammatory markers across repeated longitudinal sessions for both OC users and NC participants would allow for more precise characterization of how physiological immune activity relates to BIS reactivity across hormonal conditions.

Timing variability between the pathogen manipulation and providing the saliva sample is an additional limitation. Salivary IL-6 responses in stress paradigms typically peak approximately 30-45 minutes following exposure to the stressor and can remain elevated for up to 90 minutes (Izawa et al., 2013). In contrast, Stevenson and colleagues (2011, 2012) demonstrated rapid changes in other oral immunomarkers (i.e., sIgA, albumin, TNF-α) that emerged within approximately 10 minutes following exposure to

contamination cues. These findings suggest that localized mucosal immune responses may precede changes in systemic cytokines. Thus, the absence of complementary IL-6 elevation shortly after disgust induction does not rule out complementary responding in other immune processes. It is possible that patterns of complementary versus compensatory activity may depend, in part, on the timing and biomarker assessed. Future studies incorporating both rapid mucosal indicators and slower systemic markers would allow for clearer tests of when complementary versus compensatory responding occurs.

Sample sizes also limit interpretations, particularly within OC phase groups. Analyses examining progestogenic activity involved relatively small numbers of active-phase users, limiting statistical power to detect moderation effects and increasing the risk of Type II error. Although analyses were repeated with participants in their reminder phase (with exogenous progestin levels set to zero), these analyses were also based on modest group sizes. Consequently, findings regarding dose-dependent effects of progestogenic activity should be interpreted cautiously and, as above, replication with larger and more pharmacologically diverse samples is necessary to better understand whether and how progestin potency meaningfully shapes compensatory versus complementary responding.

Additionally, Study 2 excluded users of non-oral hormonal contraceptive methods (e.g., hormonal IUDs, injectables), potentially overlooking distinct immunomodulatory effects associated with these formulations. For example, injectable contraceptives such as Depo Provera (MPA) have been associated with immunosuppressive effects (e.g., Hapgood et al., 2018), which may alter the relationship between hormonal status, IL-6 reactivity, and BIS activation. Including non-oral HC users in future research would

allow for a more comprehensive test of how different forms of progestin exposure shape physiological and behavioral immune responses.

Finally, immune reactivity is influenced by numerous endogenous and exogenous factors, including stress, sleep, diet, chronic inflammation, and subclinical infection. Although participants were screened for recent illness and relevant covariates were included where possible, unmeasured variability likely remains. Future longitudinal designs incorporating repeated hormonal assays, multiple inflammatory markers, and tighter control of covariates would provide a more comprehensive test of compensatory prophylaxis across shifting vulnerability states.

Study 2 Conclusion

Together with Study 1, these findings clarify several aspects of compensatory prophylaxis. Study 1 showed that hormonal context and PI shape who becomes emotionally reactive to pathogen cues. Study 2 extends this pattern by showing that when emotional reactivity occurs, it is associated with stronger avoidance motivation and reduced inflammatory reactivity. Hormonal context appeared to further shape the downstream effects of experiencing disgust. Endogenous progesterone influenced how disgust was translated into behavioral defenses and immunological reactions, with a compensatory prophylactic response observed within the luteal phase only. Exogenous progestin, on the other hand, shaped baseline inflammation, but did not meaningfully alter BIS-immune dynamics across pill phases. The present findings therefore suggest that compensatory versus complementary responding is context-dependent. Under moderately salient pathogen cues and hormonally immunomodulated contexts, BIS activation was more consistently associated with reduced inflammatory reactivity than

with co-activation of both systems. Therefore, disgust appears to function as an affective mechanism linking pathogen detection to both behavioral avoidance and immunoregulation.

If disgust serves as such a coordinating mechanism, its influence may extend beyond immediate avoidance motivations. The BIS has also been implicated in broader social judgements, including outgroup avoidance, ethnocentrism, and prejudice (e.g., Faulkner et al., 2004). If hormonal context calibrates disgust-based responding to pathogen cues, these shifts may also shape socially relevant attitudes. Study 3 tests this possibility by examining whether pathogen salience and hormonal context predict intergroup judgements.

CHAPTER FOUR

STUDY 3

The BIS evolved to detect and avoid potential sources of infection, but this pathogen avoidance system often responds to ambiguous or noninfectious stimuli that merely resemble disease (Ackerman et al., 2018). Stimuli such as visible injury, disability, or dermatological differences can elicit disgust and avoidance despite posing no actual contagion risk (Brown & Konner, 1987; Krems & Neuberg, 2022; Lieberman et al., 2012; Ryan et al., 2012; Tapp et al., 2020). One framework for understanding this tendency is signal detection theory (Haselton & Nettle, 2006; Nesse, 2001), which proposes that when false negatives are especially costly, adaptive systems should tolerate more false positives (Green & Swets, 1966). When applied to pathogen avoidance, the costs of failing to detect a true infection cue may outweigh the costs of unnecessary avoidance, leading to a bias toward caution.

Although this hypersensitivity may be adaptive for disease avoidance, it also has important social consequences. Overgeneralized pathogen avoidance can contribute to stigma, exclusion, and negative attitudes towards individuals who deviate from normative appearances or who are perceived as unfamiliar (Bressan, 2020; Oaten et al., 2011; Ryan et al., 2012; van Leeuwen et al., 2023; van Leeuwen & Jaeger, 2022). In other words, this system that likely evolved to reduce infection risk may also shape how people evaluate other individuals and groups, including those who are not infectious but are treated as if they might be (e.g., physically disabled individuals; e.g., Park et al., 2003). Consequently,

BIS activation has been linked to stigma, social exclusion, and a range of intergroup attitudes including ethnocentrism, xenophobia, and prejudice (e.g., Hromatko et al., 2021; Makhanova et al., 2023; Reid et al., 2012; Sawada et al., 2018; reviewed in Kramer & Bressan, 2021; van Leeuwen et al., 2023).

The downstream social consequences are also expected to vary as a function of individual vulnerabilities. Functional flexibility accounts propose that pathogen avoidance responses are calibrated to cues of infection risk, including both internal physiological states and external environmental information (Schaller et al., 2007). Inputs may reflect actual immunological vulnerability, including autoimmune disorders (e.g., rheumatoid arthritis; Oaten et al., 2017), hormonally immunomodulated states such as pregnancy or the luteal phase (Fessler, 2001, 2002; see also Bradshaw et al., 2022; Dlouhá et al., 2023; but see de Barra et al., 2014 for conflicting findings), or dispositional perceptions of susceptibility, such as perceived vulnerability to disease (Duncan et al., 2009) and disgust sensitivity (Fergus & Valentiner, 2009; van Overveld et al., 2006). Individuals with heightened BIS sensitivity are more likely to infer infectious risk from heuristic cues (e.g., Duncan et al., 2009; Lund & Boggero, 2014; Park et al., 2003, 2007), avoid previously contaminated targets (Mortensen et al., 2010), and perceive greater discordance in personality traits between themselves and individuals exhibiting heuristic disease cues (Makhanova et al., 2015). Thus, the same functional flexibility that calibrates immediate avoidance behavior may also shape broader social judgements under conditions of perceived vulnerability.

Ingroup Bias: Ethnocentrism, Norms, and Conformity

One way pathogen concerns may shape social judgment is through stronger

ingroup orientation. Humans depend heavily on cooperation, but group living also carries risks including exploitation, aggression, and pathogen transmission (reviewed in Ezenwa et al., 2016). Social categorization helps individuals quickly distinguish between likely allies and potential threats using cues such as appearance, accent, behavior, and shared norms (Brewer, 2001; Fiske, 1998; Kavaliers & Choleris, 2011; reviewed in Kavaliers & Choleris, 2018). Categorization does not require direct interaction, however. Those who conform to familiar expectations are more likely to be categorized as ingroup, whereas those who deviate (especially in ways that suggest a cultural or behavioral dissimilarity) are more likely to be categorized as outgroup (e.g., Ji et al., 2019; Kurzban & Leary, 2001; Petersen, 2017; Tybur et al., 2016; van Leeuwen & Peterson, 2018).

According to Social Identity Theory (Tajfel et al., 1971), people derive part of their self-concept from their group memberships (Brewer, 1991), and are therefore motivated to adopt favorable beliefs about their ingroup because ingroup outcomes are tied to self-esteem (Hogg & Abrams, 1999; Tajfel & Turner, 1979; for a recent meta-analysis, see Rivera et al., 2024). Consequently, individuals tend to extend more trust, empathy, and cooperation toward ingroup members (e.g., Balliet et al., 2014; Brewer & Caporael, 2006; Insko et al., 1990; Romano et al., 2017), but more cautious or negative expectations toward outgroup members (for reviews, see Brewer, 1991, 2001; Hewstone et al., 2002).

Pathogen avoidance mechanisms may amplify these dynamics by increasing preferences for familiar, predictable, and norm-abiding others. Over evolutionary time, such orientations may have promoted cooperation and norm enforcement within groups, while discouraging contact with those perceived as deviant or foreign (see Shapouri,

2023). Ethnocentrism (i.e., the tendency to view one's own group as central, legitimate, or morally superior) may represent one downstream expression of this orientation. Under relatively safe conditions, ethnocentrism may manifest in benign forms such as civic pride or cultural celebration (Mummendey et al., 2001). Under conditions of ecological or social threat, however, ethnocentrism tends to intensify (reviewed in Kramer & Bressan, 2021; Silva et al., 2022; van Leeuwen et al., 2023), as prioritizing cooperation, trust, and resource protection becomes increasingly important (Schaller & Park, 2011; reviewed in Schaller & Duncan, 2007). Consistent with this perspective, individuals with a more sensitive BIS report stronger ingroup loyalty (Fincher et al., 2008) and more favorable attitudes toward ingroup members (Navarrete & Fessler, 2006). Similarly, early pregnancy, a state characterized by elevated progesterone and immunomodulation, is associated with increased ingroup preference and ethnocentrism (Navarrete et al., 2007), possibly as an adaptive response to strengthen ingroup bonds and avoid outgroup risks when immune function is altered. Together, these findings suggest that pathogen concerns may increase ethnocentric attitudes by increasing the value of trusted allies and familiar social groups.

These processes may also operate through social norms and conformity. Shared norms help coordinate behavior, enhance group cohesion, lower social risk, and suppress free-riding (Grigoryan et al., 2022; Koch et al., 2020; Schaller & Duncan, 2007). Accordingly, individuals often prioritize moral alignment when evaluating potential group members (Leach et al., 2007), whereas moral dissimilarity predicts greater avoidance, social distance, and distrust (Haidt et al., 2003). When pathogen threat is salient, adherence to dominant norms may become especially valuable as familiar

practices can signal safety, cohesion, and predictability. Consistent with this account, both chronic pathogen concerns and experimentally induced disease salience have been linked to stronger norm adherence (e.g., Murray et al., 2019; Tybur et al., 2016; van Leeuwen et al., 2017) and enforcement (van Leeuwen et al., 2023), harsher judgements of norm violators (Plakias, 2018; see also Bollard, 2021; Clark & Fessler, 2014; Nussbaum, 2010; Tybur et al., 2016), greater support for obedience and authority (Cashdan & Steele, 2013; Murray & Schaller, 2012), and increased conformity (Murray et al., 2011; Murray & Schaller, 2012; van Leeuwen et al., 2023; Wu & Chang, 2012). Disgust may play a central role in these processes by signaling moral disapproval (Kupfer & Giner-Sorolla, 2016) and motivating social correction of exclusion (Giner-Sorolla & Espinosa, 2011; Rozin et al., 2009). These findings suggest that the BIS not only influences which norms are endorsed, but also how strictly they are enforced. As pathogen threat increases, so too does pressure to conform to dominant group values.

Cross-cultural evidence suggests that these patterns may scale up into broader social systems. In line with Parasite Stress Theory (Fincher et al., 2008; Thornhill & Fincher, 2014), regions facing greater chronic pathogen burdens tend to endorse more collectivist, norm-restrictive, and socially "tight" cultural systems (Fincher & Thornhill, 2012; Gelfand et al., 2011). These systems generally prioritize conformity, vigilance, and group coordination (Bennett & Nikolaev, 2020; Cukur et al., 2004; Liu et al., 2019; Shetty & Shetty, 2020) over individual autonomy, openness, and tolerance (Hofstede & McCrae, 2004; Jackson & Medvedev, 2024; Mullett et al., 2020; Schaller & Murray, 2008; Thornhill et al., 2010; Thornhill & Fincher, 2014; reviewed in Shapouri, 2023). Higher pathogen prevalence has also been associated with stronger ingroup favoritism,

greater support for authoritarian governance, and greater distrust of outsiders (Bennett & Nikolaev, 2020; Jackson & Medvedev, 2024; Faulkner et al., 2004; Murray et al., 2013; Zmigrod et al., 2021). Although these large-scale findings do not directly test individual-level BIS activation, they support a broader idea that pathogen threat can shape preferences for cohesion, order, and ingroup protection.

Taken together, these findings suggest that the BIS may extend beyond immediate pathogen avoidance to influence broader ingroup processes, including ethnocentrism, conformity, and preferences for social order. If pathogen concerns increase the value of trusted allies, shared norms, and coordinated group behavior, then experimentally activating pathogen salience may also shift attitudes in ways that favor ingroup cohesion.

Outgroup Bias: Xenophobia and Symbolic Prejudice

Although ingroup favoritism does not inevitably produce hostility toward outsiders, negative evaluations of outgroups become more likely under conditions of perceived threat (see Brewer, 2001; Hewstone et al., 2002 for reviews). These responses are context-dependent and often reflect concerns about maintaining group safety, stability, or cohesion (Neuberg & Schaller, 2016). Perceived threats may involve challenges to group identity, competition over resources, risks to physical safety, or violations of social norms (Brewer, 2001). Emotional reactions often correspond to the nature of the threat, whereby resentment is more common toward groups perceived as exploiting shared resources, fear and hostility toward those viewed as dangerous or destabilizing, and disgust toward groups perceived as contaminating, deviant, or morally offensive (Cottrell & Neuberg, 2005; Mackie et al., 2000; Neuberg & Schaller, 2016). These emotions, in turn, motivate functionally relevant responses such as avoidance,

exclusion, or punishment (Brewer, 2001).

One especially relevant threat for the present context is infectious disease. Even when disease risk is high across all groups (Peng & Bai, 2024), outgroups are often perceived as disproportionately responsible for disease transmission (Nesse, 2005; Reicher et al., 2016). This bias may be fueled by perceptions that unfamiliar groups introduce novel pathogens or fail to follow local norms that are implicitly associated with safety and hygiene (Brewer & Caporael, 2006; Fincher & Thornhill, 2012; Thornhill & Fincher, 2014; Thornhill et al., 2009). Historical instances of disease outbreaks following first-contact, such as the catastrophic depopulation of Indigenous peoples after European invasion and occupation (e.g., Lindo et al., 2016; O'Fallon & Fehren-Schmitz, 2011; reviewed in Collen et al., 2022) or the high mortality for European colonizers in West Africa and Papua New Guinea (e.g., Eves, 2005; Öberg & Rönnbäck, 2016), underscore the evolutionary salience of this bias.

More broadly, pathogen concerns may increase attention to unfamiliarity and norm violation, thereby lowering the threshold for perceiving social danger and increasing the likelihood of detecting false positives. Within this framework, norm-abiding ingroup members are perceived as trustworthy, whereas norm-deviant outgroup members are seen as unpredictable and disruptive (Insko et al., 1990; Sherman & Billing, 1999). While the BIS facilitates trust, loyalty, and norm enforcement within the ingroup, it also promotes caution, withdrawal, and even punitive responses toward outsiders when under conditions of perceived threat (e.g., Dasgupta et al., 2009).

Consistent with this framework, a growing body of research links BIS activity to prejudice toward groups perceived as violating social norms related to health, morality, or

social cohesion, including sexual minorities, individuals experiencing homelessness, people with substance use disorders, and atheists (e.g., Curtis & Biran, 2001; Inbar et al., 2009; O'Shea et al., 2020; Vartanian, 2010; reviewed in van Leeuwen et al., 2023). BIS responses are also sensitive to heuristic cues such as physical dissimilarity, unfamiliar behaviors, and accents that signal group membership and potential threat. For example, when disease salience is experimentally heightened, individuals high in pathogen disgust perceive foreign-accented speakers as more dissimilar from themselves (Reid et al., 2012), particularly when those accents are unfamiliar (e.g., Romanian, Jiangsu, Sierra Leone) rather than culturally familiar English accents (e.g., Irish, Scottish, New Zealand). These findings suggest that pathogen concerns may amplify perceptions of social differences in ways that may underlie prejudice.

One of the most robust findings in this literature is the association between pathogen sensitivity and xenophobia, often expressed as anti-immigrant and anti-refugee attitudes (reviewed in Kramer & Bressan, 2021; Silva et al., 2022). Individuals higher in disgust sensitivity are more likely to prefer greater social distance from foreigners (Szymkow et al., 2021), endorse ideologies and policies that reduce intergroup contact (Aarøe et al., 2020; Kam & Estes, 2016; Terrizzi et al., 2010), and oppose immigration across diverse national contexts (Clifford et al., 2023; Faulkner et al., 2004; Huang et al., 2011a; Makhanova et al., 2023; Navarrete & Fessler, 2006; reviewed in Aarøe et al., 2017). Similar patterns have also been observed for racial prejudice (e.g., Aaroe et al., 2020), even after controlling for political ideology, income, or education (Aarøe et al., 2017). This prejudice is often mediated by perceptions of dissimilarity from an outgroup members' national origin, especially if the outgroup is framed as violating cultural norms

or threatening group cohesion (Zakrzewska et al., 2019). Together, these findings suggest that pathogen avoidance may generalize into broader forms of outgroup negativity, particularly when others are perceived as unfamiliar, norm violating, or threatening to group cohesion.

The COVID-19 pandemic provided an ecologically valid case study of these dynamics. Xenophobic attitudes towards Asians increased early in the pandemic (Cheng et al., 2021; Nam et al., 2024; Nguyen et al., 2020; Reny & Barreto, 2020; but see Kim, 2024 for alternative findings), particularly among individuals who felt more vulnerable to infection (Reny & Barreto, 2020). Interestingly, objective infection risk was less predictive of these attitudes than perceived threat and pre-existing prejudice (Dai et al., 2024; Huber et al., 2022). Disease concerns during the pandemic were also associated with greater social distancing from minorities (Meleady et al., 2021), stronger support for punitive responses to those violating COVID-19 preventative behaviors (Hromatko et al., 2021; but see Kang, 2024), and support for restrictive travel bans targeting higher-risk regions (Adam-Troian & Bagci, 2021; Moran et al., 2021). These patterns reinforce the broader conclusion that pathogen threat can shift social attitudes toward ethnocentrism, xenophobia, and prejudice, particularly for those with greater perceptions of disease susceptibility (Faulkner et al., 2004; Green et al., 2010; Huang et al., 2011a; Navarrete et al., 2007).

Intergroup bias is often expressed indirectly rather than through overt hostility. Symbolic racism (also called symbolic prejudice) reflects subtle negative attitudes toward racialized outgroups framed through moral, cultural, or ideological concerns rather than explicit beliefs in biological inferiority (Henry & Sears, 2002; Sears & Henry, 2005). It is

often expressed through beliefs that racial discrimination is no longer an obstacle and in opposition to policies perceived as giving unfair advantages to minority groups (e.g., affirmative action) while holding beliefs and attitudes that one opposes racism and is not racist. Because symbolic prejudice emphasizes values such as hard work, conformity, deservingness, and respect for social rules, it may be especially relevant to behavioral immune processes that are sensitive to unfamiliarity, deviance, and norm enforcement.

The Role of Inflammation

Intergroup attitudes may also be shaped by physiological immune activity itself. Individuals with rheumatoid arthritis, who experience elevated infection vulnerability and report greater perceived susceptibility, are significantly more likely to judge ethnic outgroups as sources of contagion relative to their ethnic ingroup (Oaten et al., 2017). More broadly, interventions that reduce perceptions of disease susceptibility, such as hand washing (Huang et al., 2011), vaccine availability (Meleady & Hodson, 2022), or vaccination status (Huang et al., 2011), have in some cases been associated with lower outgroup bias. However, these effects are not entirely consistent (Makhanova et al., 2023; Meleady & Hodson, 2022), suggesting that intergroup responses may instead track fluctuations in the physiological immune system itself.

Consistent with this possibility, inflammatory activity has been linked to social cognition relevant to outgroup bias. Increases in IL-6 are associated with greater avoidance of strangers, though not close others (Jolink et al., 2022), as well as heightened vigilance to social threats (Inagaki et al., 2012). These findings suggest that inflammatory signaling may bias attention and behavior toward caution in the social environment, particularly in interactions involving unfamiliar others. If so, immune activation may

operate as an internal cue that influences broader social threat sensitivity alongside more immediate pathogen avoidance behaviors. Research involving recent vaccination highlights the complexity of these dynamics. In some studies, recently vaccinated individuals show reduced anti-immigrant prejudice following pathogen threat exposure (Huang et al., 2011a), consistent with the idea that lowered perceived susceptibility reduces the need for defensive behavioral responses. Similarly, during the COVID-19 pandemic, widespread vaccine availability was associated with decreases in self-reported outgroup avoidance (Meleady & Hodson, 2022). Yet, other findings suggest a different short-term process. Makhanova and colleagues (2023) found that recent flu vaccination was associated with increased IL-1β reactivity and more negative evaluations of a Latina (but not White) job candidate among predominantly (82%) White participants.

Taken together, these findings suggest that the timing and meaning of immune activation may be critical. Over longer intervals, vaccination may reduce perceived vulnerability and thereby attenuate outgroup bias. In the short-term, however, acute inflammatory activation may instead function as a cue that physiological resources are already in use, thereby increasing sensitivity to potential threats and motivating further behavioral defenses (Cole, 2006). For context, Makhanova and colleagues (2023) tested xenophobia 24 hours after vaccination, when post-vaccine cytokine expression peaks (Radin et al., 2021), whereas Meleady and Hudson (2022) asked about vaccination within the last two months.

Within this framework, intergroup bias may be shaped by both perceived susceptibility and momentary immune signaling, with different effects depending on whether the body is preparing for future protection or responding to an immediate

immune challenge. If physiological immune activity contributes to broader social threat sensitivity, then the same processes linking disgust and IL-6 may also extend to downstream intergroup judgements.

The Current Study

Across Studies 1 and 2, pathogen cues reliably increased state disgust, which predicted situational pathogen avoidance and lower inflammatory reactivity. These findings identified state disgust as the primary motivator of BIS activation linking pathogen detection to downstream behavioral and physiological responses. Study 3 tested whether the disgust responses observed in Studies 1 and 2 generalize to the social domain. Moreover, Study 3 extends the investigation of compensatory prophylaxis by examining whether pathogen salience and hormonal context predicted intergroup attitudes, and whether these more distal social outcomes followed the behavioral and physiological patterns observed in earlier studies.

Using the same experimental manipulation and participant sample as Study 2, Study 3 examined whether pathogen salience, state disgust, hormonal context, and PI were associated with variation in intergroup attitudes. Primary outcomes included symbolic racism toward immigrants, symbolic racism toward People of Color (POC), and ethnocentrism. Together, these outcomes captured both ingroup-oriented processes (e.g., ethnocentric endorsement of one's own cultural group) and outgroup-oriented processes (e.g., subtle prejudice toward marginalized or foreign groups). Consistent with the broader BIS framework, Study 3 tested whether state disgust mediated the association between pathogen salience and intergroup attitudes. Specifically, exposure to pathogen cues was expected to increase state disgust, which in turn was expected to predict tighter

social boundaries, reflected by higher ethnocentrism and greater symbolic prejudice. Hormonal context and PI were again examined as moderators of this pathway. Based on Studies 1 and 2, hormonally immunomodulated contexts (i.e., the luteal phase and active OC phase) and higher PI were expected to strengthen associations between pathogen salience, disgust, and intergroup bias. More generally, vulnerability signals were expected to shape either emotional responsiveness to pathogen cues or the downstream translation of disgust into intergroup bias.

By extending the examination of BIS activation from pathogen avoidance (Study 1) and inflammatory reactivity (Study 2) to intergroup attitudes, Study 3 provides a broader test of how pathogen cues and internal vulnerability signals may shape behavioral immune responding across social domains.

Method

Design

Study 3 measures were administered immediately following completion of Study 2 using the same sample mixed-combined design. Participants had been randomly assigned to either a pathogen-salient (experimental) or pathogen-free (control) condition in Study 2, with NC participants completing one session and OC participants completing two sessions (active and reminder pill phases). This study examined whether pathogen salience, hormonal context, and individual differences in PI were associated with intergroup attitudes, including symbolic racism and ethnocentrism.

Participants

Participants were the same 254 women who completed Study 2. Of these, 188 were NC (99 follicular phase, 85 luteal phase, 4 missing phase data) and 66 were OC

users (42 active pill phase, 23 reminder pill phase, 1 missing phase data). Recruitment procedures, eligibility criteria, and participation requirements were identical to those described in Study 2.

Materials

Hormonal Context

NC cycle phase, OC pill phase, and effective progestogenic activity values calculated in Study 2 were retained for Study 3 analyses.

Perceived Vulnerability to Disease Questionnaire

Scores on the PI subscale of the PVD (Duncan et al., 2009), calculated in Study 2, were used as a trait-level measure of perceived susceptibility to infection.

Culpepper Disgust Image Set

Experimental condition (pathogen-salient, control), determined by exposure to images from the C-DIS (Culpepper et al., 2018), was retained from Study 2.

Behavioral Immune System Reactivity

Scores on the two measurements of BIS reactivity were retained from Study 2: (1) a single state disgust item, “How disgusted do you feel right now?”, and (2) SPA (α = .813; Makhanova et al., 2021).

Symbolic Racism 2000 Scale

The Symbolic Racism 2000 Scale (SR2K; Henry & Sears, 2002; Sears & Henry, 2005) was used to assess subtle prejudice framed through moral, cultural, and ideological beliefs rather than explicit racial hostility. The scale was administered twice with the order randomized: once referencing immigrants (SR-Immigrants) and once referencing POC (SR-POC). The SR2K includes statements such as “It’s really a matter of some

people not trying hard enough; if [immigrants/POC] would only try harder they could be just as well off as whites," "Over the past few years, [immigrants/POC] have gotten more economically than they deserve," and "How much of the racial tension that exists in the United States today do you think [immigrants/POC] are responsible for creating?" Participants responded using 4-point Likert-type scales, with higher scores indicating greater symbolic racism (after reverse scoring items, as necessary).

The SR2K demonstrates acceptable internal consistency, with Cronbach's α values generally exceeding .73 (Henry & Sears, 2002). It also shows strong discriminant validity, as symbolic racism emerges as distinct from conservative ideology and traditional racist attitudes, as well as strong predictive validity for opposition to race-related government spending and affirmative action policies (Henry & Sears, 2002). Due to a coding error, one item assessing perceived discrimination, "How much discrimination against [immigrants/POC] do you feel there is in the United States today, limiting their chances to get ahead?" was omitted from both versions. Scores were averaged across items within each scale, resulting in 7-item composite scores rather than the standard 8-item scale. Internal consistency was acceptable for both SR-Immigrant (α = .74) and SR-POC (α = .78).

Revised Generalized Ethnocentrism Scale

The revised Generalized Ethnocentrism scale (GENE; Neuliep, 2002; Neuliep & McCroskey, 1997) was used to assess favorable attitudes towards individuals' own cultural group. The GENE demonstrates strong reliability, with test-retest correlations ranging from r = .82 to r = .92 (Neuliep, 2002). It also shows strong criterion validity, as higher ethnocentrism predicts greater distrust of outgroups, less work with foreigners,

and less international travel (Neuliep et al., 2005), as well as strong concurrent and construct validity through correlations with the Gudykunst Ethnocentrism Scale and measures of patriotism (Adorno et al., 1950; Gudykunst, 1997). Participants responded to 22 items on a 5-point Likert-scale, with higher scores indicating greater ethnocentrism (after reverse scoring, as necessary). Example items include "People in my culture have just about the best lifestyles of anywhere" and "Other cultures are smart to look up to our culture". Although items are traditionally summed to calculate composite ethnocentrism scores, scores were instead averaged to accommodate minimal missing data (1-2 responses missing across five items). Internal consistency was good (α = 0.84, excluding 7 distractor items).

Procedures

All Study 3 measures were administered immediately after participants completed the SPA and Honesty Verification item in Study 2. Participants then completed the intergroup attitude measures, including the two SR2K versions (SR-Immigrants, SR-POC) and the GENE in a random order. As in Study 2, NC participants completed the procedure once, whereas OC users completed it twice (once during their active pill phase and once during their reminder pill phase).

Results

Data Analysis

Following the data preparation procedures described in Study 2, analyses for Study 3 were conducted in SPSS (Version 31), with statistical significance set at α = .05. Unless otherwise noted, assumptions of normality, linearity, and homogeneity of variance were met.

Where Study 2 examined the relationship between BIS activation and physiological immune responses, Study 3 evaluated whether BIS activation extended to downstream social outcomes. Specifically, these analyses tested whether pathogen salience, state disgust, SPA, and hormonal context were associated with SR-Immigrants, SR-POC, and ethnocentrism. First, descriptive statistics and zero-order correlations were examined to understand associations among BIS-related variables (PI, state disgust, SPA) and intergroup attitudes. Second, direct effects of pathogen salience and hormonal context on intergroup attitudes were tested using multivariate analyses of covariance (MANCOVAs). Third, serial mediation models tested whether the compensatory pathway identified in Study 2 extended to intergroup attitudes. Specifically, serial mediation models were used to examine whether experimental condition predicted intergroup attitudes indirectly through state disgust and changes in IL-6 reactivity. Fourth, exploratory hierarchical regressions examined whether state disgust and SPA predicted intergroup attitudes beyond condition and PI, and whether these associations varied by hormonal context. Finally, among OC users, within-subject analyses examined whether shifts across women's own pill phases were associated with changes in intergroup attitudes. Repeated-measures ANCOVAs tested the effects of pill phase and condition while controlling for PI. Additional analyses examined whether progestogenic activity was associated with within-woman change scores in intergroup attitudes across pill phases.

Descriptive Statistics and Preliminary Analyses

Descriptive statistics for participant demographics and trait-level BIS variables were previously reported in Study 2 (see Table 3.3). Descriptive statistics for Study 3

intergroup outcomes are presented in Table 4.1. Independent-samples *t*-tests found no significant differences in SR-Immigrants, SR-POC, or ethnocentrism as a function of hormone group (NC, OC; all *p*s ≥ .178) or experimental condition (pathogen-salient, control; all *p*s ≥ .093), suggesting that intergroup attitudes did not differ systematically across groups.

To understand the associations among BIS-related variables and intergroup attitudes, zero-order correlations were examined (see Table 4.2). PI, state disgust, and SPA were positively intercorrelated (all *p*s < .015). PI and state disgust were not significantly associated with symbolic racism or ethnocentrism (*p*s ≥ .089). SPA was weakly negatively associated with SR-Immigrants ($p = .044$), but was not significantly related to SR-POC ($p = .099$) or ethnocentrism ($p = .136$). The intergroup outcomes were positively intercorrelated. SR-Immigrants and SR-POC were highly correlated ($p < .001$), and both were moderately positively correlated with ethnocentrism (*p*s < .001).

Table 4.1

Intergroup Outcomes by Hormone Group and Condition

Intergroup Outcomes	Total (*N* = 254)	NC (*n* = 188)	OC (*n* = 66)	Control (*n* = 127)	Experimental (*n* = 127)
SR-Immigrants	.26 (.16)	.25 (.16)	.28 (.17)	.27 (.16)	.25 (.17)
SR-POC	.27 (.18)	.26 (.18)	.27 (.18)	.29 (.17)	.25 (.18)
Ethnocentrism	1.72 (.51)	1.74 (.52)	1.65 (.47)	1.73 (.52)	1.72 (.51)

Note. Values are presented as *M* (*SD*). Group comparisons between hormone groups and experimental conditions were conducted using independent samples *t*-tests. Abbreviations: SR-Immigrants = symbolic racism toward immigrants, SR-POC = symbolic racism toward People of Color.

Table 4.2

Zero-Order Correlations Among Behavioral Immune System Predictors and Intergroup Attitude Outcomes Used in Study 3.

	Variable	1	2	3	4	5
1.	PI	–				
2.	State Disgust	.18**	–			
3.	SPA	.18**	.16*	–		
4.	SR-Immigrants	-0.05	-0.09	-0.13*	–	
5.	SR-POC	-0.05	-0.11	-0.10	.75***	–
6.	Ethnocentrism	-0.003	.04	.09	.33***	.33***

Abbreviations: PI = perceived infectability, SPA = situational pathogen avoidance, SR-Immigrants = symbolic racism toward immigrants, SR-POC = symbolic racism toward People of Color.

*$p < .05$, **$p < .01$, ***$p < .001$

Multivariate Analyses of Intergroup Attitudes

MANCOVAs were conducted to evaluate whether pathogen salience and hormonal context jointly predicted intergroup attitudes while controlling for individual differences in BIS sensitivity. Because NC and OC participants differed on measured (relationship status, race; see Chapter 3 Descriptive Statistics and Preliminary Analyses) and possibly unmeasured demographic or psychosocial characteristics (e.g., religiosity, political orientation; see Zettermark, 2024), analyses were conducted separately for endogenous and exogenous hormonal contexts. This approach also improved interpretability for phase-specific effects.

For NC participants, condition (experimental, control) and cycle phase (follicular,

luteal) were entered as fixed factors. For OC participants, condition and pill phase (active, reminder) were entered as fixed factors. In both models, PI was included as a covariate and the three intergroup outcomes (SR-Immigrants, SR-POC, ethnocentrism) were modeled simultaneously.

Intergroup Attitudes Across Naturally Cycling Hormonal Contexts

Among NC participants, the multivariate model indicated no significant effects of PI (Pillai's Trace = .01, $F(3, 177) = .46$, $p = .709$, $\eta^2 = .01$), condition (Pillai's Trace = .01, $F(3, 177) = .41$, $p = .749$, $\eta^2 = .01$), cycle phase (Pillai's Trace = .02, $F(3, 177) = .92$, $p = .433$, $\eta^2 = .02$), nor the condition × cycle phase interaction (Pillai's Trace = .02, $F(3, 177) = 1.13$, $p = .338$, $\eta^2 = .02$). Thus, neither pathogen salience nor endogenous hormonal context were associated with multivariate differences in intergroup attitudes among NC participants. Follow-up univariate tests were consistent. PI did not significantly predict SR-Immigrants, SR-POC, or ethnocentrism (all ps $\geq .264$), and there were no significant main effects of condition or cycle phase on any intergroup outcome (all ps $\geq .271$). The condition × cycle phase interaction was also nonsignificant for SR-POC ($F(1, 179) = 1.20$, $p = .275$, $\eta^2 = .01$) and ethnocentrism ($F(1, 179) = 1.50$, $p = .223$, $\eta^2 = .01$). Likewise, the condition × SR-Immigrants interaction did not reach significance ($F(1, 179) = 2.83$, $p = .094$, $\eta^2 = .02$).

Intergroup Attitudes Across Oral Contraceptive Pill Phases

Among OC participants, the multivariate model similarly revealed no significant effects of PI (Pillai's Trace = .02, $F(3, 57) = .36$, $p = .785$, $\eta^2 = .02$), condition (Pillai's Trace = .02, $F(3, 57) = .42$, $p = .742$, $\eta^2 = .02$), pill phase (Pillai's Trace = .03, $F(3, 57) = .51$, $p = .674$, $\eta^2 = .03$), nor the condition × pill phase interaction (Pillai's Trace = .01,

$F(3, 57) = .25$, $p = .862$, $\eta^2 = .01$), suggesting that pathogen salience and exogenous hormonal context are not associated with multivariate variance in intergroup attitudes among OC users. Follow-up univariate tests were again consistent. Neither PI, condition, pill phase, nor the condition × pill phase interaction significantly predicted SR-Immigrants (all ps ≥ .284), SR-POC (all ps ≥ .276), or ethnocentrism (all ps ≥ .299).

Compensatory Shifts in Intergroup Attitudes

Building on Study 2, additional analyses tested whether the compensatory pathway identified for IL-6 reactivity extended to intergroup attitudes. Serial mediation models were estimated using PROCESS (Model 6; Hayes, 2022). These models tested whether experimental condition (X) predicted intergroup outcomes (Y: SR-Immigrants, SR-POC, ethnocentrism) indirectly through state disgust (M_1) and ΔIL-6 (M_2). PI and recent illness were included as covariates. SPA was not included in these models because Study 2 indicated that SPA did not significantly predict IL-6 reactivity and was therefore not part of the compensatory pathway.

The analytic sample was limited to participants who had valid data for both IL-6 timepoints and all variables included in each model, with the additional requirement that at least one IL-6 value fall within the assay's detectable range (n = 196). Indirect effects were estimated using 5,000 bias-corrected bootstrap resamples with HC4 standard errors. Unstandardized regression coefficients are reported, with pathways labeled according to standard notation (a_1: X → M_1, a_2: X → M_2, d_{21}: M_1 → M_2; b_1: M_1 → Y; b_2: M_2 → Y; c': X → Y). Multicollinearity was acceptable across models (all VIFs < 2.04).

Across the three models, the upstream BIS pathway was consistent, although coefficients varied slightly due to outcome-specific missing data. Experimental condition

significantly predicted greater state disgust (a_1 = 45.38-45.61, ps < .001), and greater state disgust predicted lower ΔIL-6 (d_{21} = -0.01, ps = .003). Condition did not directly predict ΔIL-6 (a_2 = .18, ps = .434-.441). Neither PI nor recent illness significantly predicted state disgust or IL-6 reactivity across models (all ps ≥ .065). These findings replicate the proximal BIS pathway observed in Study 2 and provide the basis for testing whether this pathway extends to downstream intergroup attitudes.

In the downstream model predicting SR-Immigrants, neither condition (c' = .01, SE = .03, t = .24, p = .808, 95% CI [-0.06, .08]), state disgust (b_1 = .000, SE = .001, t = -0.62, p = .537, 95% CI [-0.001, .001]), nor ΔIL-6 (b_2 = .003, SE = .01, t = .35, p = .728, 95% CI [-0.02, .02]) significantly predicted the outcome. No indirect effects emerged, as all bootstrap confidence intervals included zero. A similar pattern was observed for SR-POC. Neither state disgust (b_1 = .000, SE = .000, t = -0.50, p = .621, 95% CI [-0.001, .001]) nor ΔIL-6 (b_2 = .01, SE = .01, t = 1.09, p = .279, 95% CI [-0.01, .03]) significantly predicted the outcome. The direct effect of condition was also not significant (c' = -0.003, SE = .03, t = -0.11, p = .915, 95% CI [-0.07, .06]), and no significant indirect effects were observed. Finally, neither state disgust (b_1 = .001, SE = .002, t = .74, p = .460, 95% CI [-0.002, .004]) nor ΔIL-6 (b_2 = .02, SE = .04, t = .68, p = .498, 95% CI [-0.05, .09]) significantly predicted ethnocentrism. The direct effect of condition was not significant (c' = .02, SE = .09, t = .19, p = .851, 95% CI [-0.17, .20]), and all indirect effects were nonsignificant.

Taken together, these findings replicated the compensatory BIS pathway at the level of proximal responses (i.e., pathogen cues increased disgust that in turn predicted reduced IL-6 reactivity). However, this pathway did not extend to intergroup attitudes.

Across all outcomes, no significant direct, indirect, or serial indirect effects emerged. Thus, there was no evidence that the physiological compensatory pathway observed in Study 2 generalized to downstream intergroup outcomes in the present sample.

Exploratory Analyses

Although no direct effects of pathogen salience or hormonal context emerged for explicit intergroup attitudes, findings from Study 2 established robust effects of pathogen salience on BIS-related states, particularly state disgust. Accordingly, exploratory analyses examined whether individual differences in state disgust and SPA were associated with intergroup attitudes, and whether these associations varied across hormonal contexts. Condition and PI were retained as covariates because the manipulation reliably increased state disgust and could otherwise confound associations between BIS responses and intergroup outcomes if not statistically controlled. Analyses involving hormonal context were again run separately for NC and OC participants.

BIS Responses and Intergroup Attitudes

A series of hierarchical multiple regressions tested whether state disgust and SPA predicted intergroup attitudes beyond condition and PI. In each model, condition and PI were entered at Step 1, followed by state disgust and SPA at Step 2. Outcomes included SR-Immigrants, SR-POC, and ethnocentrism.

For SR-Immigrants, the Step 1 model was not significant (R^2 = .01, F(2, 232) = .69, p = .501). Adding state disgust and SPA did not significantly improve model fit (ΔR^2 = .02, ΔF(2, 230) = 2.26, p = .106). In the full model, SPA showed a marginal negative association with SR-Immigrants (β = -0.13, t = -1.85, p = .065), whereas state disgust was not related (β = -0.05, t = -0.54, p = .591).

The model predicting SR-POC was similar. The Step 1 model was not significant (R^2 = .01, F(2, 231) = 1.34, p = .264), and Step 2 did not improve fit (ΔR^2 = .02, ΔF(2, 229) = 2.09, p = .126). Neither state disgust (β = -0.05, t = -0.50, p = .619) nor SPA (β = -0.12, t = -1.79, p = .075) were associated with anti-POC attitudes.

For ethnocentrism, the Step 1 model was not significant (R^2 = .001, F(2, 232) = .13, p = .878), and adding BIS predictors at Step 2 did not improve model fit (ΔR^2 = .01, ΔF(2, 230) = 1.08, p = .342). Neither state disgust (β = .02, t = .20, p = .845) nor SPA (β = .09, t = 1.36, p = .176) were significant predictors of ethnocentrism.

Taken together, BIS-related reactions did not meaningfully account for variance in intergroup attitudes when condition and PI were controlled. Although SPA showed small negative trends for the two symbolic racism outcomes, these effects did not reach statistical significance.

Moderation by Hormonal Context

To test whether hormonal context moderated BIS–intergroup attitude associations, a second set of hierarchical regressions were conducted, separately for NC and OC participants. All continuous predictors were mean-centered prior to computing interaction terms to reduce multicollinearity and improve interpretability. Across models, condition and centered PI were entered at Step 1, centered state disgust, centered SPA, and centered hormonal context (cycle phase for NC participants or pill phase for OC participants) were entered at Step 2, and the interaction terms (state disgust × phase, SPA × phase) were entered at Step 3.

Among NC participants, the only significant finding emerged for SR-POC. Adding BIS predictors and cycle phase at Step 2 significantly improved model fit (ΔR^2 =

.06, $\Delta F(3, 162) = 3.21$, $p = .025$). Higher SPA was associated with lower symbolic racism toward POC in both Step 2 ($\beta = -0.19$, $t = -2.39$, $p = .018$) and the final model ($\beta = -0.19$, $t = -2.28$, $p = .024$). No other predictors or interaction terms were significant for any NC outcome (see Table 4.3).

Among OC participants, no hierarchical regression model significantly predicted SR-Immigrants, SR-POC, or ethnocentrism. Across outcomes, Step 1 models were not significant (all ps $\geq .540$), adding main effects at Step 2 did not improve model fit (all ps $\geq .551$), and adding the interaction terms at Step 3 also did not improve model fit (all ps $\geq .563$; see Table 4.4).

Within-Subject Analyses Across OC Phases

Given the absence of broad between-group effects, additional analyses examined whether within-woman shifts across OC pill phases were associated with changes in intergroup attitudes. A series of repeated-measures ANCOVAs were conducted with pill phase (active, reminder) as a within-subjects factor, condition (experimental, control) as a between-subjects factor, and PI as a covariate[2]. Across all intergroup outcomes, there were no significant main effects of condition (ps $\geq .575$) or pill phase (ps $\geq .210$), no significant condition × pill phase interactions (ps $\geq .152$; see Table 4.5), and PI was not a significant covariate (ps $\geq .555$). These findings provide no evidence that within-woman changes in hormonal exposure across pill phases is associated with changes in intergroup attitudes.

[2] Because the primary question for this analysis concerned within-woman changes across pill phases rather than between-group differences by experimental condition, parallel models were also conducted with condition entered as a covariate rather than a between-subjects factor. No meaningful differences emerged across outcome variables.

Table 4.3

Hierarchical Regression Models Predicting Intergroup Attitudes from Behavioral Immune System Responses Among Naturally Cycling Participants.

Step	Predictor Variables	Outcomes								
		SR-Immigrants			SR-POC			Ethnocentrism		
		β	*p*	*ΔR²*	*β*	*p*	*ΔR²*	*β*	*p*	*ΔR²*
1	Condition	-0.04	.589	.01	-0.06	.452	.02	-0.01	.912	.000
	PI	-0.10	.214		-0.12	.133		-0.01	.937	
2	Condition	.07	.641	.04	.01	.900	.06*	.01	.957	.01
	PI	-0.04	.602		-0.06	.445		-0.01	.890	
	State disgust	-0.18	.101		-0.12	.278		-0.03	.795	
	SPA	-0.12	.138		-0.19*	.018		.06	.443	
	Cycle phase	-0.001	.991		-0.05	.564		.06	.456	
3	Condition	.08	.459	.01	.02	.859	.002	.01	.931	.001
	PI	-0.04	.620		-0.06	.461		-0.01	.897	
	State disgust	-0.20	.081		-0.12	.264		-0.03	.769	
	SPA	-0.11	.167		-0.19*	.024		.07	.432	
	Cycle phase	-0.004	.959		-0.05	.563		.06	.467	
	State disgust × Cycle phase	.10	.198		.01	.857		.04	.622	
	SPA × Cycle phase	-0.07	.378		-0.05	.525		-0.02	.783	
	Final model R^2			.06			.08			.01

Note: Values are standardized regression coefficients (*β*) and significance levels (*p*) for each outcome, and change in explained variance (ΔR^2) at each step of the hierarchical regression. Final model degrees of freedom were (7, 160).

Abbreviations: SR-Immigrants = symbolic racism toward immigrants, SR-POC = symbolic racism toward People of Color, PI = perceived infectability, SPA = situational pathogen avoidance.

*$p < .05$.

Table 4.4

Hierarchical Regression Models Predicting Intergroup Attitudes from Behavioral Immune System Responses Among Oral Contraceptive Users.

Step	Predictor Variables	Outcomes								
		SR-Immigrants			SR-POC			Ethnocentrism		
		β	*p*	*ΔR²*	*β*	*p*	*ΔR²*	*β*	*p*	*ΔR²*
1	Condition	-0.07	.574	.01	-0.14	.295	.02	-0.01	.968	.02
	PI	.01	.947		.05	.708		.13	.334	
2	Condition	-0.27	.152	.05	-0.22	.248	.03	-0.05	.774	.05
	PI	.03	.829		.03	.817		.09	.495	
	State disgust	.23	.220		.13	.505		.10	.582	
	SPA	-0.14	.333		.11	.451		.21	.137	
	Pill phase	.13	.361		.05	.742		.01	.967	
3	Condition	-0.29	.146	.004	-0.23	.240	.01	-0.09	.627	.03
	PI	.05	.749		.03	.808		.11	.411	
	State disgust	.24	.209		.13	.489		.13	.503	
	SPA	-0.15	.321		.08	.589		.16	.270	
	Pill phase	.13	.367		.07	.638		.04	.764	
	State disgust × Pill phase	-0.06	.657		.04	.758		-0.01	.965	
	SPA × Pill phase	.03	.842		.11	.445		.18	.181	
	Final model *R²*			.06			.06			.10

Note: Values are standardized regression coefficients (*β*) and significance levels (*p*) for each outcome, and change in explained variance (ΔR^2) at each step of the hierarchical regression. Final model degrees of freedom varied slightly by outcome due to missing data: SR-Immigrants (7, 54), SR-POC (7, 53), ethnocentrism (7, 54).

Abbreviations: SR-Immigrants = symbolic racism toward immigrants, SR-POC = symbolic racism toward People of Color, PI = perceived infectability, SPA = situational pathogen avoidance.

Table 4.5

Repeated-Measures ANCOVA Results for Experimental Condition and Pill Phase Predicting Intergroup Attitudes.

Predictors	Outcomes								
	SR-Immigrants			SR-POC			Ethnocentrism		
	$F(1, 58)$	p	η^2	$F(1, 57)$	p	η^2	$F(1, 58)$	p	η^2
Condition	.32	.575	.01	.30	.584	.01	.24	.626	.01
Pill phase	.19	.664	.003	.21	.649	.004	1.36	.249	.02
Condition × Pill phase	2.11	.152	.04	.09	.763	.002	.48	.492	.01

Note: Pill phase (active, reminder) was modeled as a within-subjects factor, and experimental condition (control, experimental) was modeled as a between-subjects factor. Values represent omnibus tests controlling for PI.

Abbreviations: η^2 = partial eta squared, PI = perceived infectability, SR-Immigrants = symbolic racism toward immigrants, SR-POC = symbolic racism toward People of Color.

Intergroup Attitudes and Progestogenic Activity

Although the original analysis plan proposed examining progestogenic activity as both a predictor of intergroup attitudes and a potential moderator of the effects of condition, these analyses were simplified due to the small OC subsample and the absence of higher-order effects in the above-reported models. Final analyses instead examined whether within-woman changes in progestogenic exposure across pill phases were associated with corresponding changes in intergroup attitudes.

To capture within-woman change, difference scores were computed for each intergroup outcome by subtracting scores during the reminder phase from scores during

the active phase. Positive values reflect higher intergroup bias during the active pill phase, whereas negative values reflect higher intergroup bias during the reminder pill phase. Because reminder pills contain no progestin, change in progestogenic activity was equivalent to progestogenic activity of each participant's active pill formulation. Because these analyses focus on within-woman changes, stable characteristics that did not vary across sessions were not included as covariates. Specifically, condition, PI, state disgust, and SPA did not significantly differ across women's own pill phases[3] and therefore were not controlled. These correlational analyses therefore tested whether women who experienced larger shifts in progestogenic exposure also showed larger shifts in intergroup attitudes. Progestogenic activity was not significantly associated with within-woman change in SR-Immigrants (r = .11, p = .424), SR-POC (r = .03, p = .825), or ethnocentrism (r = .10, p = .434), suggesting that variation in progestogenic exposure was not associated with explicit intergroup attitudes among OC users.

Discussion

Study 3 examined whether the BIS processes observed in Studies 1 and 2 extended beyond emotional and physiological responses to explicit intergroup attitudes. Specifically, this study tested whether pathogen salience, hormonal context, and individual differences in BIS-related reactions were associated with symbolic racism and ethnocentrism. Contrary to predictions, no direct effects of pathogen salience or hormonal context emerged in the primary analyses. Likewise, within-subject comparisons across OC pill phases revealed no reliable changes in attitudes across women's own

[3]Paired-samples *t*-tests indicated that PI, state disgust, and SPA did not significantly differ across women's own active and reminder pill phases (all *p*s > .306).

shifting hormonal states. Exploratory models similarly provided limited evidence that state disgust or SPA predicted intergroup attitudes. The only significant association was that higher SPA predicted lower symbolic racism toward POC among NC participants. Notably, this effect was in the opposite direction from predictions, suggesting that SPA may reflect pathogen avoidance motivations that do not straightforwardly map onto explicit anti-POC attitudes in this sample. Importantly, even the compensatory pathway established in Study 2 (i.e., where pathogen cues increased disgust, which in turn predicted reduced IL-6 reactivity) did not extend to any intergroup attitude outcome. Taken together, these findings provide no compelling evidence that BIS reactivity generalizes to explicit social attitudes as readily as some theoretical accounts imply (e.g., Faulkner et al., 2004; Navarrete & Fessler, 2006; Navarrete et al., 2007).

The current results may help to clarify the mixed findings in prior literature linking BIS sensitivity to exclusionary social attitudes and ideologies. Some previous work has reported associations between disgust sensitivity or pathogen concern and anti-immigrant attitudes (e.g., Aarøe et al., 2017; Kam & Estes, 2016), traditionalism (Karinen et al., 2019; Tybur et al., 2016), social conservatism (see Terrizzi et al., 2013 for a meta-analysis), authoritarianism (Pazhoohi & Kingstone, 2021; Tybur et al., 2016), religious fundamentalism (Terrizzi et al., 2013), opposition to marginalized groups (e.g., Aarøe et al., 2020; Clifford & Piston, 2017; Hodson & Costello, 2007; Inbar et al., 2009, 2012; Terrizzi et al., 2010), and other preferences that prioritize social conformity or intergroup boundaries (e.g., Aarøe et al., 2016). However, these relationships appear to be highly conditional, and may depend on factors such as whether target groups are framed as foreign or norm-violating, local disease ecology, and whether political

meaning is attached to disease threats (e.g., Dhanani & Franz, 2021; Freitag & Hofstetter, 2022; Ji et al., 2019; Karinen et al., 2019; Silva et al., 2022). One possible explanation is that the outcomes assessed in Study 3 operate at a different psychological level than those assessed in Studies 1 and 2. State disgust, pathogen avoidance motivations, and short-term inflammatory reactions are rapid responses to perceived contamination threats that are closely tied to immediately managing self-protection (Ersche et al., 2014; Gassen et al., 2019; Miller & Maner, 2011; Schaller et al., 2010; Stevenson et al., 2011, 2012, 2015). Symbolic racism and ethnocentrism, however, are broader constructs shaped by identity processes, social learning, political ideology, and lived experiences within cultural systems (see Kusche & Barker, 2019 for a review). As such, they are comparably more stable and may be less sensitive to brief or ambiguous pathogen manipulations like those used in the current study, particularly if disease threats are more generalized rather than being attached to socially meaningful targets. This distinction between generalized cues and those linked to specific social groups may be especially relevant outside of a research setting, where pathogen messaging is often embedded within cultural, political, and media rhetoric that explicitly links social groups with contamination or danger. Framing immigrants or minorities as disease carriers, or using contamination metaphors and dehumanizing rhetoric, has been shown to increase anti-immigrant attitudes (Landau et al., 2009; Marshall & Shapiro, 2018), social distancing (Szymków et al., 2021), and support for exclusionary policies (Aarøe et al., 2017), particularly among individuals already higher in germ aversion or disgust sensitivity (Brown et al., 2019; Hodson & Costello, 2007). Relatedly, research during the COVID-19 pandemic demonstrated that linking the virus to specific groups heightened xenophobia (Dhanani & Franz, 2021).

Prior work also suggests that BIS-related processes may be more detectable through implicit or behavioral outcomes (e.g., interpersonal distancing, dehumanization, behavioral avoidance; Buckels & Trapnell, 2013; Haslam, 2006) than through direct self-reports of prejudice. During real-world infectious outbreaks, for example, disease threat has been found to predict xenophobia and anti-immigrant bias through mechanisms such as anger (e.g., Freitag & Hofstetter, 2022) or dehumanization (Landry et al., 2022), rather than through disgust alone (e.g., Fan et al., 2024; Szymków et al., 2021). Together, these findings suggest that pathogen avoidance psychology may be most consequential for intergroup attitudes when pathogen threats are tied to specific target groups through social and cultural narratives, rather than through generic contamination cues. Moreover, the present findings may reflect limits in the sensitivity of the measures assessed here, and suggest that the impact of pathogen psychology on explicit intergroup attitudes may depend on how outcomes are operationalized and assessed (e.g., implicit vs. explicit).

Several features of the present sample and historical context in which these data were collected may help explain the limited effects observed here. Participants were university students, a population that tends to lean politically liberal (Scott, 2025) and report stronger generalized social trust than the general population (Huang et al., 2011b). Indeed, mean levels of symbolic racism and ethnocentrism in this sample were relatively low, which may have restricted variability and reduced the ability to detect meaningful associations. Moreover, most of these data were collected between late 2021 and late 2022, during a relatively polarized period in the United States characterized by intense public discourse surrounding race, immigration, inequality, and public health. Within this context, responses to explicit prejudice-related measures may have been influenced by

social desirability concerns or heightened moral norms. Accordingly, the present null effects should be interpreted as evidence that BIS-related mechanisms did not reliably predict these explicit self-reported attitudes in this sample and context, rather than as conclusive evidence that the BIS is irrelevant to intergroup attitudes more broadly.

Hormonal context also failed to reliably predict intergroup attitudes. Neither ovulatory cycle phase among NC participants nor pill phase among OC users were associated with differences in symbolic racism or ethnocentrism, and within-subject comparisons across women's own pill phases produced similar null findings. These results are notable when considered alongside Studies 1 and 2, in which hormonal context was involved in both proximal BIS and physiological immune processes. Across the three studies, reproductive hormones appear to be more relevant to the calibration of compensatory disgust and inflammatory reactivity than to explicit social attitudes. Correspondingly, hormonal shifts linked to cycle or pill phase may be more directly influential for managing immediate pathogen avoidance (e.g., vigilance, avoidance motivations, inflammatory signaling) than broader downstream ideological beliefs or consciously-endorsed social evaluations.

The absence of hormonal effects in Study 3 may also help to refine how the CPH is applied to ethnocentrism and prejudice-related outcomes (e.g., Navarrete et al., 2007). If progesterone-induced immunomodulation increases behavioral defenses during times of heightened vulnerability, those compensatory responses may be most visible in tightly coupled, domain-specific outputs like disgust sensitivity, behavioral avoidance motivations, health-protective behaviors, or immunological signaling rather than in more general intergroup ideologies. In this sense, the present findings suggest that the most

sensitive indicators of compensatory BIS responses are those that are more proximal, whereas intergroup attitudes may represent more distal constructs shaped by additional social and cultural variables.

Limitations and Future Directions

Several limitations should be considered when interpreting Study 3. First, all intergroup outcomes were assessed via explicit self-report measures, which are more vulnerable to social desirability and attribution biases (Gomez & Wilson, 2006). This concern may be especially relevant in morally charged contexts surrounding race, immigration, and public health (like that during data collection), where reported attitudes may relate to perceived norms as much as underlying BIS processes. Future research would benefit from incorporating implicit (e.g., evaluative priming tasks, Implicit Association Tasks; Greenwald et al., 1998) and behavioral measures to capture less consciously regulated forms of intergroup bias.

Second, the sample consisted of young university students that may be lower in explicit prejudice and more politically homogenous than the general population. Restricted variability in intergroup attitudes may have reduced the power to detect small effects. Replication in more diverse and politically heterogenous community samples is needed to examine whether BIS–attitude links emerge more strongly under different ideological or demographic contexts.

Third, both Studies 2 and 3 tested IL-6 as a single index of inflammatory responding, providing a narrow investigation of complex, dynamic immune processes. Other investigations of compensatory prophylaxis highlight the value of broader cytokine markers, white blood cell profiles, and secretory markers (e.g., sIgA) to characterize the

potential dynamics between behavioral and physiological defenses (Bradshaw et al., 2022; Kaňková et al., 2022; Keller & Diekhof, 2024). Future studies should include multiple inflammatory, endocrine, and autonomic markers to more comprehensively evaluate how physiological states relate to behavioral immune responding and social attitudes.

As with Study 2, some analyses (particularly those involving OC users) were based on small subsamples, which may have had limited sensitivity to detect small interaction effects. Moreover, the contracepted sample was restricted to OC users and did not include users of other non-oral HC methods (e.g., hormonal IUDs, implants, injections, patches, rings) that differ meaningfully in hormonal profiles and downstream psychological or physiological effects (e.g., D'Souza et al., 2023). Future work should expand contraceptive methods and increase OC subsample sizes to improve generalizability and provide a more complete understanding of exogenous hormonal influences.

Finally, interpretations of some intergroup outcomes are limited by the absence of direct measures of participants' identification with the target groups. Negative evaluations may therefore reflect outgroup prejudice, ingroup derogation (Jost et al., 2002), or other processes rather than straightforward prejudice toward an outgroup target. Ingroup derogation can increase when ingroups are heuristically associated with contamination risk under high disease salience (Wu et al., 2015). Although participant responses may still reflect BIS-related reactivity, future work should directly assess ingroup identification to disentangle these processes, where theoretically relevant. Similarly, future work should also continue to identify the boundary conditions under

which pathogen concerns do and do not translate into intergroup bias. For example, testing whether BIS activation predicts implicit rather than explicit attitudes, and whether dehumanization or interpersonal distancing are more sensitive than explicit prejudice, would clarify associations between the BIS and intergroup bias. Examining whether ideological orientation, chronic threat sensitivity, media exposure, and perceived cultural norms moderate BIS–prejudice links should be a priority. Repeated-measures designs may also clarify whether repeated or chronic pathogen salience leads to cumulative disgust or avoidance that gradually shapes broader social attitudes, even when single-session manipulations have weak or null effects. More broadly, future research would benefit from disentangling when pathogen avoidance motivates self-protection strategies (e.g., disgust, vigilance, avoidance) versus when it is socially targeted through cultural narratives, political messaging, or dehumanizing rhetoric that explicitly links specific groups with contamination. Integrating hormonal, immune, and social-context variables may illuminate how reproductive hormones and BIS processes jointly influence social attitudes without necessarily reshaping explicit ideological beliefs.

CHAPTER FIVE

GENERAL DISCUSSION

This dissertation examined whether cycle phases differing in endogenous progesterone level and exogenous progestin modulate behavioral and physiological responses to pathogen threat within the framework of the CPH (Fessler, 2001). Across three studies, this dissertation tested whether hormonal context predicted state disgust, situational pathogen avoidance, inflammatory reactivity, and intergroup attitudes following exposure to pathogen-salient cues. More broadly, this dissertation evaluated whether the BIS and physiological immune system operate in compensatory or complementary ways, and whether these relationships vary across NC women and OC users.

Across the three studies, several patterns emerged. First, pathogen cues reliably increased state disgust, which was the most consistent indicator of BIS activation. Second, disgust was associated with stronger pathogen avoidance motivations and reduced inflammatory reactivity, suggesting that affective, cognitive, and inflammatory defenses may coordinate in a compensatory manner under some conditions. These effects were shaped by internal vulnerability signals: PI and hormonal context influenced when and for whom the compensatory pattern was most robust across NC women and OC users. Third, there was limited evidence that these same processes generalized to explicit intergroup attitudes.

Overall, pathogen cues most consistently influenced emotional, motivational, and

immunological outcomes, whereas more distal social attitudes appeared to be relatively resistant to the experimental manipulation. These findings suggest that the BIS may be best understood as a *flexible*, context-sensitive defense system that prioritizes rapid and relatively low-cost protective responses when pathogen threat is perceived as manageable or ambiguous. Importantly, coordination between behavioral and physiological defenses appeared to depend on internal vulnerability, hormonal context, and the immediate protective relevance of the outcome being assessed.

Theoretical Implications

Across this dissertation, disgust emerged as the most consistent predictor of pathogen avoidance motivation. This is notable considering that pathogen-salient cues (Culpepper et al., 2018) did not uniformly increase avoidance motivations on their own. Instead, the relationship between pathogen cues and pathogen avoidance motivations was consistently transmitted through disgust, which statistically accounted for the condition effects in Studies 1 and 2 (full mediation in Study 1 and replication of the same general pattern in Study 2). In other words, pathogen cues appeared to motivate avoidance primarily when they were psychologically interpreted as contamination-relevant, rather than exerting a direct effect on avoidance tendencies. These findings align with theoretical accounts that position disgust as the central psychological mechanism of the BIS and suggest that it is the mechanism through which pathogen-relevant information is translated into reactive pathogen avoidance (Bradshaw et al., 2022; Campbell et al., 2020; Harris et al., 2019; Shook et al., 2019, 2020; Thorpe et al., 2011; Tolin et al., 2006; van Leeuwen & Jaeger, 2022; see Curtis & Biran, 2001; Curtis et al., 2011; Lieberman & Patrick, 2014; Oaten et al., 2009; Schaller, 2014; Söylemez & Kapucu, 2024 for reviews).

Disgust also influenced downstream immune activation, but not in the predicted direction. In Study 2, pathogen exposure did not elicit increases in IL-6. Instead, greater disgust following pathogen cues predicted *attenuated* IL-6 reactivity, even while corresponding with stronger pathogen avoidance motivation. This pattern suggests a divergence in response domains, whereby increased behavioral reactivity occurred alongside reduced inflammatory reactivity, consistent with a compensatory trade-off between behavioral and physiological defenses. These findings align with compensatory accounts of BIS-immune coordination (e.g., Bradshaw et al., 2022; Cepon-Robins et al., 2021; Fessler, 2001; Gassen et al., 2018; Oaten et al., 2017), which propose that engaging pathogen-avoidant psychology allows the body to downregulate costly inflammation. For example, higher trait pathogen avoidance motivation predicts lower cytokine release from immune cells and lower plasma IL-6 (Gassen et al., 2018), consistent with behavioral defenses compensating for chronic inflammatory investment. Similar patterns emerge in research on cognitive control over pathogen exposure, where individuals tend to rely more on disgust when their immunological investment is reduced, and vice versa (Bradshaw et al., 2022). From this perspective, disgust may serve as a coordinating signal that biases defensive resource allocation toward behavioral avoidance when inflammatory reactivity is inhibited.

These results do not necessarily contradict prior evidence that pathogen cues can pre-emptively upregulate inflammation (e.g., Ersche et al., 2014; Gassen et al., 2019; Juran et al., 2023; Muehlenbein et al., 2022; Schaller et al., 2010; Stevenson et al., 2011, 2012, 2015; reviewed in Murray et al., 2019). Rather, the present results suggest that BIS–immune coordination may vary systematically as a function of factors such as cue

salience, perceived control over exposure, the immune marker assayed (e.g., IL-6, TNF-α), and the timing of sampling relative to exposure. In the present studies, substantial variability in disgust responses within each condition suggests that pathogen cues were not uniformly interpreted as highly threatening. Under these more ambiguous conditions, behavioral avoidance may represent a sufficient low-cost response, whereas greater certainty of infection threat may be more likely to elicit complementary behavioral and inflammatory mobilization. Future research using more ecologically salient or personally relevant pathogen cues should investigate this possibility.

Relatedly, the present findings suggest that compensatory BIS–immune coordination may operate through more than one pathway. In some contexts, strong disgust and avoidance responses may reduce the need for inflammatory upregulation and help conserve physiological resources (e.g., Gassen et al., 2018). In other contexts, heightened behavioral defenses may compensate for physiological vulnerability. Findings that disgust sensitivity is negatively associated with several inflammatory cytokines during early pregnancy (Kaňková et al., 2022) are consistent with this latter interpretation. Similarly, in the present studies, the indirect pathway linking pathogen cues, disgust, and reduced IL-6 reactivity was strongest among participants who perceived themselves as more vulnerable to infection, consistent with research linking perceived vulnerability to disease to both behavioral and immunological pathogen defenses (reviewed in Lopes, 2017). Although PI did not significantly moderate the individual model pathways, it was associated with a greater likelihood of exhibiting an overall compensatory pattern.

Taken together, the present dissertation supports a context-dependent and

functionally flexible model of pathogen defense in which behavioral and physiological defensive systems are not necessarily coupled in parallel, but instead may trade off depending on perceived threat, internal vulnerability, and the relative costs of mobilizing resources. Because inflammatory capacity was not directly manipulated and hormone concentrations were not assayed, the present data cannot determine whether behavioral responses actively suppressed inflammatory activity or emerged alongside reduced physiological responsiveness. Nonetheless, the findings are consistent with a broader compensatory framework in which both resource conservation and vulnerability-based calibration contribute to pathogen defense.

Hormonal Modulation of Pathogen Defense

A central contribution of this dissertation is that pathogen avoidance mechanisms did not operate uniformly across participants. Consistent with functional flexibility models in which the BIS is calibrated to both environmental and internal cues of vulnerability (e.g., Hlay et al., 2024; Makhanova et al., 2022b; Sawada et al., 2018; Shook et al., 2020; reviewed in Ackerman et al., 2018; Schaller & Park, 2011; Schaller et al., 2021), hormonal context repeatedly shaped when and how pathogen defense responses emerged, particularly for proximal outcomes such as disgust, avoidance motivation, and inflammatory signalling, but not for broader social attitudes. From this perspective, the present findings extend traditional accounts of the CPH (Fessler, 2001), which typically focus on whether hormonally immunomodulated states are associated with elevated trait disgust or general disease avoidance (e.g., Bressan & Kramer, 2022; Fleischman et al., 2011; Jones et al., 2017; Miłkowska et al., 2021; Stern & Shiramizu, 2021; but see Keller & Diekhof, 2024). Rather than increasing pathogen responding in

general, hormonal context in Study 1 shaped whether responses were uniformly reactive or selectively calibrated to perceived vulnerability. Among users of lower progestogenic formulations, pathogen cues elicited relatively strong disgust regardless of perceived vulnerability, suggesting a lower threshold for behavioral immune activation. In contrast, among users of moderate- to high-progestogenic formulations, stronger disgust and avoidance responses were concentrated among participants who also perceived themselves as vulnerable to infection. Under stronger progestogenic immunomodulation, pathogen responding therefore appeared to become more strongly guided by internal estimates of susceptibility rather than environmental cues alone.

Study 2 further suggested that hormonal context shaped coordination between behavioral and physiological defenses. Greater state disgust predicted both stronger pathogen avoidance motivations and reduced IL-6 reactivity, consistent with compensatory BIS–immune trade-offs (e.g., Bradshaw et al., 2022; Gassen et al., 2018; Kaňková et al., 2022). Among NC women, this compensatory pathway emerged specifically during the luteal phase, when endogenous progesterone is elevated, but was absent during the follicular phase. This is direct evidence in support of the CPH, and suggests that progesterone-linked physiological states may increase reliance on behavioral defenses during pathogen threat. Among OC users, a similar compensatory pathway was observed across pill phases. However, the strength of this pathway weakened as progestogenic activity increased across formulations. Endogenous and exogenous hormonal contexts therefore appear to differ in how they may influence compensatory BIS-immune coordination. Cycle phase–specifically the luteal phase, which is characterized by heightened endogenous progesterone–was associated with

stronger compensatory coordination, whereas exogenous progestogenic activity appeared to attenuate this pathway.

Hormonal effects also emerged in baseline inflammatory functioning, with OC users in the reminder phase exhibiting higher IL-6 concentrations than during the active phase, a pattern replicated within participants across their own pill phases. This suggests that synthetic progestin exposure may suppress inflammatory activity, whereas hormonal withdrawal is associated with comparatively elevated inflammation. Together, these findings suggest that reproductive hormones calibrate both the threshold for BIS activation and the coordination of behavioral and physiological defenses, while also indicating that endogenous and exogenous hormones may exert different influences.

Boundary Conditions of the BIS

Although pathogen cues reliably shaped emotional, motivational, and physiological responses across this dissertation, these effects did not generalize to explicit intergroup attitudes. Study 3 found little evidence that pathogen salience, hormonal context, or their interaction reliably predicted symbolic racism or ethnocentrism. Similarly, the compensatory pathway observed in Study 2 did not extend to downstream social attitudes, suggesting that the mechanisms linking pathogen avoidance to social cognition may be more context dependent or psychologically distal than those involved in immediate self-protection from pathogens.

Compared to the other outcomes analyzed here, explicit social attitudes are more likely to reflect broader belief systems shaped by ideology, identity, social norms, and lived experiences (Kusche & Barker, 2019). Consequently, brief pathogen cues may be insufficient to influence intergroup beliefs in the anticipated ways. Consistent with this

interpretation, cross-cultural work finds that ecological parasite stress and disgust sensitivity relate more strongly to traditionalism than to endorsement of intergroup dominance and outgroup hostility (Tybur et al., 2016), and other studies report little evidence that experimental pathogen primes influence outgroup attitudes (Ji et al., 2019). Some researchers have further argued that the BIS evolved primarily to avoid infected individuals rather than outgroups per se, and suggest that links to outgroup prejudice may be byproducts of social learning and culturally transmitted beliefs about which groups are or are not "risky" (Ji et al., 2019; Kusche & Barker, 2019; van Leeuwen & Petersen, 2018).

The null findings of Study 3 are theoretically informative, as they may help clarify boundary conditions of BIS-related social cognition. BIS activation may be most relevant for proximal affective and behavioral responses to immediate pathogen cues (e.g., disgust, interpersonal distancing, comfort with contact; Hlay et al., 2021; Kavaliers et al., 2022; Meleady et al., 2021; Olivera-La Rosa et al., 2020; Tybur et al., 2020b), whereas broader social attitudes may require pathogen-relevant signals to be chronically embedded within normative social contexts before downstream attitudinal changes emerge (see Schaller et al., 2021).

Strengths, Limitations, and Future Directions

This dissertation has several notable strengths that advance theoretical and methodological understanding of behavioral immune functioning and compensatory prophylaxis. Most importantly, this project directly evaluated a central but unresolved question within the BIS literature: whether behavioral and physiological immune defenses operate in complementary or compensatory ways under pathogen threat. Rather

than relying exclusively on trait self-report measures of pathogen concern or disgust sensitivity, the present studies integrated an experimental pathogen manipulation with state emotional responding, pathogen avoidance motivation, inflammatory reactivity, and distal intergroup attitudes. Examining these processes together provided a broad test of pathogen avoidance psychology across emotional, motivational, physiological, and social domains.

A particular strength of this dissertation was the direct assessment of inflammatory reactivity through repeated salivary IL-6 measurements. Much prior BIS research has inferred physiological vulnerability indirectly or has relied primarily on self-report measures of disgust sensitivity or disease concern (e.g., Fessler & Navarrete, 2003; Jones et al., 2018; Zelaźniewicz et al., 2016). In contrast, the present studies assessed within-session inflammatory reactivity with pre- and post-manipulation IL-6 measurements, which allowed behavioral and physiological responses to pathogen cues to be examined simultaneously. This design provided a more direct test of BIS–immune coordination than is typical in the literature, and allowed compensatory versus complementary response patterns to be evaluated empirically rather than inferred.

This dissertation also incorporated multiple levels of hormonal analysis that extend prior work on the CPH. By including both NC and OC participants, Studies 2 and 3 were able to compare the influence of natural cycles and exogenous hormones directly. Repeating the session across OC users' active and reminder pill phases enabled further examination of within-person changes in inflammatory functioning and pathogen response, which increased ecological validity relative to cross-sectional comparisons alone. In addition, the inclusion of formulation-specific progestogenic activity provided a

more nuanced approach to HC research by examining pharmacological variability across OC formulations rather than treating OC users as a single homogenous group. Although this is not the first study to consider progestogenic activity on psychological outcomes (e.g., Hampson et al., 2022), relatively little research, and no BIS studies, have incorporated this level of specificity of OC formulation.

Finally, the integration of both trait and state indicators of pathogen susceptibility strengthened these studies' ability to examine the dynamics of behavioral immune calibration. By considering PI alongside experimentally induced disgust, the present work examined how beliefs about individual vulnerability interact with situational pathogen cues and hormonal context to shape pathogen avoidance responding. This approach allowed behavioral immune functioning to be examined as a dynamic process calibrated according to both internal and external vulnerability signals.

These strengths should be interpreted alongside several important limitations. Although the current approach aligns with established research practices (e.g., Gangestad et al., 2016; Welling & Burris, 2019), the absence of direct measurement of circulating progesterone or progestin concentrations limits the precision of hormonal estimates. Similarly, physiological immune response was assessed using IL-6 alone. Although IL-6 is widely used in psychobehavioral research to examine inflammatory responses to pathogen cues (e.g., Ersche et al., 2014; Gassen et al., 2019; Schaller et al., 2010), its pleiotropic role in both pro- and anti-inflammatory processes (reviewed in Fuster & Walsh, 2014; Gruys et al., 2005) complicates interpretation. Although pre- and post-manipulation IL-6 assays strengthened interpretation of within-session inflammatory reactivity, additional cytokines (e.g., TNF-α, IL-1β), acute phase markers (e.g., CRP),

cellular immune indices, or mucosal markers (e.g., sIgA) would provide a more comprehensive understanding of how behavioral and physiological defenses are coordinated across contexts. Future work should therefore incorporate salivary or serum hormone assays and consider additional markers of immune activity to more precisely characterize how hormonal fluctuations shape pathogen defense processes.

Several additional limitations relate to sample compositions and generalizability. Although this dissertation incorporated both between- and within-subject hormonal comparisons, some subgroup analyses (particularly among OC users) were based on modest sample sizes that may have limited sensitivity to detect smaller interaction effects. In addition, the exclusive focus on OC users (as opposed to more varied forms of HCs) limits generalizability to individuals using other, non-oral contraceptive methods, such as IUDs, injectables, or patches. Because oral formulations have more predictable pharmacokinetics (Bick et al., 2021), this restriction improves confidence in estimating dose and progestogenic potency, but future studies should expand the range of HC methods to include those that bypass first-pass hepatic metabolism and exhibit unique pharmacokinetic profiles to better characterize how distinct exogenous hormonal profiles shape pathogen avoidance. These samples also primarily consisted of young, cisgender, female university students and likely reflected a predominantly WEIRD population. Replication in more diverse community, cross-cultural, and gender-diverse (e.g., nonbinary, transgender men not using gender-affirming hormone therapy) samples will be important for establishing the scope and boundaries of these effects. Future longitudinal and repeated-measures designs may also clarify whether broader social consequences of pathogen concern emerge more reliably under chronic or repeated threat

than under brief experimental manipulations.

Finally, the intergroup attitude outcomes in Study 3 were assessed using explicit self-report measures, which may be less sensitive to subtle BIS-related processes and more vulnerable to social desirability or demand characteristics (Gomez & Wilson, 2006), particularly in politically and morally charged domains. In addition, participants were not asked whether they personally identified with the target groups assessed, making it impossible to distinguish outgroup bias from potential ingroup derogation processes (Jost et al., 2002; Wu et al., 2015). Future work would benefit from incorporating implicit, behavioral, and physiological measures of intergroup bias alongside assessments of group identification to better characterize how pathogen concerns shape social cognition outside of conscious awareness.

Beyond the limitations of the present studies, this dissertation raises several broader theoretical questions regarding how pathogen avoidance psychology is conceptualized and investigated within BIS research. The present findings suggest that behavioral and physiological defenses may coordinate more flexibly than many existing BIS models imply, particularly under conditions of more ambiguous or manageable pathogen threat. Findings also highlight the importance of incorporating hormonal context into pathogen-avoidance research rather than treating hormonal status as a categorical grouping variable (e.g., NC vs. HC user) or covariate. Existing research demonstrates that progesterone and progestins shape inflammatory functioning across reproductive contexts (reviewed in Hall & Klein, 2017; Raghupathy & Szekeres-Bartho, 2022; Zwahlen et al., 2023), yet much BIS research either ignores hormonal context entirely or operationalizes it in ways that may obscure opportunities for nuanced

understanding. The patterns observed here are consistent with views of disgust as part of a flexible disease-avoidance system that is shaped by endocrine context and coordinates with physiological immune responses. Future BIS work will therefore benefit from integrating hormonal assays and more comprehensive immune profiling into models of pathogen defense.

The present findings also align with growing literature suggesting that links between pathogen concern and broad ideological or intergroup attitudes may be more mixed and context-dependent than early BIS accounts proposed (e.g., Fan et al., 2022, 2024; Karinen et al., 2019; Makhanova et al., 2022a; Moran et al., 2021; Pazhoohi & Kingstone, 2021; see Howard et al., 2026 for a meta-analysis). Rather than directly motivating generalized outgroup avoidance or hostility, pathogen-defense psychology may become socially generalized when disease threat is linked to culturally meaningful groups through political rhetoric, media framing, or existing cultural narratives. For example, framing immigrants or marginalized groups as sources of contamination has been shown to increase anti-immigrant attitudes and support for exclusionary policies (Landau et al., 2009; Marshall & Shapiro, 2018), particularly among those already higher in germ aversion (Brown et al., 2019). Other work similarly suggests that pathogen concerns may amplify existing social or ideological biases rather than generating novel prejudices outright (e.g., Terrizzi et al., 2010). Although these responses may in part reflect evolved disease avoidance strategies (Kam & Estes, 2016; Schaller and Neuberg 2012), they can disproportionately target socially marginalized groups (Hodson & Costello, 2007; Inbar et al., 2012) and thus contribute to ideologies that undermine social inclusion and tolerance. Identifying the conditions under which avoidance of infection-

relevant cues becomes generalized onto social groups, including the psychological (e.g., dehumanization), cultural (e.g., social norms), ecological (e.g., pathogen prevalence), and political processes (e.g., disease-relevant rhetoric) that shape this shift, will be an important future direction.

Conclusion

This dissertation examined how exogenous progestin and cycle phases characterized by differing endogenous progesterone levels shape behavioral and physiological responses to pathogen threat within the framework of the CPH (Fessler, 2001). Across three studies, the most consistent finding was that pathogen cues reliably increased state disgust, which was linked to stronger pathogen avoidance motivation and reduced inflammatory reactivity, supporting a compensatory account in which behavioral and physiological defenses are flexibly coordinated rather than mobilized in parallel. These compensatory dynamics were further shaped by PI and hormonal context, particularly for proximal outcomes such as disgust, pathogen avoidance, and inflammatory signaling.

Hormonal context consistently influenced when pathogen defense responses emerged and how behavioral and physiological defenses were coordinated. Across studies, endogenous and exogenous hormones appeared to calibrate the threshold and organization of pathogen-defense responses rather than uniformly amplifying pathogen avoidance. These effects were most evident for proximal outcomes such as disgust, pathogen avoidance motivation, and inflammatory signaling, but did not extend to intergroup attitudes. Findings highlight important boundary conditions of the BIS and suggest that pathogen-defense processes may operate most reliably in domains involving

immediate self-protection from infection.

Taken together, this dissertation supports a view of the BIS as a flexible, context-sensitive system that helps regulate pathogen defense according to perceived vulnerability, hormonal state, and the relative cost of available responses. By integrating hormonal, immunological, and psychological approaches, this work clarifies the conditions under which compensatory prophylaxis is most evident and provides a foundation for future research on the behavioral, physiological, and social dynamics of human pathogen avoidance.

REFERENCES

Aarøe, L., Osmundsen, M., & Petersen, M. B. (2016). Distrust as a disease avoidance strategy: Individual differences in disgust sensitivity regulate generalized social trust. *Frontiers in Psychology*, *7*. https://doi.org/10.3389/fpsyg.2016.01038

Aarøe, L., Petersen, M. B., & Arceneaux, K. (2017). The behavioral immune system shapes political intuitions: Why and how individual differences in disgust sensitivity underlie opposition to immigration. *The American Political Science Review*, *111*(2), 277-294. https://doi.org/10.1017/S0003055416000770

Aarøe, L., Petersen, M. B., & Arceneaux, K. (2020). The behavioral immune system shapes partisan preferences in modern democracies: Disgust sensitivity predicts voting for socially conservative parties. *Political Psychology*, *41*(6), 1073-1091. https://doi.org/10.1111/pops.12665

Ackerman, J. M., Hill, S. E., & Murray, D. R. (2018). The behavioral immune system: Current concerns and future directions. *Social and Personality Psychology Compass*, *12*(2), e12371. https://doi.org/10.1111/SPC3.12371

Adam-Troian, J., & Bagci, S. C. (2021). The pathogen paradox: Evidence that perceived COVID-19 threat is associated with both pro-and anti-immigrant attitudes. *International Review of Social Psychology*, 34(1), 11. https://doi.org/10.5334/irsp.469

Adorno, T. W., Frenkel-Brunswik, E., Levinson, D. J., & Sanford, R. N. (1950). *The authoritarian personality*. Harper & Brothers.

Africander, D., Verhoog, N., & Hapgood, J. P. (2011). Molecular mechanisms of steroid receptor-mediated actions by synthetic progestins used in HRT and contraception.

Steroids, *76*(7), 636–652. https://doi.org/10.1016/J.STEROIDS.2011.03.001

Ainsworth, S. E., & Maner, J. K. (2019). Pathogen avoidance mechanisms affect women's preferences for symmetrical male faces. *Evolutionary Behavioral Sciences*, *13*(3), 265-271. https://psycnet.apa.org/doi/10.1037/ebs0000139

Alfaraj, S. H., Al-Tawfiq, J. A., & Memish, Z. A. (2019). Middle East Respiratory Syndrome Coronavirus (MERS-CoV) infection during pregnancy: Report of two cases & review of the literature. *Journal of Microbiology, Immunology and Infection*, *52*(3), 501–503. https://doi.org/10.1016/J.JMII.2018.04.005

Aliyu, M., Zohora, F. T., Anka, A. U., Ali, K., Maleknia, S., Saffarioun, M., & Azizi, G. (2022). Interleukin-6 cytokine: An overview of the immune regulation, immune dysregulation, and therapeutic approach. *International Immunopharmacology*, *111*, 109130. https://doi.org/10.1016/j.intimp.2022.109130

Allotey, J., Stallings, E., Bonet, M., Yap, M., Chatterjee, S., Kew, T., Debenham, L., Llavall, A. C., Dixit, A., Zhou, D., Balaji, R., Lee, S. I., Qiu, X., Yuan, M., Coomar, D., Van Wely, M., Van Leeuwen, E., Kostova, E., Kunst, H., … Thangaratinam, S. (2020). Clinical manifestations, risk factors, and maternal and perinatal outcomes of coronavirus disease 2019 in pregnancy: Living systematic review and meta-analysis. *BMJ*, *370*, m3320. https://doi.org/10.1136/BMJ.M3320

Alvarez, D., Vollmann, E. H., & von Andrian, U. H. (2008). Mechanisms and consequences of dendritic cell migration. *Immunity*, *29*(3), 325–342. https://doi.org/10.1016/J.IMMUNI.2008.08.006

Anderson, D. A., Dutertre, C. A., Ginhoux, F., & Murphy, K. M. (2020). Genetic models

of human and mouse dendritic cell development and function. *Nature Reviews Immunology*, *21*(2), 101–115. https://doi.org/10.1038/s41577-020-00413-x

Apter, D., Gemzell-Danielsson, K., Hauck, B., Rosen, K., & Zurth, C. (2014). Pharmacokinetics of two low-dose levonorgestrel-releasing intrauterine systems and effects on ovulation rate and cervical function: pooled analyses of phase II and III studies. *Fertility and Sterility*, *101*(6), 1656-1662.e4. https://doi.org/10.1016/J.FERTNSTERT.2014.03.004

Arruvito, L., Sanz, M., Banham, A. H., & Fainboim, L. (2007). Expansion of $CD4^{+}CD25^{+}$ and $FOXP3^{+}$ regulatory T cells during the follicular phase of the menstrual cycle: Implications for human reproduction. *Journal of Immunology, 178*(4), 2572-2578. https://doi.org/10.4049/jimmunol.178.4.2572

Arruvito, L., Giulianelli, S., Flores, A. C., Paladino, N., Barboza, M., Lanari, C., & Fainboim, L. (2008). NK cells expressing a progesterone receptor are susceptible to progesterone-induced apoptosis. *Journal of Immunology*, *180*(8), 5746–5753. https://doi.org/10.4049/JIMMUNOL.180.8.5746

Auerbach, L., Hafner, T., Huber, J. C., & Panzer, S. (2002). Influence of low-dose oral contraception on peripheral blood lymphocyte subsets at particular phases of the hormonal cycle. *Fertility and Sterility*, *78*(1), 83–89. https://doi.org/10.1016/S0015-0282(02)03173-4

Avelino, M. M., Campos, D., De Parada, J. D. C. B., & De Castro, A. M. (2003). Pregnancy as a risk factor for acute toxoplasmosis seroconversion. *European Journal of Obstetrics & Gynecology and Reproductive Biology*, *108*(1), 19–24. https://doi.org/10.1016/S0301-2115(02)00353-6

Balachandren, N., Davies, M. C., Hall, J. A., Stephenson, J. M., David, A. L., Barrett, G., O'Neill, H. C., Ploubidis, G. B., Yasmin, E., & Mavrelos, D. (2022). SARS-CoV-2 infection in the first trimester and the risk of early miscarriage: A UK population-based prospective cohort study of 3041 pregnancies conceived during the pandemic. *Human Reproduction*, *37*(6), 1126. https://doi.org/10.1093/HUMREP/DEAC062

Balliet, D., Wu, J., & De Dreu, C. K. (2014). Ingroup favoritism in cooperation: A meta-analysis. *Psychological Bulletin*, *140*(6), 1556. http://dx.doi.org/10.1037/a0037737

Ballou, S. P., & Lozanski, G. (1992). Induction of inflammatory cytokine release from cultured human monocytes by C-reactive protein. *Cytokine*, *4*(5), 361–368. https://doi.org/10.1016/1043-4666(92)90079-7

Banchereau, J., & Steinman, R. M. (1998). Dendritic cells and the control of immunity. *Nature*, *392*(6673), 245–252. https://doi.org/10.1038/32588

Barañao, R. I., Tenenbaum, A., & Rumi, L. S. (1991). Effects of sexual steroid hormones on the functionality of murine peritoneal macrophages. *Steroids*, *56*(9), 481–485. https://doi.org/10.1016/0039-128X(91)90006-H

Bastianelli, C., Farris, M., Rosato, E., Brosens, I., & Benagiano, G. (2018). Pharmacodynamics of combined estrogen-progestin oral contraceptives 3. Inhibition of ovulation. *Expert Review of Clinical Pharmacology*, *11*(11), 1085–1098. https://doi.org/10.1080/17512433.2018.1536544

Baud, D., & Greub, G. (2011). Intracellular bacteria and adverse pregnancy outcomes. *Clinical Microbiology and Infection*, *17*(9), 1312–1322.

https://doi.org/10.1111/J.1469-0691.2011.03604.X

Benagiano, G., Primiero, F. M., & Farris, M. (2004). Clinical profile of contraceptive progestins. *The European Journal of Contraception & Reproductive Care*, *9*(3), 182–193. https://doi.org/10.1080/13625180400007736

Bennett, D. L., & Nikolaev, B. (2020). Historical disease prevalence, cultural values, and global innovation. *Entrepreneurship Theory and Practice, 45*(1), 145-174. https://doi.org/10.1177/1042258720914506

Bick, A. J., Louw-du Toit, R., Skosana, S. B., Africander, D., & Hapgood, J. P. (2021). Pharmacokinetics, metabolism and serum concentrations of progestins used in contraception. *Pharmacology and Therapeutics*, *222*. https://doi.org/10.1016/J.PHARMTHERA.2020.107789

Binshtok, A. M., Wang, H., Zimmermann, K., Amaya, F., Vardeh, D., Shi, L., Brenner, G. J., Ji, R. R., Bean, B. P., Woolf, C. J., & Samad, T. A. (2008). Nociceptors are interleukin-1β sensors. *Journal of Neuroscience*, *28*(52), 14062–14073. https://doi.org/10.1523/JNEUROSCI.3795-08.2008

Bitran, D., Shiekh, M., & McLeod, M. (1995). Anxiolytic effect of progesterone is mediated by the neurosteroid allopregnanolone at brain $GABA_A$ receptors. *Journal of Neuroendocrinology, 7*(3), 171-177. https://doi.org/10.1111/j.1365-2826.1995.tb00744.x

Blomqvist, A., & Engblom, D. (2018). Neural mechanisms of inflammation-induced fever. *Neuroscientist*, *24*(4), 381–399. https://doi.org/10.1177/1073858418760481

Bollard, M. (2021). Is there such a thing as genuinely moral disgust? *Review of*

Philosophy and Psychology, *13*, 501-522. https://doi.org/10.1007/s13164-021-00539-4

Boltaña, S., Rey, S., Roher, N., Vargas, R., Huerta, M., Huntingford, F. A., Goetz, F. W., Moore, J., Garcia-Valtanen, P., Estepa, A., & MacKenzie, S. (2013). Behavioural fever is a synergic signal amplifying the innate immune response. *Proceedings of the Royal Society B: Biological Sciences*, *280*(1766). https://doi.org/10.1098/RSPB.2013.1381

Box, G. E., & Cox, D. R. (1964). An analysis of transformations. *Journal of the Royal Statistical Society Series B: Statistical Methodology*, *26*(2), 211-243. https://doi.org/10.1111/j.2517-6161.1964.tb00553.x

Brabin, L. (2004). Interactions of the female hormonal environment, susceptibility to viral infections, and disease progression. *AIDS Patient Care and STDs*, *16*(5), 211–221. https://doi.org/10.1089/10872910252972267

Bradshaw, H., Gassen, J., Prokosch, M., Boehm, G., & Hill, S. (2022). Control over pathogen exposure and basal immunological activity influence disgust and pathogen-avoidance motivation. *Cognition and Emotion*. https://doi.org/10.1080/02699931.2022.2031905

Brady, R. E., Badour, C. L., Arega, E. A., Levy, J. J., & Adams, T. G. (2021). Evaluating the mediating effects of perceived vulnerability to disease in the relation between disgust and contamination-based OCD. *Journal of Anxiety Disorders*, *79*, 102384. https://doi.org/10.1016/J.JANXDIS.2021.102384

Breder, C. D., Dinarello, C. A., & Saper, C. B. (1988). Interleukin-1 immunoreactive innervation of the human hypothalamus. *Science*, *240*(4850), 321–324.

https://doi.org/10.1126/SCIENCE.3258444

Bressan, P. (2020). Strangers look sicker (with implications in times of COVID-19). *BioEssays, 43*, 2000158. https://doi.org/10.1002/bies.202000158

Bressan, P., & Kramer, P. (2022). Progesterone does raise disgust. *Hormones and Behavior, 137*, 104937. https://doi.org/10.1016/j.yhbeh.2021.104937

Brewer, M. B. (1991). The social self: On being the same and different at the same time. *Perspectives in Social Psychology Bulletin, 17*(5), 475-482. https://doi.org/10.1177/0146167291175001

Brewer, M. B. (2001). Ingroup identification and intergroup conflict: When does ingroup love become outgroup hate? In R. Ashmore, L. Jussim, & D. Wilder (Eds.), *Social identity, intergroup conflict, and conflict reduction.* Oxford University Press.

Brewer, M. B., & Caporael, L. R. (2006). An evolutionary perspective on social identity: Revisiting groups. In M. Schaller, J. A. Simpson, & D. T. Kenrick (Eds.), *Evolution and social psychology* (pp. 143–161). Psychosocial Press.

Brown, B. P., & Allen, R. H. (2020). Progestin-only oral contraceptives. *The Handbook of Contraception*, 45–51. https://doi.org/10.1007/978-3-030-46391-5_3

Brown, M., Keefer, L. A., Sacco, D. F., & Bermond, A. (2019). Is the cure a wall? Behavioral immune system responses to a disease metaphor for immigration. *Evolutionary Psychological Science, 5*, 343-356. https://doi.org/10.1007/s40806-019-00191-3

Brown, P. J., & Konner, M. (1987). An anthropological perspective on obesity. *Annals of the New York Academy of Sciences, 499*, 29-46.

Buckels, E. E., & Trapnell, P. D. (2013). Disgust facilitates outgroup dehumanization.

Group Processes & Intergroup Relations, *16*(6), 771-780. https://doi.org/10.1177/1368430212471738

Burns, G. L., Keortge, S. G., Formea, G. M., & Sternberger, L. G. (1996). Revision of the Padua Inventory of Obsessive Compulsive Disorder symptoms: Distinctions between worry, obsessions, and compulsions. *Behaviour Research and Therapy*, *34*(2), 163-173. https://doi.org/10.1016/0005-7967(95)00035-6

Butts, C. L., Shukair, S. A., Duncan, K. M., Bowers, E., Horn, C., Belyavskaya, E., Tonelli, L., & Sternberg, E. M. (2007). Progesterone inhibits mature rat dendritic cells in a receptor-mediated fashion. *International Immunology*, *19*(3), 287–296. https://doi.org/10.1093/INTIMM/DXL145

Campbell, R. L., Bynion, T. M., Forte, J., Feldner, M. T., & Adams Jr, T. G. (2020). Specificity of disgust in the prediction of behavioral avoidance of possible contaminants. *Cognitive Therapy and Research*, *44*(2), 386-392. https://doi.org/10.1007/s10608-019-10067-2

Capitano, M. L., Nemeth, M. J., Mace, T. A., Salisbury-Ruf, C., Segal, B. H., McCarthy, P. L., & Repasky, E. A. (2012). Elevating body temperature enhances hematopoiesis and neutrophil recovery after total body irradiation in an IL-1–, IL-17–, and G-CSF–dependent manner. *Blood*, *120*(13), 2600–2609. https://doi.org/10.1182/BLOOD-2012-02-409805

Capuron, L., & Miller, A. H. (2011). Immune system to brain signaling: Neuropsychopharmacological implications. *Pharmacology & Therapeutics*, *130*(2), 226–238. https://doi.org/10.1016/J.PHARMTHERA.2011.01.014

Cashdan, E., & Steele, M. (2013). Pathogen prevalence, group bias, and collectivism in

the standard cross-cultural sample. *Human Nature, 24*, 59-75. https://doi.org/10.1007/s12110-012-9159-3

Cepon-Robins, T. J., Blackwell, A. D., Gildner, T. E., Liebert, M. A., Urlacher, S. S., Madimenos, F. C., Eick, G. N., Josh Snodgrass, J., & Sugiyama, L. S. (2021). Pathogen disgust sensitivity protects against infection in a high pathogen environment. *Proceedings of the National Academy of Sciences of the United States of America*, *118*(8), 2018552118. https://doi.org/10.1073/pnas.2018552118

Chai, Z., Gatti, S., Toniatti, C., Poli, V., & Bartfai, T. (1996). Interleukin (IL)-6 gene expression in the central nervous system is necessary for fever response to lipopolysaccharide or IL-1 beta: a study on IL-6-deficient mice. *Journal of Experimental Medicine*, *183*(1), 311–316. https://doi.org/10.1084/JEM.183.1.311

Chang, H. R., & Bistrian, B. (1998). The role of cytokines in the catabolic consequences of infection and injury. *Journal of Parenteral and Enteral Nutrition, 22*(3), 156-166. https://doi.org/10.1177/0148607198022003156

Chaouat, G., Assal Meliani, A., Martal, J., Raghupathy, R., Elliott, J. F., Elliot, J., ... & Wegmann, T. G. (1995). IL-10 prevents naturally occurring fetal loss in the CBA x DBA/2 mating combination, and local defect in IL-10 production in this abortion-prone combination is corrected by in vivo injection of IFN-tau. *The Journal of Immunology*, *154*(9), 4261-4268. https://doi.org/10.4049/jimmunol.154.9.4261

Chaouat, G., Menu, E., Clark, D. A., Dy, M., Minkowski, M., & Wegmann, T. G. (1990). Control of fetal survival in CBA × DBA/2 mice by lymphokine therapy. *Reproduction*, *89*(2), 447–458. https://doi.org/10.1530/JRF.0.0890447

Chatzileontiadou, D. S. M., Sloane, H., Nguyen, A. T., Gras, S., & Grant, E. J. (2021). The many faces of CD4+ T cells: Immunological and structural characteristics. *International Journal of Molecular Sciences*, *22*(1), 73. https://doi.org/10.3390/IJMS22010073

Cheng, H.-L., Kim, H. Y., Reynolds, J. D., Tsong, Y., & Joel Wong, Y. (2021). COVID-19 anti-Asian racism: A tripartite model of collective psychosocial resilience. *American Psychologist*, *76*(4), 627–642. https://doi.org/10.1037/amp0000808

Chihal, H. J. W., Peppler, R. D., & Dickey, R. P. (1975). Estrogen potency of oral contraceptive pills. *American Journal of Obstetrics and Gynecology*, *121*(1), 75-83. https://doi.org/10.1016/0002-9378(75)90979-5

Chrousos, G. P. (1995). The hypothalamic-pituitary-adrenal axis and immune-mediated inflammation. *The New England Journal of Medicine*, *332*(20), 1351–1363. https://doi.org/10.1056/NEJM199505183322008

Clark, J. A., & Fessler, D. M. T. (2014). The role of disgust in norms, and of norms in disgust research: Why liberals shouldn't be morally disgusted by moral disgust. *Topoi*, *34*, 483-498. https://doi.org/10.1007/s11245-014-9240-0

Clifford, S., Erisen, C., Wendell, D., & Cantú, F. (2023). Disgust sensitivity and support for immigration across five nations. *Politics and the Life Sciences, 42*(1), 65-80. https://doi.org/10.1017/pls.2022.6

Clifford, S., Piston, S. (2017). Explaining public support for counterproductive homelessness policy: The role of disgust. *Political Behavior, 39,* 503–525. https://doi.org/10.1007/s11109-016-9366-4

Cole, S.W. (2006). *The complexity of dynamic host networks.* In: Deisboeck, T.S., Kresh,

J.Y. (eds) Complex Systems Science in Biomedicine. Topics in Biomedical Engineering International Book Series. Springer, Boston, MA. https://doi.org/10.1007/978-0-387-33532-2_26

Collen, E. J., Johar, A. S., Teixeira, J. C., & Llamas, B. (2022). The immunogenetic impact of European colonization in the Americas. *Frontiers in Genetics, 13*(918227). https://doi.org/10.3389/fgene.2022.918227

Cottrell, C. A., & Neuberg, S. L. (2005). Different emotional reactions to different groups: A sociofunctional threat-based approach to "prejudice". *Journal of Personality and Social Psychology, 88(5),* 770-789. https://doi.org/10.1037/0022-3514.88.5.770

Cray, C. (2012). Acute phase proteins in animals. *Progress in Molecular Biology and Translational Science, 105*, 113–150. https://doi.org/10.1016/B978-0-12-394596-9.00005-6

Crameri, F. (2018). Scientific colour maps. Zenodo. http://doi.org/10.5281/zenodo.1243862

Crameri, F., Shephard, G. E., & Heron, P. J. (2020). The misuse of colour in science communication, *Nature Communications, 11*, 5444. https://doi.org/10.1038/s41467-020-19160-7

Crotty, S. (2019). T Follicular Helper Cell biology: A decade of discovery and diseases. *Immunity*, *50*(5), 1132–1148. https://doi.org/10.1016/J.IMMUNI.2019.04.011

Cukur, C. S., De Guzman, M. R. T., & Carlo, G. (2004). Religiosity, values, and horizontal and vertical individualism—collectivism: A study of Turkey, the United States, and the Philippines. *The Journal of Social Psychology, 144*(6),

613–634. https://doi.org/10.3200/SOCP.144.6.613-634

Culpepper, P. D., Havlícek, J., Leongómez, J. D., & Craig Roberts, S. (2018). Visually activating pathogen disgust: A new instrument for studying the behavioral immune system. *Frontiers in Psychology*, *9*(1397), 1–15. https://doi.org/10.3389/fpsyg.2018.01397

Curtis, V., Aunger, R., & Rabie, T. (2004). Evidence that disgust evolved to protect from risk of disease. *Proceedings of the Royal Society of London. Series B: Biological Sciences*, *271*(4). https://doi.org/10.1098/RSBL.2003.0144

Curtis, V., & Biran, A. (2001). Dirt, disgust, and disease: Is hygiene in our genes? *Perspectives in Biology and Medicine*, *44*(1), 17–31. https://doi.org/10.1353/PBM.2001.0001

D'Souza, A., Wageh, M., Williams, J., Colenso-Semple, L., McCarthy, D., McKay, A., Elliott-Sale, K., Burke, L., Parise, G., MacDonald, M., Tarnopolsky, M., & Phillips, S. (2023). Menstrual cycle hormones and oral contraceptives: A multimethod systems physiology-based review of their impact on key aspects of female physiology. *Journal of Applied Physiology, 135*, 1284-1299. https://doi.org/10.1152/japplphysiol.00346.2023

Dai, Y., Gao, J., & Radford, B. J. (2024). From fear to hate: Sources of anti-Asian sentiment during COVID-19. *Social Science Quarterly, 105*(3), 612-635. https://doi.org/10.1111/ssqu.13387

Daniels, K., & Abma, J. C. (2020). Current contraceptive status among women aged 15–49: United States, 2017–2019. *NCHS Data Brief, 388*. https://www.cdc.gov/nchs/products/index.htm.

Dantzer, R. (2001). Cytokine-induced sickness behavior: Mechanisms and implications. *Annals of the New York Academy of Sciences*, *933*(1), 222–234. https://doi.org/10.1111/J.1749-6632.2001.TB05827.X

Dantzer, R. (2004). Cytokine-induced sickness behaviour: A neuroimmune response to activation of innate immunity. *European Journal of Pharmacology*, *500*(1–3), 399–411. https://doi.org/10.1016/J.EJPHAR.2004.07.040

Dasgupta, N., DeSteno, D., Williams, L. A., & Hunsinger, M. (2009). Fanning the flames of prejudice: The influence of specific incidental emotions on implicit prejudice. *Emotion, 9*(4), 585–591. https://doi.org/10.1037/a0015961

de Barra, M., Islam, M. S., & Curtis, V. (2014). Disgust sensitivity is not associated with health in a rural Bangladeshi sample. *PLOS ONE, 9*(6), e100444. https://doi.org/10.1371/journal.pone.0100444

de Bosscher, K., Berghe, V., & Haegeman, G. (2000). Mechanisms of anti-inflammatory action and of immunosuppression by glucocorticoids: Negative interference of activated glucocorticoid receptor with transcription factors. *Journal of Neuroimmunology*, *109*, 16–22. https://doi.org/10.1016/S0165-5728(00)00297-6

Del Giudice, M., & Gangestad, S. W. (2018). Rethinking IL-6 and CRP: Why they are more than inflammatory biomarkers, and why it matters. *Brain, Behavior, and Immunity. 70*, 61-75. https://doi.org/10.1016/j.bbi.2018.02.013

Denney, J. M., Nelson, E. L., Wadhwa, P. D., Waters, T. P., Mathew, L., Chung, E. K., Goldenberg, R. L., & Culhane, J. F. (2011). Longitudinal modulation of immune system cytokine profile during pregnancy. *Cytokine, 53*(2), 170-177. https://doi.org/10.1016/j.cyto.2010.11.005

DeSisto, C. L., Wallace, B., Simeone, R. M., Polen, K., Ko, J. Y., Meaney-Delman, D., & Ellington, S. R. (2021). Risk for stillbirth among women with and without COVID-19 at delivery hospitalization — United States, March 2020–September 2021. *Morbidity and Mortality Weekly Report, 70*(47), 1640. https://doi.org/10.15585/MMWR.MM7047E1

Dhanani, L. Y., & Franz, B. (2021). Why public health framing matters: An experimental study of the effects of COVID-19 framing on prejudice and xenophobia in the United States. *Social Science & Medicine, 269*, 113572. https://doi.org/10.1016/j.socscimed.2020.113572

Díaz, A., Soriano, J. F., & Beleña, Á. (2016). Perceived Vulnerability to Disease questionnaire: Factor structure, psychometric properties and gender differences. *Personality and Individual Differences*, *101*, 42–49. https://doi.org/10.1016/J.PAID.2016.05.036

Dickey, R. P., & Seymour, M. L. (2021). *Managing contraceptive pill patients and other hormonal contraceptives* (17th edition). EMIS Medical Publishers.

Dixon, G. W., Schlesselman, J. J., Ory, H. W., & Blye, R. P. (1980). Ethinyl estradiol and conjugated estrogens as postcoital contraceptives. *JAMA, 244*(12), 1336-1339. https://doi.org/10.1001/jama.244.12.1336

Dlouhá, D., Roberts, S. C., Hlaváčová, J., Nouzová, K., & Kaňková, Š. (2023). Longitudinal changes in disgust sensitivity during pregnancy and the early postpartum period, and the role of recent health problems. *Scientific Reports, 13*(1), 1–10. https://doi.org/10.1038/s41598-023-31060-6

Dressing, G. E., Goldberg, J. E., Charles, N. J., Schwertfeger, K. L., & Lange, C. A.

(2011). Membrane progesterone receptor expression in mammalian tissues: A review of regulation and physiological implications. *Steroids*, *76*(1–2), 11–17. https://doi.org/10.1016/J.STEROIDS.2010.09.006

Duijkers, I. J. M., Klipping, C., Rautenberg, T., Schug, B. S., Kochhar, P. S., Osterwald, H., & Oettel, M. (2022). Effect on ovarian activity and ovulation inhibition of different oral dosages of levonorgestrel. *Contraception*, *110*, 6–15. https://doi.org/10.1016/J.CONTRACEPTION.2022.01.018

Duncan, L. A., Schaller, M., & Park, J. H. (2009). Perceived vulnerability to disease: Development and validation of a 15-item self-report instrument. *Personality and Individual Differences*, *47*(6), 541–546. https://doi.org/10.1016/J.PAID.2009.05.001

Dutta, A., Venkataganesh, H., & Love, P. E. (2021). New insights into epigenetic regulation of T cell differentiation. *Cells*, *10*(12), 3459. https://doi.org/10.3390/cells10123459

Ecochard, R., & Gougeon, A. (2000). Side of ovulation and cycle characteristics in normally fertile women. *Human Reproduction*, *15*(4), 752-755. https://doi.org/10.1093/humrep/15.4.752

Elenkov, I. J., & Chrousos, G. P. (1999). Stress hormones, Th1/Th2 patterns, pro/anti-inflammatory cytokines and susceptibility to disease. *Trends in Endocrinology & Metabolism*, *10*(9), 359–368. https://doi.org/10.1016/S1043-2760(99)00188-5

Ersche, K. D., Hagan, C. C., Smith, D. G., Abbott, S., Jones, P. S., Apergis-Schoute, A. M., & Döffinger, R. (2014). Aberrant disgust responses and immune reactivity in cocaine-dependent men. *Biological Psychiatry*, *75*(2), 140–147.

https://doi.org/10.1016/J.BIOPSYCH.2013.08.004

Evans, S. S., Repasky, E. A., & Fisher, D. T. (2015). Fever and the thermal regulation of immunity: The immune system feels the heat. *Nature Reviews Immunology*, *15*(6), 335–349. https://doi.org/10.1038/nri3843

Eves, R. (2005). Unsettling settler colonialism: Debates over climate and colonization in New Guinea, 1875-1914. *Ethnic and Racial Studies*, *28*(2), 304-330. https://doi.org/10.1080/0141987042000031586l

Ewies, A. A. A. (2009). Levonorgestrel-releasing intrauterine system - The discontinuing story. *Gynecological Endocrinology*, *25*(10), 668–673. https://doi.org/10.1080/09513590903159656

Ezenwa, V. O., Ghai, R. R., McKay, A. F., & Williams, A. E. (2016). Group living and pathogen infection revisited. *Current Opinion in Behavioral Sciences*, *12*, 66-72. https://doi.org/10.1016/j.cobeha.2016.09.006

Fan, L., Tybur, J. M., & Jones, B. C. (2022). Are people more averse to microbe-sharing contact with ethnic outgroup members? A registered report. *Evolution and Human Behavior*, *43*(6), 490-500. https://doi.org/10.1016/j.evolhumbehav.2022.08.007

Fan, L., Tybur, J., & Van Lange, P. (2024). Salience of infectious diseases did not increase xenophobia during the COVID-19 pandemic. *Evolutionary Human Sciences*, *6*, e34. https://doi.org/10.1017/ehs.2024.28

Faulkner, J., Schaller, M., Park, J. H., & Duncan, L. A. (2004). Evolved disease-avoidance mechanisms and contemporary xenophobic attitudes. *Group Processes & Intergroup Relations*, *7*(4), 333-353.

Faust, Z., Laškarin, G., & Rukavina, D. (1999). Progesterone-induced blocking factor

inhibits degranulation of natural killer cells. *American Journal of Reproductive Immunology*, *42*(2), 71–75. https://doi.org/10.1111/J.1600-0897.1999.TB00468.X

Fenton, R. R., Molesworth-Kenyon, S., Oakes, J. E., & Lausch, R. N. (2002). Linkage of IL-6 with neutrophil chemoattractant expression in virus-induced ocular inflammation. *Immunology and Microbiology*, *43*(3), 737–743.

Fergus, T. A., & Valentiner, D. P. (2009). The Disgust Propensity and Sensitivity Scale-Revised: An examination of a reduced-item version. *Journal of Anxiety Disorders*, *23*(5), 703–710. https://doi.org/10.1016/J.JANXDIS.2009.02.009

Fessler, D. M. T. (2001). Luteal phase immunosuppression and meat eating. *Biology Forum*, *94*(3), 403–426.

Fessler, D. M. T. (2002). Reproductive immunosuppression and diet. *Current Anthropology*, *43*(1), 19–61. https://doi.org/10.1086/324128

Fessler, D. M. T., Eng, S. J., & Navarrete, C. D. (2005). Elevated disgust sensitivity in the first trimester of pregnancy: Evidence supporting the compensatory prophylaxis hypothesis. *Evolution and Human Behavior*, *26*(4), 344–351. https://doi.org/10.1016/J.EVOLHUMBEHAV.2004.12.001

Fessler, D. M. T., & Navarrete, C. D. (2003). Domain-specific variation in disgust sensitivity across the menstrual cycle. *Evolution and Human Behavior*, *24*(6), 406–417. https://doi.org/10.1016/S1090-5138(03)00054-0

Fincher, C. L., & Thornhill, R. (2012). Parasite-stress promotes in-group assortative sociality: The cases of strong family ties and heightened religiosity. *The Behavioral and Brain Sciences*, *35*(6), 61-79.

https://doi.org/10.1017/s0140525x11000021

Fincher, C. L., Thornhill, R., Murray, D. R., & Schaller, M. (2008). Pathogen prevalence predicts human cross-cultural variability in individualism/collectivism. *Proceedings of the Royal Society B: Biological Sciences*, *275*(1640), 1279-1285. https://doi.org/10.1098/rspb.2008.0094

Fiske, S. T. (1998). Stereotyping, prejudice, and discrimination. In D. T. Gilbert, S., T. Fiske, & G. Lindzey (Eds.), *The handbook of social psychology* (p. 357-411). McGraw-Hill.

Flaxman, S. M., & Sherman, P. W. (2000). Morning sickness: A mechanism for protecting mother and embryo. *Quarterly Review of Biology*, *75*(2), 113–148. https://doi.org/10.1086/393377

Fleischman, D. S., & Fessler, D. M. T. (2011). Progesterone's effects on the psychology of disease avoidance: Support for the Compensatory Behavioral Prophylaxis Hypothesis. *Hormones and Behavior*, *59*(2), 271–275. https://doi.org/10.1016/J.YHBEH.2010.11.014

Follesa, P., Porcu, P., Sogliano, C., Cinus, M., Biggio, F., Mancuso, L., Mostallino, M. C., Paoletti, A. M., Purdy, R. H., Biggio, G., & Concas, A. (2002). Changes in GABAA receptor γ2 subunit gene expression induced by long-term administration of oral contraceptives in rats. *Neuropharmacology*, *42*(3), 325-336. https://doi.org./10.1016/S0028-3908(01)00187-3

Foo, Y. Z., Simmons, L. W., Perrett, D. I., Holt, P. G., Eastwood, P. R., & Rhodes, G. (2020). Immune function during early adolescence positively predicts adult facial sexual dimorphism in both men and women. *Evolution and Human Behavior*,

41(3), 199–209. https://doi.org/10.1016/J.EVOLHUMBEHAV.2020.02.002

Freitag, M., & Hofstetter, N. (2022). Pandemic threat and intergroup relations: How negative emotions associated with the threat of Covid-19 shape attitudes towards immigrants. *Journal of Ethnic and Migration Studies*, *48*, 2985-3004. https://doi.org/10.1080/1369183x.2022.2031925

Fruzzetti, F., & Fidecicchi, T. (2020). Hormonal contraception and depression: Updated evidence and implications in clinical practice. *Clinical Drug Investigation, 40*(12), 1097–1106. https://doi.org/10.1007/s40261-020-00966-8

Fu, B., Tian, Z., & Wei, H. (2014). Th17 cells in human recurrent pregnancy loss and pre-eclampsia. *Cellular & Molecular Immunology, 11*, 564-570. https://doi.org/10.1038/cmi.2014.54

Fuster, J. J., & Walsh, K. (2014). The good, the bad, and the ugly of interleukin-6 signaling. *The Embo Journal*, *33*, 1425-1427. https://doi.org/10.15252/embj.201488856

Gangestad, S. W., Haselton, M. G., Welling, L. L M., Gildersleeve, K., Pillsworth, E. G., Burriss, R. P., Larson, C. M., & Puts, D. A. (2016). How valid are assessments of conception probability in ovulatory cycle research? Evaluations, recommendations, and theoretical implications. *Evolution and Human Behavior, 37*(2), 85-96. https://doi.org/10.1016/j.evolhumbehav.2015.09.001

Gardiner, S. M., Kemp, P. A., March, J. E., Woolley, J., & Bennett, T. (1998). The influence of antibodies to TNF-alpha and IL-1beta on haemodynamic responses to the cytokines, and to lipopolysaccharide, in conscious rats. *British Journal of Pharmacology*, *125*(7), 1543–1550. https://doi.org/10.1038/SJ.BJP.0702250

Gassen, J., Makhanova, A., Maner, J. K., Plant, E. A., Eckel, L. A., Nikonova, L., Prokosch, M. L., Boehm, G. W., & Hill, S. E. (2019). Experimentally-induced inflammation predicts present focus. *Adaptive Human Behavior and Physiology*, *5*(2), 148–163. https://doi.org/10.1007/S40750-019-00110-7

Gassen, J., Prokosch, M. L., Makhanova, A., Eimerbrink, M. J., White, J. D., Proffitt Leyva, R. P., Peterman, J. L., Nicolas, S. C., Reynolds, T. A., Maner, J. K., McNulty, J. K., Eckel, L. A., Nikonova, L., Brinkworth, J. F., Phillips, M. D., Mitchell, J. B., Boehm, G. W., & Hill, S. E. (2018). Behavioral immune system activity predicts downregulation of chronic basal inflammation. *PloS One*, *13*(9). https://doi.org/10.1371/journal.pone.0203961

Gelfand, M. J., Raver, J. L., Nishii, L., Leslie, L. M., Lun, J., Lim, B. C., ... & Yamaguchi, S. (2011). Differences between tight and loose cultures: A 33-nation study. *Science, 332*(6033), 1100–1104. https://doi.org/10.1126/science.1197754

Gezer, A., & Oral, E. (2015). Progestin therapy in endometriosis. *Women's Health*, *11*(5), 643–652. https://doi.org/10.2217/whe.15.42

Ghaebi, M., Abdolmohammadi-Vahid, S., Ahmadi, M., Eghbal-Fard, S., Dolati, S., Nouri, M., Talebi, M., Hamdi, K., Marofi, F., Aghebati-Maleki, L., Jadidi-Niaragh, F., Dopour, M., & Yousefi, M. (2019). T cell subsets in peripheral blood of women with recurrent implantation failure. *Journal of Reproductive Immunology*, *131*, 21–29. https://doi.org/10.1016/J.JRI.2018.11.002

Giner-Sorolla, R., & Espinosa, P. (2011). Social cuing of guilt by anger and of shame by disgust. *Psychological Science, 22*(1), 49-53. https://doi.org/10.1177/0956797610392925

Giraldo, E., Hinchado, M. D., Garcia, J. J., & Ortega, E. (2008). Influence of gender and oral contraceptives intake on innate and inflammatory response. Role of neuroendocrine factors. *Molecular and Cellular Biochemistry*, *313*, 147–153. https://doi.org/10.1007/s11010-008-9752-2

Gomez, B. T., & Wilson, J. M. (2006). Rethinking symbolic racism: Evidence of attribution bias. *The Journal of Politics, 68*(3), 611-625. https://doi.org/10.1111/j.1468-2508.2006.00449.x

Goodman, W. A., Cooper, K. D., & McCormick, T. S. (2012). Regulation generation: The suppressive functions of human regulatory T cells. *Critical Reviews in Immunology*, *32*(1), 65-79. https://doi.org/10.1615/CritRevImmunol.v32.i1.40

Goovaerts, O., Jennes, W., Massinga-Loembé, M., Ceulemans, A., Worodria, W., Mayanja-Kizza, H., ... & TB-IRIS Study Group. (2013). LPS-binding protein and IL-6 mark paradoxical tuberculosis immune reconstitution inflammatory syndrome in HIV patients. *PloS One, 8*(11), e81856. https://doi.org/10.1371/journal.pone.0081856

Goulet, V., Hebert, M., Hedberg, C., Laurent, E., Vaillant, V., De Valk, H., & Desenclos, J. C. (2012). Incidence of Listeriosis and related mortality among groups at risk of acquiring Listeriosis. *Clinical Infectious Diseases*, *54*(5), 652–660. https://doi.org/10.1093/CID/CIR902

Grandi, G., Mueller, M., Bersinger, N. A., Cagnacci, A., Volpe, A., & McKinnon, B. (2016a). Does dienogest influence the inflammatory response of endometriotic cells? A systematic review. *Inflammation Research, 65*(3), 183–192. https://doi.org/10.1007/s00011-015-0909-7

Grandi, G., Mueller, M., Bersinger, N., Papadia, A., Nirgianakis, K., Cagnacci, A., & McKinnon, B. (2016b). Progestin suppressed inflammation and cell viability of tumor necrosis factor-α-stimulated endometriotic stromal cells. *American Journal of Reproductive Immunology*, *76*(4), 292–298. https://doi.org/10.1111/AJI.12552

Gravelsins, L., Zhao, S., & Einstein, G. (2023). Hormonal contraception and cognition: Considering the influence of endogenous ovarian hormones and genes for clinical translation. *Frontiers in Neuroendocrinology*, 101067. https://doi.org/10.1016/J.YFRNE.2023.101067

Green, E. G., Krings, F., Staerklé, C., Bangerter, A., Clémence, A., Wagner-Egger, P., & Bornand, T. (2010). Keeping the vermin out: Perceived disease threat and ideological orientations as predictors of exclusionary immigration attitudes. *Journal of Community & Applied Social Psychology, 20*(4), 299-316.

Green, D. M., & Swets, J. A. (1966). *Signal detection theory and psychophysics* (Vol. 1, pp. 1969-2012). New York: Wiley.

Greenwald, A. G., McGhee, D. E., & Schwartz, J. L. K. (1998). Measuring individual differences in implicit cognition: The implicit association test. *Journal of Personality and Social Psychology, 74*(6), 1464–1480. https://doi.org/10.1037/0022-3514.74.6.1464

Grigoryan, L., Jones, B. H., Cohrs, J. C., Boehnke, K., & Easterbrook, M. J. (2023). Differentiating between belief-indicative and status-indicative groups improves predictions of intergroup attitudes. *Personality and Social Psychology Bulletin, 49*(7), 1097-1112. https://doi.org/10.1177/01461672221092852

Grøntvedt, T. V., Grebe, N. M., Kennair, L. E. O., & Gangestad, S. W. (2017).

Estrogenic and progestogenic effects of hormonal contraceptives in relation to sexual behavior: Insights into extended sexuality. *Evolution and Human Behavior*, *38*(3), 283–292. https://doi.org/10.1016/J.EVOLHUMBEHAV.2016.10.006

Grover, P., Goel, P. N., & Greene, M. I. (2021). Regulatory T cells: Regulation of identity and function. *Frontiers in Immunology*, *12*, 750542 https://doi.org/10.3389/fimmu.2021.750542

Gruys, E., Toussaint, M. J. M., Niewold, T. A., & Koopmans, S. J. (2005). Acute phase reaction and acute phase proteins. *Journal of Zhejiang University. Science. B*, *6*(11), 1045. https://doi.org/10.1631/JZUS.2005.B1045

Gudykunst, W. B. (1997). Cultural variability in communication: An introduction. *Communication Research, 24,* 327-348.

Hagenacker, T., Czeschik, J. C., Schäfers, M., & Büsselberg, D. (2010). Sensitization of voltage activated calcium channel currents for capsaicin in nociceptive neurons by tumor-necrosis-factor-α. *Brain Research Bulletin*, *81*(1), 157–163. https://doi.org/10.1016/J.BRAINRESBULL.2009.09.012

Haidt, J., McCauley, C., & Rozin, P. (1994). Individual differences in sensitivity to disgust: A scale sampling seven domains of disgust elicitors. *Personality and Individual Differences*, *16*(5), 701–713. https://doi.org/10.1016/0191-8869(94)90212-7

Haidt, J., Rosenberg, E., & Hom, H. (2003). Differentiating diversities: Moral diversity is not like other kinds. *Journal of Applied Social Psychology*, *33*(1), 1-36. https://doi.org/10.1111/j.1559-1816.2003.tb02071.x

Hall, O. J., & Klein, S. L. (2017). Progesterone-based compounds affect immune responses and susceptibility to infections at diverse mucosal sites. *Mucosal Immunology*, *10*(5), 1097–1107. https://doi.org/10.1038/MI.2017.35

Hammad, H., Debeuf, N., Aegerter, H., Brown, A. S., & Lambrecht, B. N. (2022). Emerging paradigms in Type 2 immunity. *Annual Review of Immunology*, *40*, 443-467. https://doi.org/10.1146/annurev-immunol-101320-030339

Hampson, E., Morley, E. E., Evans, K. L., & Fleury, C. (2022). Effects of oral contraceptives on spatial cognition depend on pharmacological properties and phase of the contraceptive cycle. *Frontiers in Endocrinology*, *13*, 888510. https://doi.org/10.3389/fendo.2022.888510

Hamstra, D. A., De Rover, M., De Rijk, R. H., & Van der Does, W. (2014). Oral contraceptives may alter the detection of emotions in facial expressions. *European Neuropsychopharmacology*, *24*(11), 1855–1859. https://doi.org/10.1016/J.EURONEURO.2014.08.015

Hansen, M. K., O'Connor, K. A., Goehler, L. E., Watkins, L. R., & Maier, S. F. (2001). The contribution of the vagus nerve in interleukin-1β-induced fever is dependent on dose. *American Journal of Physiology - Regulatory Integrative and Comparative Physiology*, *280*(4 49-4). https://doi.org/10.1152/ajpregu.2001.280.4.R929

Hapgood, J. P., Kaushic, C., & Hel, Z. (2018). Hormonal contraception and HIV-1 acquisition: Biological mechanisms. *Endocrine Reviews*, *39*(1), 36–78. https://doi.org/10.1210/ER.2017-00103

Hapgood, J. P., Ray, R. M., Govender, Y., Avenant, C., & Tomasicchio, M. (2014).

Differential glucocorticoid receptor-mediated effects on immunomodulatory gene expression by progestin contraceptives: Implications for HIV-1 pathogenesis. *American Journal of Reproductive Immunology*, *71*(6), 505–512. https://doi.org/10.1111/AJI.12214

Harris, A. A., Romer, A. L., Hanna, E. K., Keeling, L. A., LaBar, K. S., Sinnott-Armstrong, W., Strauman, T. J., Wagner, H. R., Marcus, M. D., & Zucker, N. L. (2019). The central role of disgust in disorders of food avoidance. *International Journal of Eating Disorders*, *52*(5), 543-553. https://doi.org/10.1002/eat.23047

Hart, B. L. (1988). Biological basis of the behavior of sick animals. *Neuroscience & Biobehavioral Reviews*, *12*(2), 123–137. https://doi.org/10.1016/S0149-7634(88)80004-6

Haselton, M. G., & Nettle, D. (2006). The paranoid optimist: An integrative evolutionary model of cognitive biases. *Personality and Social Psychology Review*, *10*(1), 47-66. https://doi.org/10.1207/s15327957pspr1001_3

Haslam, N. (2006). Dehumanization: An integrative review. *Personality and Social Psychology Review*, *10*(3), 252-264. https://doi.org/10.1207/s15327957pspr1003_4

Hayes, A. F. (2022). *Introduction to mediation, moderation, and condition process analysis: A regression-based approach* (3rd Edition). Guilford Publications.

Hays, A., Duan, X., Zhu, J., Zhou, W., Upadhyayula, S., Shivde, J., Song, L., Wang, H., Su, L., Zhou, X., & Liang, S. (2019). Down-regulated Treg cells in exacerbated periodontal disease during pregnancy. *International Immunopharmacology*, *69*, 299-306. https://doi.org/10.1016/j.intimp.2019.01.031

Heffron, R., Achilles, S. L., Dorflinger, L. J., Hapgood, J. P., Kiarie, J., Polis, C. B., & Steyn, P. S. (2019). Pharmacokinetic, biologic and epidemiologic differences in MPA- and NET-based progestin-only injectable contraceptives relative to the potential impact on HIV acquisition in women. *Contraception*, *99*(4), 199–204. https://doi.org/10.1016/J.CONTRACEPTION.2018.12.001

Henry, P. J., & Sears, D. O. (2002). The Symbolic Racism 2000 Scale. *Political Psychology*, *23*(2), 253-283. https://doi.org/10.1111/0162-895X.00281

Hewstone, M., Rubin, M., & Willis, H. (2002). Intergroup bias. *Annual Review of Psychology*, *53*, 575-604.

Heydarifard, Z., Zadheidar, S., Yavarian, J., Malekshahi, S. S., Kalantari, S., Mokhtari-Azad, T., & Shafiei-Jandaghi, N-Z. (2022). Potential role of viral infections in miscarriage and insights into the underlying molecular mechanisms. *Congenital Anomalies*, *62*(2), 54–67. https://doi.org/10.1111/CGA.12458

Hilligan, K. L., & Ronchese, F. (2020). Antigen presentation by dendritic cells and their instruction of CD4+ T helper cell responses. *Cellular and Molecular Immunology*, *17*(6) 587–599. doi.org/10.1038/s41423-020-0465-0

Hlay, J. K., Albert, G., Batres, C., Richardson, G., Placek, C., Arnocky, S., Lieberman, D., & Hodges-Simeon, C. R. (2021). The evolution of disgust for pathogen detection and avoidance. Scientific Reports, 11, 13468. https://doi.org/10.1038/s41598-021-91712-3

Hlay, J. K., Albert, G., Batres, C., Richardson, G., Placek, C., Landry, N., Arnocky, S., Blackwell, A. D., & Hodges-Simeon, C. R. (2024). Greater self-reported health is associated with lower disgust: Evidence for individual calibration of the

behavioral immune system. Adaptive Human Behavior and Physiology, 10, 211-231. https://doi.org/10.1007/s40750-024-00243-4

Hodson, G., & Costello, K. (2007). Interpersonal disgust, ideological orientations, and dehumanization as predictors of intergroup attitudes. *Psychological Science, 18*(8), 691-698. https://doi.org/10.1111/j.1467-9280.2007.01962.x

Hofstede, G., & McCrae, R. R. (2004). Personality and culture revisited: Linking traits and dimensions of culture. *Cross-Cultural Research, 38*(1), 52-88. https://doi.org/10.1177/1069397103259443

Hogg, M.A., & Abrams, D. (1999). Social identity and social cognition: Historical background and current trends. In D. Abrams & M.A. Hogg (Eds.), S*ocial identity and social cognition* (pp. 1–25). Blackwell.

Hornung, R. W., & Reed, L. D. (1990). Estimation of average concentration in the presence of nondetectable values. *Applied Occupational and Environmental Hygiene*, *5*(1), 46-51. https://doi.org/10.1080/1047322X.1990.10389587

Hosokawa, H. & Rothenberg, E. V. (2020). How transcription factors drive choice of T cell fate. *Nature Reviews Immunology, 21*(3), 162-176. https://doi.org/10.1038/s41577-020-00426-6

Howard, M. C., Davis, M. M., & Serviss, E. (2026). A meta-analysis of perceived infectability, germ aversion, disgust and outgroup perceptions: Evaluating research on the behavioural immune system. British Journal of Psychology (Early View). https://doi.org/10.1111/bjop.70076

Hromatko, I., Grus, A., & Kolđeraj, G. (2021). Do islanders have a more reactive behavioral immune system? Social cognitions and preferred interpersonal

distances during the COVID-19 pandemic. *Frontiers in Psychology, 12*, 647586. https://doi.org/10.3389/fpsyg.2021.647586

Huang, J. Y., Sedlovskaya, A., Ackerman, J. M., & Bargh, J. A. (2011a). Immunizing against prejudice: Effects of disease protection on attitudes toward out-groups. *Psychological Science, 22*(12), 1550-1556. https://doi.org/10.1177/0956797611417261

Huang, J., Van Den Brink, H. M., & Groot, W. (2011b). College education and social trust: An evidence-based study on the causal mechanisms. *Social Indicators Research, 104*(2), 287-310. https://doi.org/10.1007/s11205-010-9744-y

Huber, C., Brietzke, S., Inagaki, T. K., & Meyer, M. L. (2022). American prejudice during the COVID-19 pandemic. *Scientific Reports, 12*(1), 22278. https://doi.org/10.1038/s41598-022-26163-5

Huber, J. C., Heskamp, M. L. S., & Schramm, G. A. (2008). Effect of an oral contraceptive with chlormadinone acetate on depressive mood: Analysis of data from four observational studies. *Clinical Drug Investigation, 28*(12), 783-791. https://doi-org.proxy.lib.umich.edu/10.2165/0044011-200828120-00006

Hughes, S. M., Levy, C. N., Katz, R., Lokken, E. M., Anahtar, M. N., Hall, M. B., Bradley, F., Castle, P. E., Cortez, V., Doncel, G. F., Fichorova, R., Fidel, P. L., Jr., Fowke, K. R., Francis, S. C., Ghosh, M., Hwang, L. Y., Jais, M., Jespers, V., Joag, V., Kaul, R., Kyongo, J., Lahey, T., Li, H., Makinde, J., McKinnon, L. R., Moscicki, A.-B., Novak, R. M., Patel, M. V., Sriprasert, I., Thurman, A. R., Yegorov, S., Mugo, N. R., Roxby, A. C., Micks, E., Hladik, F., & The Consortium for Assessing Immunity Across the Menstrual Cycle. (2022).

Changes in concentrations of cervicovaginal immune mediators across the menstrual cycle: A systematic review and meta-analysis of individual patient data. *BMC Medicine, 20*, 353. https://doi.org/10.1186/s12916-022-02532-9

Ichioka, M., Mita, S., Shimizu, Y., Imada, K., Kiyono, T., Bono, Y., & Kyo, S. (2015). Dienogest, a synthetic progestin, down-regulates expression of CYP19A1 and inflammatory and neuroangiogenesis factors through progesterone receptor isoforms A and B in endometriotic cells. *Journal of Steroid Biochemistry & Molecular Biology*, *147*, 103–110. https://doi.org/10.1016/j.jsbmb.2014.12.008

Inagaki, T. K., Muscatell, K. A., Irwin, M. R., Cole, S. W., & Eisenberger, N. I. (2012). Inflammation selectively enhances amygdala activity to socially threatening images. *Neuroimage, 59*(4), 3222-3226. https://doi.org/10.1016/j.neuroimage.2011.10.090

Inbar, Y., Pizarro, D. A., Knobe, J., & Bloom, P. (2009). Disgust sensitivity predicts intuitive disapproval of gays. *Emotion, 9*(3), 435–439. https://doi.org/10.1037/a0015960

Insko, C. A., Schopler, J., Hoyle, R. H., Dardis, G. J., & Graetz, K. A. (1990). Individual-group discontinuity as a function of fear and greed. *Journal of Personality and Social Psychology*, 58(1), 68-79. https://doi/10.1037/0022-3514.58.1.68

Izawa, S., Sugaya, N., Kimura, K., Ogawa, N., Yamada, K. C., Shirotsuki, K., Mikami, I., Hirata, K., Nagano, Y., & Nomura, S. (2013). An increase in salivary interleukin-6 level following acute psychosocial stress and its biological correlates in healthy young adults. *Biological Psychology, 94,* 249-254. https://doi.org/10.1016/j.biopsycho.2013.06.006

Jackson, J. C., & Medvedev, D. (2024). Worldwide divergence of values. *Nature Communications, 15*(2650). https://doi.org/10.1038/s41467-024-46581-5

Jain, J., Dutton, C., Nicosia, A., Wajszczuk, C., Bode, F. R., & Mishell, D. R. (2004). Pharmacokinetics, ovulation suppression and return to ovulation following a lower dose subcutaneous formulation of Depo-Provera®. *Contraception*, *70*(1), 11–18. https://doi.org/10.1016/j.contraception.2004.01.011

Jamieson, D. J., & Rasmussen, S. A. (2022). An update on COVID-19 and pregnancy. *American Journal of Obstetrics and Gynecology*, *226*(2), 177–186. https://doi.org/10.1016/j.ajog.2021.08.054

Jennings, M., & Cribbie, R. (2016). Comparing pre-post change across groups: Guidelines for choosing between difference scores, ANCOVA, and residual change scores. *Journal of Data Science, 14*(2), 205-229. https://doi.org/10.6339/JDS.201604_14(2).0002

Ji, T., Tybur, J. M., & van Vugt, M. (2019). Generalized or origin-specific out-group prejudice: The role of temporary and chronic pathogen-avoidance motivation in intergroup relations. *Evolutionary Psychology, 17*, 1-14. https://doi.org/10.1177/1474704919826851

Jin, L. P., Fan, D. X., Zhang, T., Guo, P. F., & Li, D. J. (2011). The costimulatory signal upregulation is associated with th1 bias at the maternal–fetal interface in human miscarriage. *American Journal of Reproductive Immunology*, *66*(4), 270–278. https://doi.org/10.1111/J.1600-0897.2011.00997.X

Jolink, T. A., Fendinger, N. J., Alvarez, G. M., Feldman, M. J., Gaudier-Diaz, M. M., & Muscatell, K. A. (2022). Inflammatory reactivity to the influenza vaccine is

associated with changes in automatic social behavior. *Brain, Behavior, and Immunity, 99*, 339-349. https://doi.org/10.1016/j.bbi.2021.10.019

Jones, B. C., Hahn, A. C., Fisher, C. I., Wang, H., Kandrik, M., Lee, A. J., Tybur, J. M., & DeBruine, L. M. (2018). Hormonal correlates of pathogen disgust: testing the compensatory prophylaxis hypothesis. *Evolution and Human Behavior*, *39*(2), 166–169. https://doi.org/10.1016/J.EVOLHUMBEHAV.2017.12.004

Jones, B. C., Little, A. C., Penton-Voak, I. S., Tiddeman, B. P., Burt, D. M., & Perrett, D. I. (2001). Facial symmetry and judgements of apparent health: Support for a "good genes" explanation of the attractiveness–symmetry relationship. *Evolution and Human Behavior*, *22*(6), 417–429. https://doi.org/10.1016/S1090-5138(01)00083-6

Jones, B. C., Perrett, D. I., Little, A. C., Boothroyd, L., Cornwell, R. E., Feinberg, D. R., Tiddeman, B. P., Whiten, S., Pitman, R. M., Hillier, S. G., Burt, D. M., Stirrat, M. R., Smith, M. J. L., & Moore, F. R. (2005). Menstrual cycle, pregnancy and oral contraceptive use alter attraction to apparent health in faces. *Proceedings of the Royal Society B: Biological Sciences*, *272*(1561), 347–354. https://doi.org/10.1098/RSPB.2004.2962

Jones, L. A., Kreem, S., Shweash, M., Paul, A., Alexander, J., & Roberts, C. W. (2010). Differential modulation of tlr3- and tlr4-mediated dendritic cell maturation and function by progesterone. *The Journal of Immunology*, *185*(8), 4525–4534. https://doi.org/10.4049/JIMMUNOL.0901155

Jost, J. T., Pelham, B. W., & Carvallo, M. R. (2002). Non-conscious forms of system justification: Implicit and behavioral preferences for higher status groups. J*ournal*

of Experimental Social Psychology, 38(6), 586-602. https://doi.org/10.1016/S0022-1031(02)00505-X

Juran, S. A., Tognetti, A., Lundström, J. N., Kumar, L., Stevenson, R. J., Lekander, M., Olsson, M. J. (2023). Disgusting odors trigger the oral immune system. *Evolution, Medicine, and Public Health, 11*(1), 8-17. https://doi.org/10.1093/emph/eoac042

Kaislasuo, J., Simpson, S., Petersen, J. F., Peng, G., Aldo, P., Lokkegaard, E., Paidas, M., Pal, L., Guller, S., & Mor, G. (2020). IL-10 to TNFα ratios throughout early first trimester can discriminate healthy pregnancies from pregnancy losses. *American Journal of Reproductive Immunology*, *83*(1), e13195. https://doi.org/10.1111/AJI.13195

Kalo-Klein, A., & Witkin, S. S. (1989). Candida albicans: Cellular immune system interactions during different stages of the menstrual cycle. *American Journal of Obstetrics & Gynecology*, *161*(5), 1132–1136. https://doi.org/10.1016/0002-9378(89)90649-2

Kam, C. D., & Estes, B. A. (2016). Disgust sensitivity and public demand for protection. *The Journal of Politics, 78*(2), 481-496. https://doi.org/10.1086/684611

Kamber, M. S., Sutter, M., Navarini, A., & Mueller, S. M. (2020). Contagious itch and disgust during a lecture on ectoparasitic infestations: Two co-activated complementary parasite defense systems? *Dermatologic Therapy*, *33*(6), e14456. https://doi.org/10.1111/DTH.14456

Kaňková, Š., Hlaváčová, J., Roberts, K., Benešová, J., Havlíček, J., Calda, P., Dlouhá, D., & Roberts, S. C. (2023). Associations between nausea and vomiting in pregnancy, disgust sensitivity, and first-trimester maternal serum free β-hCG and PAPP-A.

Hormones and Behavior, *152*, 105360. https://doi.org/10.1016/J.YHBEH.2023.105360

Kaňková, Š., Takács, L., Krulová, M., Hlaváčová, J., Nouzová, K., Hill, M., Včelák, J., & Monk, C. (2022). Disgust sensitivity is negatively associated with immune system activity in early pregnancy: Direct support for the Compensatory Prophylaxis Hypothesis. *Evolution and Human Behavior*. https://doi.org/10.1016/J.EVOLHUMBEHAV.2022.02.001

Kang, Y. (2024). Pathogen avoidance and outgroup avoidance during the first wave of the COVID-19 pandemic. *Japanese Psychological Research*. https://doi.org/10.1111/jpr.12511

Karg, S. T., Wiener-Blotner, A., & Schnall, S. (2019). Disgust sensitivity is associated with heightened risk perception. *Journal of Risk Research, 22*(5), 627-642.mhttps://doi.org/10.1080/13669877.2018.1474244

Karinen, A. K., Molho, C., Kupfer, T. R., & Tybur, J. M. (2019). Disgust sensitivity and opposition to immigration: Does contact avoidance or resistance to foreign norms explain the relationship? *Journal of Experimental Social Psychology, 84*, 103817. https://doi.org/10.1016/j.jesp.2019.103817

Kavaliers, M., & Choleris, E. (2011). Sociality, pathogen avoidance, and the neuropeptides oxytocin and arginine vasopressin. *Psychological Science, 22*(11), 1367-1374. https://doi.org/10.1177/0956797611420576

Kavaliers, M., & Choleris, E. (2018). The role of social cognition in parasite and pathogen avoidance. *Philosophical Transactions of the Royal Society B - Biological Sciences, 373*(1751), 20170206.

http://dx.doi.org/10.1098/rstb.2017.0206

Kavaliers, M., Ossenkopp, K., Tyson, C., Bishnoi, I., & Choleris, E. (2022). Social factors and the neurobiology of pathogen avoidance. *Biology Letters*, *18*(2), 20210371. https://doi.org/10.1098/rsbl.2021.0371

Keller, J., & Diekhof, E. (2024). Influence of female sex hormones on proactive behavioral and physiological immune parameters. *Reproductive Biology*, *24*(2), 100880. https://doi.org/10.1016/j.repbio.2024.100880

Keller, J. K., Wülfing, C., Wahl, J., & Diekhof, E. K. (2022). Disease-related disgust promotes antibody release in human saliva. *Brain, Behavior, & Immunity-Health*, *24*, 100489. https://doi.org/10.1016/j.bbih.2022.100489

Kelly, J. C., Raghuraman, N., Carter, E. B., Palanisamy, A., & Stout, M. J. (2021). Preprocedural asymptomatic coronavirus disease 2019 cases in obstetrical and surgical units. *American Journal of Obstetrics and Gynecology*, *224*(1), 114–116. https://doi.org/10.1016/J.AJOG.2020.09.023

Kent, S., Bluthé, R. M., Kelley, K. W., & Dantzer, R. (1992). Sickness behavior as a new target for drug development. *Trends in Pharmacological Sciences*, *13*(C), 24–28. https://doi.org/10.1016/0165-6147(92)90012-U

Khalil, R. H., & Al-Humadi, N. (2020). Types of acute phase reactants and their importance in vaccination (Review). *Biomedical Reports*, *12*(4), 143–152. https://doi.org/10.3892/BR.2020.1276/HTML

Kim, D. G. (2024). The politicization of COVID-19 and anti-Asian racism in the United States: An experimental approach. *Journal of Experimental Political Science*, *11*(1), 1–11. https://doi.org/10.1017/XPS.2023.16

Kimmig, A. C. S., Bischofberger, J. A., Birrenbach, A. D., Drotleff, B., Lämmerhofer, M., Sundström-Poromaa, I., & Derntl, B. (2022). No evidence for a role of oral contraceptive-use in emotion recognition but higher negativity bias in early follicular women. *Frontiers in Behavioral Neuroscience*, *15*, 352. https://doi.org/10.3389/fnbeh.2021.773961

Kindmark, C. O. (1971). Stimulating effect of C-reactive protein on phagocytosis of various species of pathogenic bacteria. *Clinical and Experimental Immunology*, *8*(6), 941-948.

Kirschbaum, C., Pirke, K. M., & Hellhammer, D. H. (1993). The 'Trier Social Stress Test' – A tool for investigating psychobiological stress responses in a laboratory setting. *Neuropsychobiology*, *28*(1–2), 76–81. https://doi.org/10.1159/000119004

Kleinknecht, R. A., Kleinknecht, E. E., & Thorndike, R. M. (1997). The role of disgust and fear in blood and injection—related fainting symptoms: A structural equation model. *Behaviour Research and Therapy*, *35*(12), 1075–1087. https://doi.org/10.1016/S0005-7967(97)80002-2

Kluger, M. J. (1986). Is Fever Beneficial? *The Yale Journal of Biology and Medicine*, *59*, 89–95.

Kluger, M. J., & Rothenburg, B. A. (1979). Fever and reduced iron: Their interaction as a host defense response to bacterial infection. *Science*, *203*(4378), 374–376. https://doi.org/10.1126/SCIENCE.760197

Knowles, K. A., Jessup, S. C., & Olatunji, B. O. (2018). Disgust in anxiety and obsessive-compulsive disorders: Recent findings and future directions. *Current Psychiatry Reports*, *20*(9), 68. https://doi.org/10.1007/s11920-018-0936-5

Koch, A., Dorrough, A., Glöckner, A., & Imhoff, R. (2020). The ABC of society: Perceived similarity in agency/socioeconomic success and conservative-progressive beliefs increases intergroup cooperation. *Journal of Experimental Social Psychology, 90*, 103996. https://doi.org/10.1016/j.jesp.2020.103996

Kramer, P., & Bressan, P. (2021). Infection threat shapes our social instincts. *Behavioral Ecology and Sociobiology, 75*(47). https://doi.org/10.1007/s00265-021-02975-9

Krattenmacher, R. (2000). Drospirenone: Pharmacology and pharmacokinetics of a unique progestogen. *Contraception*, *62*(1), 29–38. https://doi.org/10.1016/S0010-7824(00)00133-5

Krems, J. A., & Neuberg, S. L. (2022). Updating long-held assumptions about fat stigma: For women, body shape plays a critical role. *Social Psychological and Personality Science, 13*, 70-82.

Kuhl, H. (2005). Pharmacology of estrogens and progestogens: Influence of different routes of administration. *Climacteric*, *8*(1), 3–63. https://doi.org/10.1080/13697130500148875

Kupfer, T. R., & Fessler, D. M. T. (2018). Ectoparasite defence in humans: Relationships to pathogen avoidance and clinical implications. *Philosophical Transactions of the Royal Society B: Biological Sciences*, *373*(1751). https://doi.org/10.1098/RSTB.2017.0207

Kupfer, T. R., & Giner-Sorolla, R. (2016). Communicating moral motives: The social signaling function of disgust. *Social Psychological and Personality Science, 8*(6), 632-640. https://doi.org/10.1177/1948550616679236

Kurzban, R., & Leary, M. R. (2001). Evolutionary origins of stigmatization: The

functions of social exclusion. *Psychological Bulletin, 127*, 187-208. https://doi.org/10.1037//0033-2909.127.2.187

Kusche, I., & Barker, J. L. (2019). Pathogens and immigrants: A critical appraisal of the behavioral immune system as an explanation of prejudice against ethnic outgroups. *Frontiers in Psychology, 10,* 2412. https://doi.org/10.3389/fpsyg.2019.02412

Lacroix, R., Eason, E., & Melzack, R. (2000). Nausea and vomiting during pregnancy: A prospective study of its frequency, intensity, and patterns of change. *American Journal of Obstetrics and Gynecology*, *182*(4), 931–937. https://doi.org/10.1016/S0002-9378(00)70349-8

Lacroix, S., & Rivest, S. (1998). Effect of acute systemic inflammatory response and cytokines on the transcription of the genes encoding cyclooxygenase enzymes (COX-1 and COX-2) in the rat brain. *Journal of Neurochemistry*, *70*(2), 452–466. https://doi.org/10.1046/J.1471-4159.1998.70020452.X

Lam, C. M., Wong, S. F., Leung, T. N., Chow, K. M., Yu, W. C., Wong, T. Y., Lai, S. T., & Ho, L. C. (2004). A case-controlled study comparing clinical course and outcomes of pregnant and non-pregnant women with severe acute respiratory syndrome. *British Journal of Obstetrics and Gynaecology*, *111*(8), 771–774. https://doi.org/10.1111/J.1471-0528.2004.00199.X

Landau, M. J., Sullivan, D., & Greenberg, J. (2009). Evidence that self-relevant motives and metaphoric framing interact to influence political and social attitudes. *Psychological Science*, *20*(11), 1421-1427. https://doi.org/10.1111/j.1467-9280.2009.02462.x

Landry, A. P., Ihm, E., & Schooler, J. W. (2022). Filthy animals: Integrating the behavioral immune system and disgust into a model of prophylactic dehumanization. *Evolutionary Psychological Science, 8*(2), 120-133. https://doi.org/10.1007/s40806-021-00296-8

Leach, C. W., Ellemers, N., & Barreto, M. (2007). Group virtue: The importance of morality (vs. competence and sociability) in the positive evaluation of in-groups. *Journal of Personality and Social Psychology*, 93(2), 234. https://doi.org/10.1037/0022-3514.93.2.234

Lee, J. H., Ulrich, B., Cho, J., Park, J., & Kim, C. H. (2011). Progesterone promotes differentiation of human cord blood fetal t cells into t regulatory cells but suppresses their differentiation into th17 cells. *The Journal of Immunology, 187*(4), 1778–1787. https://doi.org/10.4049/JIMMUNOL.1003919

LeMay, L. G., Vander, A. J., & Kluger, M. J. (1990). Role of interleukin 6 in fever in rats. *The American Journal of Physiology, 258*(3), R798-R803. https://doi.org/10.1152/AJPREGU.1990.258.3.R798

Lenzi, E., Pluchino, N., Begliuomini, S., Ninni, F., Freschi, L., Centofanti, M., Casarosa, E., Luisi, S., Valentino, V., Luisi, M., & Genazzani, A. R. (2008). Effects of nomegestrol acetate administration on central and peripheral beta-endorphin and allopregnanolone in ovx rats. *The Journal of Steroid Biochemistry and Molecular Biology, 110*(1-2), 67-75. https://doi.org/10.1016/j.jsbmb.2008.02.002

León-Juárez, M., Martínez-Castillo, M., González-García, L. D., Helguera-Repetto, A. C., Zaga-Clavellina, V., García-Cordero, J., Flores-Pliego, A., Herrera-Salazar, A., Vázquez-Martínez, E. R., & Reyes-Muñoz, E. (2017). Cellular and molecular

mechanisms of viral infection in the human placenta. *Pathogens and Disease*, *75*(7), 93. https://doi.org/10.1093/FEMSPD/FTX093

Lieberman, D., & Patrick, C. (2014). Are the behavioral immune system and pathogen disgust identical? *Evolutionary Behavioral Sciences*, *8*(4), 244-250. https://doi.org/10.1037/ebs0000018

Lieberman, D., Tybur, J. M., & Latner, J. D. (2012). Disgust sensitivity, obesity stigma, and gender: Contamination psychology predicts weight bias for women, not men. *Obesity*, *20*, 1803-1814. https://doi.org/10.1038/oby.2011.247

Lindo, J., Huerta-Sánchez, E., Nakagome, S., Rasmussen, M., Petzelt, B., Mitchell, J., Cybulski, J. S., WIllerslev, E., DeGiorgio, M., & Malhi, R. S. (2016). A time transect of exomes from a native American population before and after European contact. *Nature Communications, 7*(13175). https://doi.org/10.1038/ncomms13175

Lissauer, D., Eldershaw, S. A., Inman, C. F., Coomarasamy, A., Moss, P. A. H., & Kilby, M. D. (2015). Progesterone promotes maternal–fetal tolerance by reducing human maternal T-cell polyfunctionality and inducing a specific cytokine profile. *European Journal of Immunology*, *45*(10), 2858–2872. https://doi.org/10.1002/EJI.201445404

Little, A. C., Jones, B. C., & Debruine, L. M. (2011). Facial attractiveness: Evolutionary based research. *Philosophical Transactions of the Royal Society - B*, *366*, 1638–1659. https://doi.org/10.1098/rstb.2010.0404

Liu, S. S., Morris, M. W., Talhelm, T., & Yang, Q. (2019). Ingroup vigilance in collectivistic cultures. *Proceedings of the National Academy of Sciences, 116*(29),

14538-14546. https://doi.org/10.1073/pnas.1817588116

Liu, M., Zhang, X., He, Z., Liang, Y., Zou, B., Ma, X., Gu, S., & Wang, F. (2023). Opposite effects of estradiol and progesterone on woman's disgust processing. *Frontiers in Psychiatry*, *14*, 508. https://doi.org/10.3389/fpsyt.2023.1161488

Lokau, J., & Garbers, C. (2020). Biological functions and therapeutic opportunities of soluble cytokine receptors. *Cytokine & Growth Factor Reviews*, *55*, 94–108. https://doi.org/10.1016/J.CYTOGFR.2020.04.003

Lopes, P. (2017). Why are behavioral and immune traits linked? *Hormones and Behavior*, *88*, 52-59. https://doi.org/10.1016/j.yhbeh.2016.09.008

Louw-du Toit, R., Perkins, M. S., Hapgood, J. P., & Africander, D. (2017). Comparing the androgenic and estrogenic properties of progestins used in contraception and hormone therapy. *Biochemical and Biophysical Research Communications*, *491*(1), 140–146. https://doi.org/10.1016/J.BBRC.2017.07.063

Lu, N. Z., Wardell, S. E., Burnstein, K. L., Defranco, D., Fuller, P. J., Giguere, V., Hochberg, R. B., Mckay, L., Renoir, J. M., Weigel, N. L., Wilson, E. M., Mcdonnell, D. P., & Cidlowski, J. A. (2006). International Union of Pharmacology. LXV. The pharmacology and classification of the nuclear receptor superfamily: Glucocorticoid, mineralocorticoid, progesterone, and androgen receptors.. *Pharmacological Reviews*, *58*(4), 782–797. https://doi.org/10.1124/PR.58.4.9

Lund, E. M., & Boggero, I. A. (2014). Sick in the head? Pathogen concerns bias implicit perceptions of mental illness. *Evolutionary Psychology*, *12*(4), 706-718. https://psycnet.apa.org/doi/10.1177/147470491401200403

Mackie, D. M., Devos, T., & Smith, E. R. (2000). Intergroup emotions: Explaining offensive action tendencies in an intergroup context. *Journal of Personality and Social Psychology, 79*(4), 602-616.

Makhanova, A., Lambert, W. A., Blanchard, R., Alcock, J., Shattuck, E. C., & Wilson, M. P. (2023). Pathogen disgust is associated with interpersonal bias among healthcare professionals. *Evolution, Medicine, and Public Health, 11*(1), 438-447. https://doi.org/10.1093/emph/eoad036

Makhanova, A., Miller, S. L., & Maner, J. K. (2015). Germs and the out-group: Chronic and situational disease concerns affect intergroup categorization. *Evolutionary Behavioral Sciences, 9*(1), 8-19. https://psycnet.apa.org/doi/10.1037/ebs0000028

Makhanova, A., Plant, E. A., Ketterman, A. B., & Maner, J. K. (2022a). Pathogen threat and intergroup prejudice using the minimal group paradigm: Evidence from a registered report. *Evolution and Human Behavior*, *43*(5), 347-357. https://doi.org/10.1016/j.evolhumbehav.2022.05.002

Makhanova, A., Plant, E. A., & Maner, J. K. (2021). Capturing fluctuations in pathogen avoidance: The Situational Pathogen Avoidance Scale. *Evolutionary Psychological Science*, *7*(1), 21–38. https://doi.org/10.1007/S40806-020-00256-8

Makhanova, A., Shepherd, M. A., Plant, E. A., Gerend, M. A., & Maner, J. K. (2022b). Childhood illness as an antecedent of perceived vulnerability to disease. *Evolutionary Behavioral Sciences, 16*(1), 53-66. https://psycnet.apa.org/doi/10.1037/ebs0000238

Malissen, B., Tamoutounour, S., & Henri, S. (2014). The origins and functions of

dendritic cells and macrophages in the skin. Nature Reviews Immunology, 14, 417-428. https://doi.org/10.1038/nri3683

Mantovani, A., & Garlanda, C. (2023). Humoral innate immunity and acute-phase proteins. *The New England Journal of Medicine, 388*(5), 439–452. https://doi.org/10.1056/NEJMra2206346

Mantovani, A., Sica, A., Sozzani, S., Allavena, P., Vecchi, A., & Locati, M. (2004). The chemokine system in diverse forms of macrophage activation and polarization. *Trends in Immunology*, *25*(12), 677–686. https://doi.org/10.1016/J.IT.2004.09.015

Marshall, S. R., & Shapiro, J. R. (2018). When "scurry" vs."hurry" makes the difference: Vermin metaphors, disgust, and anti-immigrant attitudes. *Journal of Social Issues, 74*(4), 774-789. https://doi.org/10.1111/josi.12298

Martínez-García, E. A., Chávez-Robles, B., Sánchez-Hernández, P. E., Núñez-Atahualpa, L., Martín-Máquez, B. T., Muñoz-Gómez, A., González-López, L., Gámez-Nava, J. I., Salazar-Páramo, M., Dávalos-Rodríguez, I., Petri, M. H., Zúñiga-Tamayo, D., Vargas-Ramírez, R., & Vázquez-Del Mercado, M. (2011). IL-17 increased in the third trimester in healthy women with term labor. *American Journal of Reproductive Immunology*, *65*(2), 99–103. https://doi.org/10.1111/J.1600-0897.2010.00893.X

Marzi, M., Vigano, A., Trabattoni, D., Villa, M. L., Salvaggio, A., Clerici, E., & Clerici, M. (1996). Characterization of type 1 and type 2 cytokine production profile in physiologic and pathologic human pregnancy. *Clinical & Experimental Immunology*, *106*(1), 127–133. https://doi.org/10.1046/J.1365-2249.1996.D01-

809.X

Medzhitov, R. (2007). Recognition of microorganisms and activation of the immune response. *Nature*, *449*(7164), 819–826. https://doi.org/10.1038/nature06246

Meleady, R., & Hodson, G. (2022). Reductions in perceived COVID-19 threat amid UK's mass public vaccination programme coincide with reductions in outgroup avoidance (but not prejudice). *British Journal of Social Psychology, 61(*4), 1286-1304. https://doi.org/10.1111/bjso.12537

Meleady, R., Hodson, G., & Earle, M. (2021). Person and situation effects in predicting outgroup prejudice and avoidance during the COVID-19 pandemic. *Personality and Individual Differences, 172*, 110593. https://doi.org/10.1016/j.paid.2020.110593

Memi E, Pavli P, Papagianni M, Vrachnis N, Mastorakos G. (2024). Diagnostic and therapeutic use of oral micronized progesterone in endocrinology. *Reviews in Endocrine and Metabolic Disorders, 25*, 751-772. https://doi.org/10.1007/s11154-024-09882-0

Menting-Henry, S., Hidalgo-Lopez, E., Aichhorn, M., Kronbichler, M., Kerschbaum, H., & Pletzer, B. (2022). Oral contraceptives modulate the relationship between resting brain activity, amygdala connectivity and emotion recognition – A resting state fMRI study. *Frontiers in Behavioral Neuroscience, 16*, 75796. https://doi.org/10.3389/FNBEH.2022.775796

Menzies, F. M., Henriquez, F. L., Alexander, J., & Roberts, C. W. (2011). Selective inhibition and augmentation of alternative macrophage activation by progesterone. *Immunology*, *134*(3), 281–291. https://doi.org/10.1111/J.1365-

2567.2011.03488.X

Michel, K. G., Huijbregts, R. P. H., Gleason, J. L., Richter, H. E., & Hel, Z. (2015). Effect of hormonal contraception on the function of plasmacytoid dendritic cells and distribution of immune cell populations in the female reproductive tract. *Journal of Acquired Immune Deficiency Syndromes*, *68*(5), 511–518. https://doi.org/10.1097/QAI.0000000000000531

Miguel, R. D. V., Calla, N. E. Q., & Cherpes, T. L. (2018). Levonorgestrel and female genital tract immunity: Time for a closer look. *The Journal of Infectious Diseases*, *218*(9), 1517–1518. https://doi.org/10.1093/INFDIS/JIY363

Miłkowska, K., Galbarczyk, A., & Jasienska, G. (2019). Disgust sensitivity in relation to menstrual cycle phase in women with and without an infection. *American Journal of Human Biology*, *31*(3), e23233. https://doi.org/10.1002/AJHB.23233

Miłkowska, K., Galbarczyk, A., Klimek, M., Zabłocka-Słowińska, K., & Jasienska, G. (2021). Pathogen disgust, but not moral disgust, changes across the menstrual cycle. *Evolution and Human Behavior*, *42*(5), 402–408. https://doi.org/10.1016/J.EVOLHUMBEHAV.2021.03.002

Miller, S. L., & Maner, J. K. (2011). Sick body, vigilant mind: The biological immune system activates the behavioral immune system. *Psychological Science*, *22*(12), 1467–1471. https://doi.org/10.1177/0956797611420166

Miller, R., & Plessow, F. (2013). Transformation techniques for cross-sectional and longitudinal endocrine data: Application to salivary cortisol concentrations. *Psychoneuroendocrinology*, *38*(6), 941-946. https://doi.org/10.1016/j.psyneuen.2012.09.013

Miller, R., Plessow, F., Rauh, M., Gröschl, M., & Kirschbaum, C. (2013). Comparison of salivary cortisol as measured by different immunoassays and tandem mass spectrometry. *Psychoneuroendocrinology*, *38*(1), 50-57. https://doi-org.proxy.lib.umich.edu/10.1016/j.psyneuen.2012.04.019

Mitchell, V. E., & Welling, L. L. M. (2020). Not all progestins are created equally: Considering unique progestins individually in psychobehavioral research. *Adaptive Human Behavior and Physiology*, *6*(3), 381–412. https://doi.org/10.1007/S40750-020-00137-1

Moldenhauer, L. M., Diener, K. R., Thring, D. M., Brown, M. P., Hayball, J. D., & Robertson, S. A. (2009). Cross-presentation of male seminal fluid antigens elicits T cell activation to initiate the female immune response to pregnancy. *The Journal of Immunology*, *182*(12), 8080-8093. https://doi.org/10.4049/jimmunol.0804018

Moran, J. B., Goh, J. X., Kerry, N., & Murray, D. R. (2021). Outbreaks and outgroups: Three tests of the relationship between disease avoidance motives and xenophobia during an emerging pandemic. *Evolutionary Psychological Science*, *7*(4), 419-429. https://doi.org/10.1007/s40806-021-00283-z

Mortensen, C. R., Becker, D. V., Ackerman, J. M., Neuberg, S. L., & Kenrick, D. T. (2010). Infection breeds reticence: The effects of disease salience on self-perceptions of personality and behavioral avoidance tendencies. *Psychological Science*, *21*(3), 440-447. https://doi.org/10.1177/0956797610361706

Mosmann, T. R. M., & Coffman, R. L. (1989). TH1 and TH2 cells: Different patterns of lymphokine secretion lead to different functional properties. *Annual Review of*

Immunology, *7*(1), 145–173. https://doi.org/10.1146/annurev.iy.07.040189.001045

Muehlenbein, M., Gassen, J., Nowak, T., Henderson, A., Thum, E., Weaver, S., & Baker, E. (2022). Exploring links between pathogen avoidance motivation, COVID-19 case counts, and immune function. *American Journal of Human Biology*, *35*(3), e23833. https://doi.org/10.1002/ajhb.23833

Mullett, T. L., Brown, G. D., Fincher, C. L., Kosinski, M., & Stillwell, D. (2020). Individual-level analyses of the impact of parasite stress on personality: Reduced openness only for older individuals. *Personality and Social Psychology Bulletin*, *46*(1), 79-93. https://doi.org/10.1177/0146167219843918

Mummendey, A., Klink, A., & Brown, R. (2001). Nationalism and patriotism: National identification and out-group rejection. *British Journal of Social Psychology*, *40*(2), 159-172. https://doi.org/10.1348/014466601164740

Murakami, M., Kamimura, D., & Hirano, T. (2019). Pleiotropy and specificity: Insights from the Interleukin 6 family of cytokines. *Immunity*, *50*(4), 812–831. https://doi.org/10.1016/J.IMMUNI.2019.03.027

Murray, D. R., Prokosch, M. L., & Airington, Z. (2019). PsychoBehavioroimmunology: Connecting the behavioral immune system to its physiological foundations. *Frontiers in Psychology*, *10*, 200. https://doi.org/10.3389/fpsyg.2019.00200

Murray, D. R., & Schaller, M. (2012). Threat(s) and conformity deconstructed: Perceived threat of infectious disease and its implications for conformist attitudes and behavior. *European Journal of Social Psychology*, *42*(2), 180-188. https://doi.org/10.1002/ejsp.863

Murray, D. R., Schaller, M., & Suedfeld, P. (2013). Pathogens and politics: Further evidence that parasite prevalence predicts authoritarianism. *PLoS ONE, 8*(5), e62275. https://doi.org/10.1371/journal.pone.0062275

Murray, D. R., Trudeau, R., & Schaller, M. (2011). On the origins of cultural differences in conformity: Four tests of the pathogen prevalence hypothesis. *Personality and Social Psychology Bulletin, 37*(3), 318-329. https://doi.org/10.1177/0146167210394451

Nahas, G. G., Tannieres, M. L., & Lennon, J. F. (2016). Direct measurement of leukocyte motility: Effects of pH and temperature. *Experimental Biology and Medicine, 138*(1), 350-352. https://doi.org/10.3181/00379727-138-35894

Nakashima, A., Ito, M., Yoneda, S., Shiozaki, A., Hidaka, T., & Saito, S. (2010). Circulating and decidual th17 cell levels in healthy pregnancy. *American Journal of Reproductive Immunology*, *63*(2), 104–109. https://doi.org/10.1111/J.1600-0897.2009.00771.X

Nam, H. H., Sawyer, K., & Style, H. K. (2024). Understanding anti-Asian sentiment and political behavior in the wake of COVID-19. *Politics, Groups, and Identities, 12*(2), 395-414. https://doi.org/10.1080/21565503.2022.2137051

Navarrete, C. D., & Fessler, D. M. T. (2006). Disease avoidance and ethnocentrism: The effects of disease vulnerability and disgust sensitivity on intergroup attitudes. *Evolution and Human Behavior, 27*(4), 270-282. https://doi.org/10.1016/j.evolhumbehav.2005.12.001

Navarrete, C. D., Fessler, D. M. T., & Eng, S. J. (2007). Elevated ethnocentrism in the first trimester of pregnancy. *Evolution and Human Behavior, 28*(1), 60-65.

https://doi.org/10.1016/j.evolhumbehav.2006.06.002

Nesse, R. M. (2001). The smoke detector principle: Natural selection and the regulation of defensive responses. *Annals of the New York Academy of Science, 935,* 75-85.

Nesse, R. M. (2005). Natural selection and the regulation of defenses: A signal detection analysis of the smoke detector principle. *Evolution and Human Behavior, 26*(1), 88-105. https://doi.org/10.1016/j.evolhumbehav.2004.08.002

Neuberg, S. L., Kenrick, D. T., & Schaller, M. (2011). Human threat management systems: Self-protection and disease avoidance. *Neuroscience & Biobehavioral Reviews*, *35*(4), 1042–1051. https://doi.org/10.1016/J.NEUBIOREV.2010.08.011

Neuberg, S. L., & Schaller, M. (2016). An evolutionary threat-management approach to prejudices. *Current Opinion in Psychology, 7*, 1-5. https://doi.org/10.1016/j.copsyc.2015.06.004

Neuliep, J. W. (2002). Assessing the reliability and validity of the Generalized Ethnocentrism Scale. *Journal of Intercultural Communication Research, 31,* 201-215.

Neuliep, J. W., Hintz, S. M., & McCroskey, J. C. (2005). The influence of ethnocentrism in organizational contexts: Perceptions on interviewee and managerial attractiveness, credibility, and effectiveness. *Communication Quarterly, 53*(1), 41-56.

Neuliep, J. W., & McCroskey, J. C. (1997). The development of a U.S. and generalized ethnocentrism scale. *Communication Research Reports, 14,* 385-398. https://doi.org/10.1080/08824099709388682

Ng, S. C., Gilman-Sachs, A., Thaker, P., Beaman, K. D., Beer, A. E., & Kwak-Kim, J.

(2002). Expression of intracellular Th1 and Th2 cytokines in women with recurrent spontaneous abortion, implantation failures after IVF/ET or normal pregnancy. *American Journal of Reproductive Immunology*, *48*(2), 77–86. https://doi.org/10.1034/J.1600-0897.2002.01105.X

Nguyen, T. T., Criss, S., Dwivedi, P., Huang, D., Keralis, J., Hsu, E., Phan, L., Nguyen, L. H., Yardi, I., Glymour, M. M., Allen, A. M., Chae, D. H., Gee, G. C., & Nguyen, Q. C. (2020). Exploring U.S. shifts in anti-Asian sentiment with the emergence of COVID-19. *International Journal of Environmental Research and Public Health, 17*(19), 7032. https://doi.org/10.3390/ijerph17197032

Nilsberth, C., Elander, L., Hamzic, N., Norell, M., Lönn, J., Engström, L., & Blomqvist, A. (2009). The role of interleukin-6 in lipopolysaccharide-induced fever by mechanisms independent of prostaglandin e2. *Endocrinology*, *150*(4), 1850–1860. https://doi.org/10.1210/EN.2008-0806

Nussbaum, M. C. (2010). *From disgust to humanity: Sexual orientation and constitutional law.* Oxford University Press.

O'Fallon, B. D., & Fehren-Schmitz, L. (2011). Native Americans experienced a strong population bottleneck coincident with European contact. *Proceedings of the National Academy of Sciences of the United States of America, 108*(51), 20444-20448. https://doi.org/10.1073/pnas.1112563108

O'Shea, B. A., Watson, D. G., Brown, G. D., & Fincher, C. L. (2020). Infectious disease prevalence, not race exposure, predicts both implicit and explicit racial prejudice across the United States. *Social Psychological and Personality Science, 11*(3), 345-355. https://doi.org/10.1177/1948550619862319

Oaten, M. J., Stevenson, R. J., & Case, T. I. (2017). Compensatory up-regulation of behavioral disease avoidance in immuno-compromised people with rheumatoid arthritis. *Evolution and Human Behavior*, *38*(3), 350–356. https://doi.org/10.1016/J.EVOLHUMBEHAV.2016.11.006

Oaten, M., Stevenson, R. J., & Case, T. I. (2009). Disgust as a disease-avoidance mechanism. *Psychological Bulletin*, *135*(2), 303-321. https://doi.org/10.1037/a0014823

Öberg, S., & Rönnbäck, K. (2016). Mortality among European settlers in pre-colonial West Africa: The "White Man's Grave" revisited. *Göteborg Papers in Economic History, 20*

Odell, W. D., & Molitch, M. E. (1974). The pharmacology of contraceptive agents. *Annual Review of Pharmacology*, *14*(1), 413-434. https://doi.org/10.1146/annurev.pa.14.040174.002213

Ogden, J., & Zoukas, S. (2010). Generating physical symptoms from visual cues: An experimental study. *Psychology, Health & Medicine*, *14*(6), 695-704. https://doi.org/10.1080/13548500903311547

Olatunji, B. O., Cox, R. C., & Li, I. (2020). Disgust regulation between menstrual cycle phases: Differential effects of emotional suppression and reappraisal. *Journal of Behavior Therapy and Experimental Psychiatry*, *68*, 101543. https://doi.org/10.1016/J.JBTEP.2019.101543

Olatunji, B. O., Lohr, J. M., Sawchuk, C. N., & Tolin, D. F. (2007a). Multimodal assessment of disgust in contamination-related obsessive-compulsive disorder. *Behaviour Research and Therapy*, *45*(2), 263–276.

https://doi.org/10.1016/J.BRAT.2006.03.004

Olatunji, B. O., Williams, N. L., Tolin, D. F., Abramowitz, J. S., Sawchuk, C. N., Lohr, J. M., & Elwood, L. S. (2007b). The Disgust Scale: Item analysis, factor structure, and suggestions for refinement. *Psychological Assessment*, *19*(3), 281–297. https://doi.org/10.1037/1040-3590.19.3.281

Olivera-La Rosa, A., Chuquichambi, E. G., & Ingram, G. P. (2020). Keep your (social) distance: Pathogen concerns and social perception in the time of COVID-19. *Personality and Individual Differences*, *166*, 110200. https://doi.org/10.1016/j.paid.2020.110200

Orefice, R. (2021). Immunology and the immunological response in pregnancy. *Best Practice & Research Clinical Obstetrics & Gynaecology*, *76*, 3–12. https://doi.org/10.1016/J.BPOBGYN.2020.07.013

Osborne, L. M., Brar, A., & Klein, S. L. (2019). The role of Th17 cells in the pathophysiology of pregnancy and perinatal mood and anxiety disorders. *Brain, Behavior, and Immunity, 76*, 7-16. https://doi.org/10.1016/j.bbi.2018.11.015

Osum, K. C., & Jenkins, M. K. (2023). Toward a general model of CD4+ T cell subset specification and memory cell formation. *Immunity*, *56*(3), 475–484. https://doi.org/10.1016/J.IMMUNI.2023.02.010

Pace, J. L., Russell, S. W., Schreiber, R. D., Altman, A., & Katz, D. H. (1983). Macrophage activation: Priming activity from a T-cell hybridoma is attributable to interferon-gamma. *Proceedings of the National Academy of Sciences of the United States of America*, *80*(12), 3782–3786. https://doi.org/10.1073/PNAS.80.12.3782

Paoletti, A. M., Lello, S., Fratta, S., Orrù, M., Ranuzzi, F., Sogliano, C., Concas, A., Biggio, G., & Melis, G. B. (2004). Psychological effect of the oral contraceptive formulation containing 3 mg of drospirenone plus 30 μg of ethinyl estradiol. *Fertility and Sterility*, *81*(3), 645-651. https://doi-org.proxy.lib.umich.edu/10.1016/j.fertnstert.2003.08.030

Park, J. H., Faulkner, J., & Schaller, M. (2003) Evolved disease-avoidance processes and contemporary anti-social behavior: Prejudicial attitudes and avoidance of people with physical disabilities. *Journal of Nonverbal Behavior*, *27*(2), 65-87. http://doi.org/10.1023/A:1023910408854

Park, J. H., Schaller, M., & Crandall, C. S. (2007). Pathogen-avoidance mechanisms and the stigmatization of obese people. *Evolution and Human Behavior*, *28*(6), 410-414. https://doi.org/10.1016/j.evolhumbehav.2007.05.008

Pazhoohi, F., & Kingstone, A. (2021). Associations of political orientation, xenophobia, right-wing authoritarianism, and concern of COVID-19: Cognitive responses to an actual pathogen threat. *Personality and Individual Differences*, *182*, 111081. https://doi.org/10.1016/j.paid.2021.111081

Pecchi, E., Dallaporta, M., Jean, A., Thirion, S., & Troadec, J. D. (2009). Prostaglandins and sickness behavior: Old story, new insights. *Physiology & Behavior*, *97*(3–4), 279–292. https://doi.org/10.1016/J.PHYSBEH.2009.02.040

Peng, M., & Bai, X. (2024). Ingroup favoritism on perceived risk of infectious diseases. *Risk Analysis*, *44*(2), 285-294. https://doi.org/10.1111/risa.14183

Petersen, M. B. (2017). Healthy out-group members are represented psychologically as infected in-group members. *Psychological Science*, *28*, 1857-1863.

https://doi.org/10.1177/0956797617728270

Philibert, D., Bouchoux, F., Degryse, M., Lecaque, D., Petit, F., & Gaillard, M. (1999). The pharmacological profile of a novel norpregnane progestin (trimegestone). *Gynecological Endocrinology*, *13*(5), 316–326. https://doi.org/10.3109/09513599909167574

Piccinni, M. P., Giudizi, M. G., Biagiotti, R., Beloni, L., Giannarini, L., Sampognaro, S., Parronchi, P., Manetti, R., Annunziato, F., & Livi, C. (1995). Progesterone favors the development of human T helper cells producing Th2-type cytokines and promotes both IL-4 production and membrane CD30 expression in established Th1 cell clones. *The Journal of Immunology*, *155*(1), 128–133. https://doi.org/10.4049/JIMMUNOL.155.1.128

Piccinni, M. P., Lombardelli, L., Logiodice, F., Kullolli, O., Romagnani, S., & Bouteiller, P. Le. (2015). T helper cell mediated-tolerance towards fetal allograft in successful pregnancy. *Clinical and Molecular Allergy*, *13*(1), 1–5. https://doi.org/10.1186/s12948-015-0015-y

Plakias, A. (2018). The response model of moral disgust. *Synthese, 195*, 5453-5472. https://doi.org/10.1007/s11229-017-1455-3

Pluchino, N., Lenzi, E., Merlini, S., Giannini, A., Cubeddu, A., Casarosa, E., Begliuomini, S., Luisi, M., Cela, V., & Genazzani, A. R. (2009). Selective effect of chlormadinone acetate on brain allopregnanolone and opioids content. *Contraception, 80*(1), 53-62. https://doi.org. /10.1016/j.contraception.2009.01.005

Pluchino, N., Santoro, A., Casarosa, E., Wenger, J. M., Genazzani, A. D., Petignat, P., & Genazzani, A. R. (2013). Advances in neurosteroids: Role in clinical practice.

Climacteric, *16*(1), 8–17. https://doi.org/10.3109/13697137.2013.809647

Polis, C. B., Curtis, K. M., Hannaford, P. C., Phillips, S. J., Chipato, T., Kiarie, J. N., Westreich, D. J., & Steyn, P. S. (2016). An updated systematic review of epidemiological evidence on hormonal contraceptive methods and HIV acquisition in women. *AIDS*, *30*(17), 2665–2683. https://doi.org/10.1097/QAD.0000000000001228

Porcu, P., Serra, M., & Concas, A. (2019). The brain as a target of hormonal contraceptives: Evidence from animal studies. *Frontiers in Neuroendocrinology*, *55*, 100799. https://doi.org/10.1016/j.yfrne.2019.100799

Prokop, P., Usak, M., & Fančovičová, J. (2010). Risk of parasite transmission influences perceived vulnerability to disease and perceived danger of disease-relevant animals. *Behavioural Processes*, *85*(1), 52-57. https://doi.org/10.1016/j.beproc.2010.06.006

Quispe Calla, N. E., Vicetti Miguel, R. D., Boyaka, P. N., Hall-Stoodley, L., Kaur, B., Trout, W., Pavelko, S. D., & Cherpes, T. L. (2016a). Medroxyprogesterone acetate and levonorgestrel increase genital mucosal permeability and enhance susceptibility to genital herpes simplex virus type 2 infection. *Mucosal Immunology*, *9*(6), 1571–1583. https://doi.org/10.1038/mi.2016.22

Quispe Calla, N. E., Vicetti Miguel, R. D., Mei, A., Fan, S., Gilmore, J. R., & Cherpes, T. L. (2016b). Dendritic cell function and pathogen-specific T cell immunity are inhibited in mice administered levonorgestrel prior to intranasal Chlamydia trachomatis infection. *Scientific Reports*, *6*(1), 37723. https://doi.org/10.1038/srep37723

Quispe Calla, N. E., Vicetti Miguel, R. D., Torres, A. R., Trout, W., Gabriel, J. M., Hatfield, A. M., Aceves, K. M., Kwiek, J. J., Kaur, B., & Cherpes, T. L. (2020). Norethisterone enanthate increases mouse susceptibility to genital infection with Herpes Simplex Virus Type 2 and HIV Type 1. *ImmunoHorizons*, *4*(2), 72–81. https://doi.org/10.4049/IMMUNOHORIZONS.1900077

Radin, A. S., Kuhlman, K. R., Boyle, C. C., Haydon, M. D., & Bower, J. E. (2021). Using the influenza vaccine as a mild, exogenous inflammatory challenge: When does inflammation peak? *Brain, Behavior, & Immunity-Health*, *13*, 100239. https://doi.org/10.1016/j.bbih.2021.100239

Radke, S., & Derntl, B. (2016). Affective responsiveness is influenced by intake of oral contraceptives. *European Neuropsychopharmacology*, *26*(6), 1014–1019. https://doi.org/10.1016/J.EURONEURO.2016.03.004

Rafiee, Y., Jones, B. C., & Shiramizu, V. (2023). Is pathogen disgust increased on days of the menstrual cycle when progesterone is high? Evidence from a between-subjects study using estimated progesterone levels. *Adaptive Human Behavior and Physiology*, *9*(1), 26–36. https://doi.org/10.1007/S40750-022-00208-5

Raghupathy, R. (1997). Th1-type immunity is incompatible with successful pregnancy. *Immunology Today*, *18*(10), 478–482. https://doi.org/10.1016/S0167-5699(97)01127-4

Raghupathy, R. (2001). Pregnancy: Success and failure within the Th1/Th2/Th3 paradigm. *Seminars in Immunology*, *13*(4), 219–227. https://doi.org/10.1006/SMIM.2001.0316

Raghupathy, R., & Szekeres-Bartho, J. (2022). Progesterone: A unique hormone with

immunomodulatory roles in pregnancy. *International Journal of Molecular Sciences, 23*(3), 1333. https://doi.org/10.3390/IJMS23031333

Rahimi, K., Ahmadi, A., Hassanzadeh, K., Soleimani, Z., Sathyapalan, T., Mohammadi, A., & Sahebkar, A. (2019). Targeting the balance of T helper cell responses by curcumin in inflammatory and autoimmune states. *Autoimmunity Reviews, 18*(7), 738-748. https://doi.org/10.1016/j.autrev.2019.05.012

Rapkin, A. J., Biggio, G., & Concas, A. (2006). Oral contraceptives and neuroactive steroids. *Pharmacology Biochemistry and Behavior, 84*(4), 628-634. https://doi-org.proxy.lib.umich.edu/10.1016/j.pbb.2006.06.008

Reicher, S. D., Templeton, A., Neville, F., Ferrari, L., & Drury, J. (2016). Core disgust is attenuated by ingroup relations. *Proceedings of the National Academy of Sciences, 113*(10), 2631-2635. https://doi.org/10.1073/pnas.1517027113

Reid, S. A., Zhang, J., Anderson, G. L., Gasiorek, J., Bonilla, D., & Peinado, S. (2012). Parasite primes make foreign-accented English sound more distant to people who are disgusted by pathogens (but not by sex or morality). *Evolution and Human Behavior, 33*(5), 471-478. https://doi.org/10.1016/j.evolhumbehav.2011.12.009

Reis, F. M., Coutinho, L. M., Vannuccini, S., Batteux, F., Chapron, C., & Petraglia, F. (2020). Progesterone receptor ligands for the treatment of endometriosis: the mechanisms behind therapeutic success and failure. *Human Reproduction Update, 26*(4), 565–585. https://doi.org/10.1093/HUMUPD/DMAA009

Reny, T. T., & Barreto, M. A. (2022). Xenophobia in the time of pandemic: Othering, anti-Asian attitudes, and COVID-19. *Politics, Groups, and Identities, 10*(2), 209-232. https://doi.org/10.1080/21565503.2020.1769693

Rhodes, G., Yoshikawa, S., Palermo, R., Simmons, L. W., Peters, M., Lee, K., Halberstadto, J., & Crawford, J. R. (2007). Perceived health contributes to the attractiveness of facial symmetry, averageness, and sexual dimorphism. *Perception, 36*(8), 1244–1252. https://doi.org/10.1068/p5712

Rimer, J., Cohen, I. R., & Friedman, N. (2014). Do all creatures possess an acquired immune system of some sort? *Bioessays, 36*(3), 273–281. https://doi.org/10.1002/bies.201300124

Rivera, L. M., Vu, H. A., & Backstrom, V. L. (2024). Self-esteem, ingroup favoritism, and outgroup evaluations: A meta-analysis. *Group Processes & Intergroup Relations, 27*(7), 1569-1588. https://doi.org/10.1177/13684302231210496

Rivest, S. (2001). How circulating cytokines trigger the neural circuits that control the hypothalamic–pituitary–adrenal axis. *Psychoneuroendocrinology*, *26*(8), 761–788. https://doi.org/10.1016/S0306-4530(01)00064-6

Robertson, S. A., Care, A. S., & Moldenhauer, L. M. (2018). Regulatory T cells in embryo implantation and the immune response to pregnancy. *The Journal of Clinical Investigation, 128*(10), 4224-4235. https://doi.org/10.1172/JCI122182

Robertson, S. A., Guerin, L. R., Bromfield, J. J., Branson, K. M., Ahlström, A. C., & Care, A. S. (2009). Seminal fluid drives expansion of the $CD4^{+}CD25^{+}$ T regulatory cell pool and induces tolerance to paternal alloantigens in mice. *Biology of Reproduction*, *80*(5), 1036-1045. https://doi.org/10.1095/biolreprod.108.074658

Romano, A., Balliet, D., Yamagishi, T., & Liu, J. H. (2017). Parochial trust and cooperation across 17 societies. *Proceedings of the National Academy of*

Sciences, 114(48), 12702-12707. https://doi.org/10.1073/pnas.1712921114

Rosenkranz-Weiss, P., Sessa, W. C., Milstien, S., Kaufman, S., Watson, C. A., & Pober, J. S. (1994). Regulation of nitric oxide synthesis by proinflammatory cytokines in human umbilical vein endothelial cells. Elevations in tetrahydrobiopterin levels enhance endothelial nitric oxide synthase specific activity. *Journal of Clinical Investigation, 93*(5), 2236–2243. https://doi.org/10.1172/jci117221

Rozin, P., Haidt, J., & McCauley, C. (2009). Disgust: The body and soul emotion in the 21st century. In B. O. Olatunji & D. McKay (Eds.) *Disgust and its disorders: Theory, assessment, and treatment implications* (pp. 9-29). American Psychological Association. https://doi.org/10.1037/11856-001

Rummel, C., Matsumura, K., & Luheshi, G. N. (2011). Circulating IL-6 contributes to peripheral LPS-induced mPGES-1 expression in the rat brain. *Brain Research Bulletin*, *86*(5–6), 319–325. https://doi.org/10.1016/J.BRAINRESBULL.2011.09.006

Ryan, S., Oaten, M., Stevenson, R. J., & Case, T. I. (2012). Facial disfigurement is treated like an infectious disease. *Evolution and Human Behavior, 33*, 639-646.

Saito, S., Nakashima, A., Shima, T., & Ito, M. (2010). Th1/Th2/Th17 and regulatory t-cell paradigm in pregnancy. *American Journal of Reproductive Immunology*, *63*(6), 601–610. https://doi.org/10.1111/J.1600-0897.2010.00852.X

Saito, S., & Sakai, M. (2003). Th1/Th2 balance in preeclampsia. *Journal of Reproductive Immunology*, *59*(2), 161–173. https://doi.org/10.1016/S0165-0378(03)00045-7

Salama, K. M., Alloush, M. K., & Al hussini, R. M. (2020). Are the cytokines TNF alpha and IL 1Beta early predictors of embryo implantation? Cross sectional study.

Journal of Reproductive Immunology, *137*. https://doi.org/10.1016/J.JRI.2019.102618

Salimetrics. (2019). Salivary IL-6 enzyme immunoassay kit insert (Manual No. 1-3602). Salimetrics.

Satoh, M., Nakamura, M., Tamura, G., Makita, S., Segawa, I., Tashiro, A., Satodate, R., & Hiramori, K. (1997). Inducible nitric oxide synthase and tumor necrosis factor-alpha in myocardium in human dilated cardiomyopathy. *Journal of the American College of Cardiology*, *29*(4), 716–724. https://doi.org/10.1016/S0735-1097(96)00567-0

Sawada, N., Auger, E., & Lydon, J. E. (2018). Activation of the behavioral immune system: Putting the brakes on affiliation. *Personality and Social Psychology Bulletin, 44*(2), 224-237. https://doi.org/10.1177/0146167217736046

Scanlan, J. M., Werner, J. J., Legg, R. L., & Laudenslager, M. L. (1995). Natural killer cell activity is reduced in association with oral contraceptive use. *Psychoneuroendocrinology, 20*(3), 281–287. https://doi.org/10.1016/0306-4530(94)00059-J

Schaller, M. (2011). The behavioural immune system and the psychology of human sociality. *Philosophical Transactions of the Royal Society B: Biological Sciences, 366*(1583), 3418–3426. https://doi.org/10.1098/RSTB.2011.0029

Schaller, M., & Duncan, L. A. (2007). *The behavioral immune system: Its evolution and social psychological implications.* In J. P. Forgas, M. G. Haselton, & W. von Hippel (Eds.), Evolution and the social mind: Evolutionary psychology and social cognition (pp. 293–307). Routledge/Taylor & Francis Group.

Schaller, M., Miller, G. E., Gervais, W. M., Yager, S., & Chen, E. (2010). Mere visual perception of other people's disease symptoms facilitates a more aggressive immune response. *Psychological Science, 21*(5), 649–652. https://doi.org/10.1177/0956797610368064

Schaller, M., & Murray, D. R. (2008). Pathogens, personality, and culture: Disease prevalence predicts worldwide variability in sociosexuality, extraversion, and openness to experience. *Journal of Personality and Social Psychology, 95*(1), 212-221. https://psycnet.apa.org/doi/10.1037/0022-3514.95.1.212

Schaller, M., Murray, D., & Hofer, M. (2021). The behavioural immune system and pandemic psychology: The evolved psychology of disease-avoidance and its implications for attitudes, behaviour, and public health during epidemic outbreaks. *European Review of Social Psychology, 33*, 360 - 396. https://doi.org/10.1080/10463283.2021.1988404

Schaller, M., & Park, J. H. (2011). The Behavioral Immune System (and why it matters). *Current Directions in Psychological Science*, *20*(2), 99–103. https://doi.org/10.1177/0963721411402596

Schaller, M., Park, J. H., & Kenrick, D. T. (2007). Human evolution and social cognition. In R. I. M. Dunbar & L. Barrett (Eds.), *Oxford Handbook of Evolutionary Psychology* (pp. 491-504). Oxford University Press.

Schmitt, N., & Ueno, H. (2015). Regulation of human helper t cell subset differentiation by cytokines. *Current Opinion in Immunology*, *34*, 130. https://doi.org/10.1016/J.COI.2015.03.007

Scott, R. (2025). Why are graduates more socially liberal? Estimating the effect of higher

education on political values through variation in university experience. *Political Studies, 73*(2), 885-903. https://doi.org/10.1016/j.electstud.2022.102471

Sears, D. O., & Henry, P. J. (2005). Over thirty years later: A contemporary look at symbolic racism. In M. P. Zanna (Ed.), *Advances in Experimental Social Psychology* (Vol. 37; pp. 95-150). Elsevier Academic Press.

Shapouri, S. (2023). Of germs and culture; Parasite stress as the origin of individualism-collectivism. *Evolutionary Psychological Science, 9*, 82-89. https://doi.org/10.1007/s40806-022-00335-y

Sharkey, D. J., Tremellen, K. P., Jasper, M. J., Gemzell-Danielsson, K., & Robertson, S. A. (2012). Seminal fluid induces leukocyte recruitment and cytokine and chemokine mRNA expression in the human cervix after coitus. *The Journal of Immunology*, *188*(5), 2445-2454. https://doi.org/10.4049/jimmunol.1102736

Sherman, P. W., & Billing, J. (1999). Darwinian gastronomy: Why we use spices: Spices taste good because they are good for us. *BioScience, 49*(6), 453-463. https://doi.org/10.2307/1313553

Shetty, N. P., & Shetty, P. S. (2020). Epidemiology of disease in the tropics. *Manson's Tropical Diseases*, 19.

Shevyrev, D., & Tereshchenko, V. (2020). Treg heterogeneity, function, and homeostasis. *Frontiers in Immunology*, *10*, 3100. https://doi.org/10.3389/FIMMU.2019.03100

Shindo, T., Ikeda, U., Ohkawa, F., Kawahara, Y., Yokoyama, M., & Shimada, K. (1995). Nitric oxide synthesis in cardiac myocytes and fibroblasts by inflammatory cytokines. *Cardiovascular Research*, *29*(6), 813–819.

https://doi.org/10.1016/S0008-6363(96)88617-2

Shook, N. J., Sevi, B., Lee, J., Oosterhoff, B., & Fitzgerald, H. N. (2020). Disease avoidance in the time of COVID-19: The behavioral immune system is associated with concern and preventative health behaviors. *PLOS ONE, 15*(8), e0238015. https://doi.org/10.1371/JOURNAL.PONE.0238015

Shook, N. J., Thomas, R., & Ford, C. G. (2019). Testing the relation between disgust and general avoidance behavior. *Personality and Individual Differences, 150*, 109457. https://doi.org/10.1016/j.paid.2019.05.063

Silasi, M., Cardenas, I., Kwon, J. Y., Racicot, K., Aldo, P., & Mor, G. (2015). Viral infections during pregnancy. *American Journal of Reproductive Immunology, 73*(3), 199–213. https://doi.org/10.1111/AJI.12355

Silk, B. J., Date, K. A., Jackson, K. A., Pouillot, R., Holt, K. G., Graves, L. M., Ong, K. L., Hurd, S., Meyer, R., Marcus, R., Shiferaw, B., Norton, D. M., Medus, C., Zansky, S. M., Cronquist, A. B., Henao, O. L., Jones, T. F., Vugia, D. J., Farley, M. M., & Mahon, B. E. (2012). Invasive listeriosis in the foodborne diseases active surveillance network (FoodNet), 2004-2009: Further targeted prevention needed for higher-risk groups. *Clinical Infectious Diseases, 54*(5). https://doi.org/10.1093/cid/cis268

Silva, T. M., Cade, M. V., Figueiras, A., Roque, F., Herdeiro, M. T., & Devakumar, D. (2022). Impact of infectious disease epidemics on xenophobia: A systematic review. *Journal of Migration and Health, 5,* 100085. https://doi.org/10.1016/j.jmh.2022.100085

Skolnick, A. J., & Dzokoto, V. A. (2013). Disgust and contamination: A cross-national

comparison of Ghana and the United States. *Frontiers in Psychology*, *4*, https://doi.org/10.3389/fpsyg.2013.00091

Slavish, D. C., Graham-Engeland, J. E., Smyth, J. M., & Engeland, C. G. (2015). Salivary markers of inflammation in response to acute stress. *Brain, Behavior, and Immunity*, *44*, 253-269. http://dx.doi.org/10.1016/j.bbi.2014.08.008

Smith, J. L. (1999). Foodborne infections during pregnancy. *Journal of Food Protection*, *62*(7), 818–829. https://doi.org/10.4315/0362-028X-62.7.818

Soderberg, K. A., Payne, G. W., Sato, A., Medzhitov, R., Segal, S. S., & Iwasaki, A. (2005). Innate control of adaptive immunity via remodeling of lymph node feed arteriole. *Proceedings of the National Academy of Sciences*, *102*(45), 16315–16320. https://doi.org/10.1073/pnas.0506190102

Söylemez, S., & Kapucu, A. (2024). Disgust as a basic, sexual, and moral emotion. *Cognitive Processing*, *25*, 193-204. https://doi.org/10.1007/s10339-024-01180-6

Spriggs, D. R., Sherman, M. L., Michie, H., Arthur, K. A., Imamura, K., Wilmore, D., Hi, E. F., & Kufe, D. W. (1988). Recombinant human tumor necrosis factor administered as a 24-hour intravenous infusion. A phase I and pharmacologic study. *Journal of the National Cancer Institute*, *80*(13), 1039–1044. https://doi.org/10.1093/jnci/80.13.1039

Stanczyk, F. Z., Hapgood, J. P., Winer, S., & Mishell, D. R. (2013). Progestogens used in postmenopausal hormone therapy: Differences in their pharmacological properties, intracellular actions, and clinical effects. *Endocrine Reviews*, *34*(2), 171–208. https://doi.org/10.1210/ER.2012-1008

Stark, R., Walter, B., Schienle, A., & Vaitl, D. (2005). Psychophysiological correlates of

disgust and disgust sensitivity. *Journal of Psychophysiology, 19*(1), 50-60. https://doi.org/10.1027/0269-8803.19.1.50

Steinberg, B. E., Silverman, H. A., Robbiati, S., Gunasekaran, M. K., Tsaava, T., Battinelli, E., Stiegler, A., Bouton, C. E., Chavan, S. S., Tracey, K. J., & Huerta, P. T. (2016). Cytokine-specific neurograms in the sensory vagus nerve. *Bioelectronic Medicine*, *3*(1), 7–17. https://doi.org/10.15424/BIOELECTRONMED.2016.00007

Stern, J., Kordsmeyer, T. L., & Penke, L. (2021). A longitudinal evaluation of ovulatory cycle shifts in women's mate attraction and preferences. *Hormones and Behavior, 128*, 104916. https://doi.org/10.1016/j.yhbeh.2020.104916

Stern, J., & Shiramizu, V. (2022). Hormones, ovulatory cycle phase and pathogen disgust: A longitudinal investigation of the Compensatory Prophylaxis Hypothesis. *Hormones and Behavior*, *138*, 105103. https://doi.org/10.1016/J.YHBEH.2021.105103

Stevenson, R. J., Case, T. I., & Oaten, M. J. (2009). Frequency and recency of infection and their relationship with disgust and contamination sensitivity. *Evolution and Human Behavior*, *30*(5), 363–368. https://doi.org/10.1016/J.EVOLHUMBEHAV.2009.02.005

Stevenson, R. J., Hodgson, D., Oaten, M. J., Barouei, J., & Case, T. I. (2011). The effect of disgust on oral immune function. *Psychophysiology*, *48*(7), 900–907. https://doi.org/10.1111/J.1469-8986.2010.01165.X

Stevenson, R. J., Hodgson, D., Oaten, M. J., Moussavi, M., Langberg, R., Case, T. I., & Barouei, J. (2012). Disgust elevates core body temperature and up-regulates

certain oral immune markers. *Brain, Behavior, and Immunity*, *26*(7), 1160–1168. https://doi.org/10.1016/J.BBI.2012.07.010

Stevenson, R. J., Hodgson, D., Oaten, M. J., Sominsky, L., Mahmut, M., & Case, T. I. (2015). Oral immune activation by disgust and disease-related pictures. *Journal of Psychophysiology*, *29*(3), 119–129. https://doi.org/10.1027/0269-8803/a000143

Stevenson, R. J., Saluja, S., & Case, T. I. (2021). The impact of the covid-19 pandemic on disgust sensitivity. *Frontiers in Psychology*, *11*, 3895. https://doi.org/10.3389/FPSYG.2020.600761/BIBTEX

Straub, R. H., Cutolo, M., Buttgereit, F., & Pongratz, G. (2010). Energy regulation and neuroendocrine–immune control in chronic inflammatory diseases. *Journal of Internal Medicine*, *267*(6), 543–560. https://doi.org/10.1111/J.1365-2796.2010.02218.X

Szabo, Y. Z., Slavish, D. C., & Graham-Engeland, J. E. (2020). The effect of acute stress on salivary markers of inflammation: A systematic review and meta-analysis. *Brain, Behavior, and Immunity*, *88*, 887-900. https://doi.org/10.1016/j.bbi.2020.04.078

Szymkow, A., Frankowska, N., & Gałasińska, K. (2021). Social distancing from foreign individuals as a disease-avoidance mechanism: Testing the assumptions of the behavioral immune system theory during the COVID-19 pandemic. *Social Psychological Bulletin*, *16*(3), e4389. https://doi.org/10.32872/spb.4389

Tafuri, A., Alferink, J., Möller, P., Hämmerling, G. J., & Arnold, B. (1995). T cell awareness of paternal alloantigens during pregnancy. *Science*, *270*(5236), 630–633. https://doi.org/10.1126/SCIENCE.270.5236.630

Tajfel, H., Billig, M. G., Bundy, R. P., & Flament, C. (1971). Social categorization and intergroup behavior. *European Journal of Social Psychology, 1*(2), 149-178. https://doi.org/10.1002/ejsp.2420010202

Tajfel, H., & Turner, J. C. (1979). An integrative theory of intergroup conflict. In W. G. Austin & S. Worchel (Eds.), *The social psychology of intergroup relations* (pp. 33-48). Brooks/Cole.

Tang, C., & Hu, W. (2023). The role of Th17 and Treg cells in normal pregnancy and unexplained recurrent spontaneous abortion (URSA): New insights into immune mechanisms. *Placenta, 142,* 18-26. https://doi.org/10.1016/j.placenta.2023.08.065

Tapp, C., Oaten, M., Stevenson, R. J., Occhipinti, S., & Thandi, R. (2020). Is obesity treated like a contagious disease? *Journal of Applied Social Psychology*, *50*(4), 205-212. https://doi.org/10.1111/jasp.12650

Taylor, S. M., Van Eijk, A. M., Hand, C. C., Mwandagalirwa, K., Messina, J. P., Tshefu, A. K., Atua, B., Emch, M., Muwonga, J., Meshnick, S. R., & Ter Kuile, F. O. (2011). Quantification of the burden and consequences of pregnancy-associated Malaria in the Democratic Republic of the Congo. *The Journal of Infectious Diseases*, *204*(11), 1762–1771. https://doi.org/10.1093/INFDIS/JIR625

Terakawa, N., Hayashida, M., Shimizu, I., Ikegami, H., Wakimoto, H., Aono, T., Tanizawa, O., Matsumoto, K., & Nishida, M. (1987). Growth inhibition by progestins in a human endometrial cancer cell line with estrogen-independent progesterone receptors. *Cancer Research*, *47*(7), 1918–1923. https://doi.org/10.1016/0022-4731(87)90517-6

Terrizzi, J. A., Shook, N. J., & McDaniel, M. A. (2013). The behavioral immune system and social conservatism: A meta-analysis. *Evolution and Human Behavior, 34*(2), 99-108. https://doi.org/10.1016/j.evolhumbehav.2012.10.003

Terrizzi, J. A., Shook, N. J., & Ventis, W. L. (2010). Disgust: A predictor of social conservatism and prejudicial attitudes toward homosexuals. *Personality and Individual Differences, 49*(6), 587-592. https://doi.org/10.1016/j.paid.2010.05.024

Tesmer, L. A., Lundy, S. K., Sarkar, S., & Fox, D. A. (2008). Th17 cells in human disease. *Immunological Reviews, 223*(1), 87. https://doi.org/10.1111/J.1600-065X.2008.00628.X

Thornhill, R., & Fincher, C. L. (2014). The parasite-stress theory of sociality, the behavioral immune system, and human social and cognitive uniqueness. *Evolutionary Behavioral Sciences, 8*(4), 257-264. https://psycnet.apa.org/doi/10.1037/ebs0000020

Thornhill, R., Fincher, C. L., Murray, D. R., & Schaller, M. (2010). Zoonotic and non-zoonotic diseases in relation to human personality and societal values: Support for the parasite-stress model. *Evolutionary Psychology, 8*(2), 151-169. https://doi.org/10.1177/147470491000800201

Thornhill, R., & Gangestad, S. W. (2006). Facial sexual dimorphism, developmental stability, and susceptibility to disease in men and women. *Evolution and Human Behavior*, *27*(2), 131–144. https://doi.org/10.1016/J.EVOLHUMBEHAV.2005.06.001

Thorpe, S. J., Barnett, J., Friend, K., & Nottingham, K. (2011). The mediating roles of

disgust sensitivity and danger expectancy in relation to hand washing behaviour. *Behavioural and Cognitive Psychotherapy*, *39*(2), 175–190. https://doi.org/10.1017/S1352465810000676

Tilburgs, T., Roelen, D. L., van der Mast, B. J., de Groot-Swings, G. M., Kleijburg, C., Scherjon, S. A., & Claas, F. H. (2008). Evidence for a selective migration of fetus-specific $CD^4CD^{25bright}$ regulatory T cells from peripheral blood to the decidua in human pregnancy. *The Journal of Immunology, 180*(8), 5737-5745. https://doi.org/10.4049/jimmunol.180.8.5737

Timmer, C. J., & Mulders, T. M. T. (2000). Pharmacokinetics of etonogestrel and ethinyl estradiol released from a combined contraceptive vaginal ring. *Clinical Pharmacokinetics*, *39*(3), 233–242. https://doi.org/10.2165/00003088-200039030-00005

Tock, M. R., & Dryden, D. T. F. (2005). The biology of restriction and anti-restriction. *Current Opinion in Microbiology*, *8*(4), 466–472. https://doi.org/10.1016/J.MIB.2005.06.003

Tolin, D. F., Woods, C. M., & Abramowitz, J. S. (2006). Disgust sensitivity and obsessive–compulsive symptoms in a non-clinical sample. *Journal of Behavior Therapy and Experimental Psychiatry*, *37*(1), 30–40. https://doi.org/10.1016/J.JBTEP.2005.09.003

Tybur, J. M., & Gangestad, S. W. (2011). Mate preferences and infectious disease: Theoretical considerations and evidence in humans. *Philosophical Transactions of the Royal Society B: Biological Sciences*, *366*(1583), 3375–3388. https://doi.org/10.1098/RSTB.2011.0136

Tybur, J. M., Inbar, Y., Aarøe, L., Barclay, P., Barlow, F. K., de Barra, M., Becker, D. V., Borovoi, L., Choi, I., Choi, J. A., Consedine, N. S., Conway, A., Conway, J. R., Conway, P., Adoric, V. C., Demirci, D. E., Fernández, A. M., Ferreira, D. C. S., Ishii, K., Jakšić, I., Ji, T., van Leeuwen, F., Lewis, D. M. G., Li, N. P., McIntyre, J. C., Mukherjee, S., Park, J. H., Pawlowski, B., Petersen, M. B., Pizarro, D., Prodromitis, G., Prokop, P., Rantala, M. J., Reynolds, L. M., Sandin, B., Sevi, B., De Smet, D., Srinivasan, N., Tewari, S., Wilson, C., Yong, J. C., & Žeželj, I. (2016). Parasite stress and pathogen avoidance relate to distinct dimensions of political ideology across 30 nations. *Proceedings of the National Academy of Sciences of the United States of America*, *113*(44), 12408–12413. https://doi.org/10.1073/pnas.1607398113

Tybur, J. M., Jones, B. C., DeBruine, L. M., Ackerman, J. M., & Fasolt, V. (2020a). Preregistered direct replication of "Sick body, vigilant mind: The biological immune system activates the behavioral immune system." *Psychological Science*, *31*(11), 1461–1469. https://doi.org/10.1177/0956797620955209

Tybur, J. M., & Karinen, A. K. (2018). Measurement and theory in disgust sensitivity. *The SAGE handbook of personality and individual differences: Applications of personality and individual differences*, 159-179.

Tybur, J. M., Lieberman, D., & Griskevicius, V. (2009). Microbes, mating, and morality: Individual differences in three functional domains of disgust. *Journal of Personality and Social Psychology*, *97*(1), 103–122. https://doi.org/10.1037/a0015474

Tybur, J., Lieberman, D., Fan, L., Kupfer, T., & De Vries, R. (2020b). Behavioral

immune trade-offs: Interpersonal value relaxes social pathogen avoidance. *Psychological Science, 31,* 1211 - 1221. https://doi.org/10.1177/0956797620960011

U.S. Environmental Protection Agency (2000). *Guidance for data quality assessment: Practical methods for data analysis (EPA QA/G-9, QA00 Update*). https://www.epa.gov/sites/default/files/2015-06/documents/g9-final.pdf

van Kerkhove, M. D., Vandemaele, K. A. H., Shinde, V., Jaramillo-Gutierrez, G., Koukounari, A., Donnelly, C. A., Carlino, L. O., Owen, R., Paterson, B., Pelletier, L., Vachon, J., Gonzalez, C., Hongjie, Y., Zijian, F., Chuang, S. K., Au, A., Buda, S., Krause, G., Haas, W., … Mounts, A. W. (2011). Risk factors for severe outcomes following 2009 influenza a (h1n1) infection: A global pooled analysis. *PLOS Medicine*, *8*(7), e1001053. https://doi.org/10.1371/JOURNAL.PMED.1001053

van Leeuwen, F., Dukes, A., Tybur, J. M., & Park, J. H. (2017). Disgust sensitivity relates to moral foundations independent of political ideology. *Evolutionary Behavioral Sciences*, *11*(1), 92-98. https://psycnet.apa.org/doi/10.1037/ebs0000075

van Leeuwen, F., & Jaeger, B. (2022). Pathogen disgust sensitivity: Individual differences in pathogen perception or pathogen avoidance? *Motivation and Emotion, 46,* 394-403.

van Leeuwen, F., Jaeger, B., & Tybur, J. M. (2023). A behavioural immune system perspective on disgust and social prejudice. *Nature Reviews Psychology*, *2*, 676-687. https://doi.org/10.1038/s44159-023-00226-4

van Leeuwen, F., & Petersen, M. B. (2018). The behavioral immune system is designed to avoid infected individuals, not outgroups. *Evolution and Human Behavior, 39*(2), 226-234. https://doi.org/10.1016/j.evolhumbehav.2017.12.003

van Overveld, M., de Jong, P. J., & Peters, M. L. (2010). The Disgust Propensity and Sensitivity Scale – Revised: Its predictive value for avoidance behavior. *Personality and Individual Differences*, *49*(7), 706–711. https://doi.org/10.1016/J.PAID.2010.06.008

van Overveld, W. J. M., de Jong, P. J., Peters, M. L., Cavanagh, K., & Davey, G. C. L. (2006). Disgust propensity and disgust sensitivity: Separate constructs that are differentially related to specific fears. *Personality and Individual Differences*, *41*(7), 1241–1252. https://doi.org/10.1016/J.PAID.2006.04.021

Vartanian, L. R. (2010). Disgust and perceived control in attitudes toward obese people. *International Journal of Obesity*, *34*(8), 1302-1307. https://doi.org/10.1038/ijo.2010.45

Veenstra van Nieuwenhoven, A. L., Bouman, A., Moes, H., Heineman, M. J., de Leij, L. F. M. H., Santema, J., & Faas, M. M. (2002). Cytokine production in natural killer cells and lymphocytes in pregnant women compared with women in the follicular phase of the ovarian cycle. *Fertility and Sterility*, *77*(5), 1032–1037. https://doi.org/10.1016/S0015-0282(02)02976-X

Vetrano, M., Wegman, A., Koes, B., Mehta, S., & King, C. A. (2020). Serum IL-1RA levels increase from follicular to luteal phase of the ovarian cycle: A pilot study on human female immune responses. *PLoS One, 15*(9), e0238520. https://doi.org/10.1371/journal.pone.0238520

Vignali, D. A. A., Collison, L. W., & Workman, C. J. (2008). How regulatory T cells work. *Nature Reviews Immunology*, *8*, 523-532. https://doi.org/10.1038/nri2343

Wang, W., Sung, N., Gilman-Sachs, A., & Kwak-Kim, J. (2020). T helper (Th) cell profiles in pregnancy and recurrent pregnancy losses: Th1/Th2/Th9/Th17/Th22/Tfh cells. *Frontiers in Immunology*, *11*, 2025. https://doi.org/10.3389/fimmu.2020.02025

Watkins, L. R., Maier, S. F., & Goehler, L. E. (1995). Cytokine-to-brain communication: A review & analysis of alternative mechanisms. *Life Sciences*, *57*(11), 1011–1026. https://doi.org/10.1016/0024-3205(95)02047-M

Wegmann, T. G., Lin, H., Guilbert, L., & Mosmann, T. R. (1993). Bidirectional cytokine interactions in the maternal-fetal relationship: is successful pregnancy a TH2 phenomenon? *Immunology Today*, *14*(7), 353–356. https://doi.org/10.1016/0167-5699(93)90235-D

Welling, L. L. M. (2025). What's in a face? Sources of variation in human facial attractiveness. In R. Seth & P. D. Knott (Eds.), *Gender affirming surgery of the face and neck*. Nature Springer.

Welling, L. L., & Burriss, R. P. (2019). Investigating the ovulatory cycle. In L. L. M. Welling & T. K. Shackelford (Eds.), *The Oxford handbook of evolutionary psychology and behavioral endocrinology*, 107-123. Oxford University Press.

Welling, L. L. M., Conway, C. A., Debruine, L. M., & Jones, B. C. (2007). Perceived vulnerability to disease is positively related to the strength of preferences for apparent health in faces. *Journal of Evolutionary Psychology*, *5*(1–4), 131–139. https://doi.org/10.1556/JEP.2007.1012

Wu, B. P., & Chang, L. (2012). The social impact of pathogen threat: How disease salience influences conformity. *Personality and Individual Differences, 53,* 50-54. https://doi.org/10.1016/j.paid.2012.02.023

Wu, Q., Tan, C., Wang, B., & Zhou, P. (2015). Behavioral immune system and ingroup derogation: The effects of infectious diseases on ingroup derogation attitudes. *PloS One, 10*(3), e0122794. https://doi.org/10.1371/journal.pone.0122794

Yang, X., Tian, Y., Zheng, L., Luu, T., & Kwak-Kim, J. (2023). The update immune-regulatory role of pro- and anti-inflammatory cytokines in recurrent pregnancy losses. *International Journal of Molecular Science, 24*(1), 132. https://doi.org/10.3390/ijms24010132

Yovel, G., Shakhar, K., & Ben-Eliyahu, S. (2001). The effects of sex, menstrual cycle, and oral contraceptives on the number and activity of natural killer cells. *Gynecologic Oncology, 81*(2), 254–262. https://doi.org/10.1006/GYNO.2001.6153

Zakrzewska, M., Olofsson, J. K., Lindholm, T., Blomkvist, A., & Liuzza, M. T. (2019). Body odor disgust sensitivity is associated with prejudice towards a fictive group of immigrants. *Physiology & Behavior, 201,* 221-227. https://doi.org/10.1016/j.physbeh.2019.01.006

Zelaźniewicz, A., Borkowska, B., Nowak, J., & Pawłowski, B. (2016). The progesterone level, leukocyte count and disgust sensitivity across the menstrual cycle. *Physiology & Behavior, 161,* 60–65. https://doi.org/10.1016/J.PHYSBEH.2016.04.002

Zelaźniewicz, A., & Pawłowski, B. (2015). Disgust in pregnancy and fetus sex—

Longitudinal study. *Physiology & Behavior*, *139*, 177–181. https://doi.org/10.1016/J.PHYSBEH.2014.11.032

Zettermark, S. (2024). 'They all of a sudden became new people': Using reproductive justice to explore narratives of hormonal contraceptive experience in Sweden. *European Journal of Women's Studies, 31*, 52-71. https://doi.org/10.1177/13505068241230821

Zmigrod, L., Ebert, T., Götz, F. M., & Rentfrow, P. J. (2021). The psychological and socio-political consequences of infectious diseases: Authoritarianism, governance, and nonzoonotic (human-to-human) infection transmission. *Journal of Social and Political Psychology, 9*(2), 456-474. https://doi.org/10.5964/jspp.7297

Zwahlen, M., & Stute, P. (2023). Impact of progesterone on the immune system in women: A systematic literature review. *Archives of Gynecology and Obstetrics*, *1*, 1-10. https://doi.org/10.1007/S00404-023-06996-9

APPENDIX A

STUDY 1 IRB LETTERS

OAKLAND UNIVERSITY

Institutional Review Board

March 17, 2021

Protocol #: IRB-FY2021-302

Research Team:
Jenna Lunge
Lisa Welling

Based on applicable federal regulations, the following study, "Oral contraceptives and emotions" has been determined to be Exempt, with the following categories Category 2.(ii). Research that only includes interactions involving educational tests (cognitive, diagnostic, aptitude, achievement), survey procedures, interview procedures, or observation of public behavior (including visual or auditory recording).
Any disclosure of the human subjects' responses outside the research would not reasonably place the subjects at risk of criminal or civil liability or be damaging to the subjects' financial standing, employability, educational advancement, or reputation.

Letter and Consent Document:
This letter along with the IRB approved (date-stamped) consent document can be found in Cayuse in the Submission Details page under Letters and Attachments, respectively.
The IRB date stamped consent document must be downloaded and used in consenting participants.

Modifications:
Any changes to this exempt project must be reviewed by the IRB prior to initiation by submitting a MODIFICATION request. Do not collect data while the changes are being reviewed. Data collected during this time cannot be used in research.

Record Retention:
Exempt projects will be retained by the IRB office for three years after the last action on the project.

You are approved to start the research. Please retain a copy of this notification for your records.

If you have any questions, please contact the IRB office.

Thank you.

The Oakland University IRB

OAKLAND UNIVERSITY

Institutional Review Board

Date: December 1, 2021

Study #: IRB-FY2021-302

Study Title: Oral contraceptives and emotions

Submission Type: Modification

IRB Decision: Exempt

Research Team:
Jenna Lunge
Lisa Welling

The IRB has reviewed the Modification submission related to the above referenced study and determined that the changes listed below do not affect the exemption status of the study. The study remains Exempt per federal regulations.

The approved modifications are the following:

- Change in time required for survey: Edited from 40 minutes to 45 minutes
- Change in incentive: Edited from 1 SONA credit to 0.75 SONA credits based on new psych dept. policy.
- Change to recruitment document: edited to reflect changes listed above.
- Change in consent document: edited to reflect changes listed above

The REVISED IRB approved (date-stamped) consent document Version 12/01/2021 has been published under Attachments in the Submission Details page. Please download the IRB date stamped consent and use it in consenting participants.

You are approved to implement the aforementioned modifications. Please retain a copy of this notification for your records.

This letter can also be found in Cayuse under the Letters tab in the Submission Details page.

If you have any questions, please contact the IRB office.

Thank you.

The Oakland University IRB

OAKLAND UNIVERSITY

Institutional Review Board

Date: June 28, 2023

Study #: IRB-FY2021-302

Study Title: Oral contraceptives and emotions

Submission Type: Modification

IRB Decision: Exempt

Research Team:
Jenna Lunge
Lisa Welling

The IRB has reviewed the Modification submission related to the above referenced study and determined that the changes listed below do not affect the exemption status of the study. The Modification submission is Approved and the study remains Exempt per federal regulations.

The approved modifications are the following:

1. Change in inclusion criteria: Users of oral contraceptive pills in general are now included.
2. Change in recruitment document: To reflect the change in inclusion criteria, pre-screener questions and recruitment materials have been updated.
3. Change in consent document: To reflect the change in inclusion criteria, the Information Sheet has been updated in the 'Who can participate' section.
4. Procedures and data collection: To reflect the change in inclusion criteria, the Hormonal Contraceptive Questionnaire has been updated to include all currently available hormonal contraceptive pills and question text has been edited to improve clarity.

The REVISED IRB approved (date-stamped) consent document Version 06282023 has been published under Attachments in the Submission Details page. Please make sure to use the most recent IRB approved version of the consent form in consenting participants.

You are approved to implement the aforementioned modifications. Please retain a copy of this notification for your records.

This letter can also be found in Cayuse under the Letters tab in the Submission Details page.

If you have any questions, please contact the IRB office.

Thank you.

The Oakland University IRB

APPENDIX B

STUDIES 2 AND 3 IRB LETTERS

OAKLAND UNIVERSITY

Institutional Review Board

April 28, 2021

Protocol #: IRB-FY2021-311

Research Team:
Jenna Lunge
Lisa Welling

Based on applicable federal regulations, the following study, "Hormone profile and women's attitudes" has been determined to involve no more than minimal risk to participants and Approved, with the following expedited categories 3. Prospective collection of biological specimens for research purposes by noninvasive means. 7. Research on individual or group characteristics or behavior (including, but not limited to, research on perception, cognition, motivation, identity, language, communication, cultural beliefs or practices, and social behavior) or research employing survey, interview, oral history, focus group, program evaluation, human factors evaluation, or quality assurance methodologies.

This decision expires on April 27, 2022.

This approval is based on an appropriate risk/benefit ratio and a project design wherein the risks have been minimized. All research must be conducted in accordance with this approved submission.

This letter along with the IRB date-stamped consent document can be found in Cayuse in the Submission Details page under Letters and Attachments, respectively.

Notes for Researchers:

Thank you for such a thorough and well written application. Good luck with your study.

Permission from Research Sites:
Please note the following:

- This IRB approval letter means that the research has met the federal criteria for approval per 45 CFR 46.111 Criteria for IRB Approval of Research.
- Before the research is initiated, permission to conduct research at a given site must be obtained from all research locations listed in the IRB submission. You must keep copies of all such permission letters for your records.
- It is the responsibility of each researcher to follow all applicable policies and procedures of any outside institution where the research will be conducted.

Letter and Consent Document:
This letter along with the IRB approved (date-stamped) consent document can be found in Cayuse in the Submission Details page under Letters and Attachments, respectively.

The IRB approved version of the consent document must be used in consenting participants. Federal regulations require that each participant receive a copy of the consent document unless a waiver is granted by the IRB. Signed consent form(s) must be retained for a minimum of three years after the completion of the project. Please remember that informed consent is a process that continues throughout the project, starting with recruitment and assurance of participant understanding of the project, and followed by a signed consent form when applicable.

Modifications:
Any changes to the approved project must be approved by the IRB prior to initiation by submitting a MODIFICATION request. Do not collect data while the changes are being reviewed. Data collected during this time cannot be used in research.

Incidents:
All unanticipated problems involving risks to participants or others, serious and unexpected adverse events, non-compliance issues and/or serious complaints regarding this project must be reported promptly to the IRB by submitting an INCIDENT report.

Renewal:
Based on the risks to participants, this project requires continuing review by the IRB on an annual basis. Prior to the expiration date of April 27, 2022, submit a RENEWAL request in order to continue with the research. The renewal submission will need to be approved before you can continue with the study.

You are approved to start the research. Please retain a copy of this notification for your records.

If you have any questions, please contact the IRB office.

Thank you.

The Oakland University IRB

OAKLAND UNIVERSITY

Institutional Biosafety Committee

November 9, 2020

Associate Professor Lisa Welling
Department of Psychology

Reference: IBC Application #2739 "Testosterone, Fragile Self-Esteem, and Mate Preferences"

Dear Dr. Welling:

The Institutional Biosafety Committee (IBC), responsible for the review of research involving materials of human origin, recombinant DNA, and biohazardous, infectious or toxic materials, has reviewed and approved the continuing review of the project referenced above.

The proposed activities will continue to be approved at BSL-2 for another 3 years starting November 9, 2020 and ending November 8, 2023. The approval number 2739-12 remains the same.

Any unanticipated problems, changes in, or halting of the activities proposed in the project, as described, must be promptly reported to the IBC or the biosafety officer.

In addition, all faculty, staff and students involved in the project must be trained and qualified to conduct the project in a responsible manner. Please note, in this regard, that you, the principal investigator, are fully responsible at all times for limiting or restricting access to laboratories and for assessing, in each circumstances, who may enter or work in a laboratory when biohazardous materials are in use.

Please let me know if you have any questions. Thank you.

Sincerely,

Domenic Luongo, CHMM
Director - Laboratory Safety and Compliance

The Research Office
371 Wilson Blvd, Rochester, MI 48309-4486
(248) 370-4898 | Fax: (248) 370-2973 | oakland.edu

OAKLAND UNIVERSITY

Institutional Review Board

Date: September 24, 2021

Study #: IRB-FY2021-311

Study Title: Hormone profile and women's attitudes

Submission Type: Modification

IRB Decision: Approved

Research Team:
Jenna Lunge
Lisa Welling

The IRB has reviewed the Modification submission for the above referenced study. The modified study with the changes listed below has been determined to involve no more than minimal risk to participants and is Approved through an expedited review per federal regulations.

The approved modifications are the following:

Modification for the following:

- Change in Procedures: The research team will email participants a link to the study survey after participants pick up their study kit.
- Change in confidentiality: Participants will be emailed a link to the Qualtrics survey from the PIs password protected Oakland.edu email address. The email listed on participants' SONA account will be used to send the survey link. At the end of each week, the PI will clear sent emails associated with the study from the email account to minimize the risk of confidentiality breaches. Email addresses will not be stored.

You are approved to implement the aforementioned modifications. Please retain a copy of this notification for your records.

This letter can also be found in Cayuse under the Letters tab in the Submission Details page.

If you have any questions, please contact the IRB office.

Thank you.

The Oakland University IRB

OAKLAND UNIVERSITY

Institutional Review Board

April 7, 2022

Protocol #: IRB-FY2021-311

Research Team:
Jenna Lunge
Lisa Welling

The renewal of the following study, "Hormone profile and women's attitudes", has been deemed to be Approved based on applicable regulations. This decision regarding the renewal of this study expires on April 6, 2023.

Notes for Researcher(s):

- This project is also under Biosafety approval, IBC Application #2739 (PI: Lisa Welling), which is valid until November 8, 2023

Please remember the following:

Consent Document(s):
IRB date-stamped consent document(s) must be used in consenting participants. Federal regulations require that each participant receives a copy of the consent document unless a waiver is granted by the IRB. Signed consent form(s) must be retained by the researcher for a minimum of three years after the completion of the project.

Modifications:
Any changes to the approved project must be approved by the IRB prior to initiation by submitting a MODIFICATION request. Do not collect data while the changes are being reviewed. Data collected during this time cannot be used in research.

Incidents:
All unanticipated problems involving risks to participants or others, serious and unexpected adverse events, non-compliance issues and/or serious complaints regarding this project must be reported promptly to the IRB by submitting an INCIDENT report.

Renewal:
Based on the risks to participants, this project requires continuing review by the IRB on an annual basis. Prior to the expiration date of April 6, 2023, submit a RENEWAL request in order to continue with the research. The renewal submission will need to be approved before you can continue with the study.

You are approved to continue the research. Please retain a copy of this notification for your records.

If you have any questions, please contact the IRB office.

Thank you.

The Oakland University IRB

OAKLAND UNIVERSITY

Institutional Review Board

Date: April 15, 2022

Study #: IRB-FY2021-311

Study Title: Hormone profile and women's attitudes

Submission Type: Modification

IRB Decision: Approved

Research Team:
Jenna Lunge
Lisa Welling

The IRB has reviewed the Modification submission for the above referenced study. The modified study with the changes listed below has been determined to involve no more than minimal risk to participants and is Approved through an expedited review per federal regulations.

FINDINGS:
Note that researchers are advised to have endorsement by the Director of Graduate Studies, Dr. Zeigler-Hill, that recruitment in classrooms is permissible for this particular study given that there is an understanding in the Psychology Department that researchers are NOT to recruit directly to mitigate potential favoritism for a study. This is not a requirement for IRB approval.

The approved modifications are the following:

- **Change in recruitment process**: Addition of virtual flyers in courses requiring SONA participation, PI also attached email message to instructors with request to post virtual flyer. Paper flyers will also be posted around campus.
- **Change in recruitment documents**: Addition of virtual flyers, paper flyers posted on OU boards and email to instructors to request the distribution of virtual flyers. Modification to existing SONA flyer to indicate room change.
- **Change in consent process:** Non-SONA participants will be asked to access a copy of the consent form through the description in their Google Appointment. This information will also be automatically emailed to them by google. All participants will still sign the consent in person.
- **Change in consent document**: changes to room number and incentive information
- **Change of location:** Room changed from Pryale Room 102 to Pryale Room 103
- **Change in incentive:** Non-SONA orally contracepted participants will receive $20 for completing 2 sessions of this study ($10 for Session 1 + $10 for Session 2) at the time of drop-off of Session 2 materials. Successful completion of the study is defined as completing 85% or more of each online survey and returning the completed saliva sample kit to the researchers. SONA credits issued as incentive is unchanged.
- **Change in confidentiality**: Participants will use Google Appointments to enroll and schedule their in person appointments

The REVISED IRB approved (date-stamped) consent document has been published under Attachments in the Submission Details page. Please download the IRB date stamped consent and use it in consenting participants.

You are approved to implement the aforementioned modifications. Please retain a copy of this notification for your records.

This letter can also be found in Cayuse under the Letters tab in the Submission Details page.

If you have any questions, please contact the IRB office.

Thank you.

The Oakland University IRB

OAKLAND UNIVERSITY

Institutional Review Board

Date: May 12, 2022

Study #: IRB-FY2021-311

Study Title: Hormone profile and women's attitudes

Submission Type: Modification

IRB Decision: Approved

Research Team:
Jenna Lunge
Lisa Welling

The IRB has reviewed the Modification submission for the above referenced study. The modified study with the changes listed below has been determined to involve no more than minimal risk to participants and is Approved through an expedited review per federal regulations.

The approved modifications are the following:

Addition of Recruitment Process and Document: Addition of an email to general psychology instructors in all undergraduate psychology courses to request distribution of virtual flyer.

You are approved to implement the aforementioned modifications. Please retain a copy of this notification for your records.

This letter can also be found in Cayuse under the Letters tab in the Submission Details page.

If you have any questions, please contact the IRB office.

Thank you.

The Oakland University IRB

OAKLAND UNIVERSITY

Institutional Review Board

March 15, 2023

Protocol #: IRB-FY2021-311

Research Team:
Jenna Lunge
Lisa Welling

The renewal of the following study, "Hormone profile and women's attitudes", has been deemed to be Approved based on applicable regulations. This decision regarding the renewal of this study expires on March 14, 2024.

Notes for Researcher(s):

- IRB approval for another year is granted with the understanding that IBC approval for IBC Application #2739 (PI: Lisa Welling) will be renewed prior to its expiration on November 8, 2023 to maintain this study's IBC biosafety approval.

Please remember the following:

Consent Document(s):
IRB date-stamped consent document(s) must be used in consenting participants. Federal regulations require that each participant receives a copy of the consent document unless a waiver is granted by the IRB. Signed consent form(s) must be retained by the researcher for a minimum of three years after the completion of the project.

Modifications:
Any changes to the approved project must be approved by the IRB prior to initiation by submitting a MODIFICATION request. Do not collect data while the changes are being reviewed. Data collected during this time cannot be used in research.

Incidents:
All unanticipated problems involving risks to participants or others, serious and unexpected adverse events, non-compliance issues and/or serious complaints regarding this project must be reported promptly to the IRB by submitting an INCIDENT report.

Renewal:
Based on the risks to participants, this project requires continuing review by the IRB on an annual basis. Prior to the expiration date of March 14, 2024, submit a RENEWAL request in order to continue with the research. The renewal submission will need to be approved before you can continue with the study.

You are approved to continue the research. Please retain a copy of this notification for your records.

If you have any questions, please contact the IRB office.

Thank you.

The Oakland University IRB

OAKLAND UNIVERSITY

Institutional Review Board

Date: February 20, 2024

Study #: IRB-FY2021-311

Study Title: Hormone profile and women's attitudes

Submission Type: Closure

IRB Decision: Closed

Research Team:
Jenna Lunge
Lisa Welling

The above referenced study has been closed as of this date.

Please remember that, if applicable to the study, signed consent forms must be kept for three years after the completion of the study in case of an audit.

Please save a copy of this letter for your records.

Thank you.

The Oakland University IRB

APPENDIX C

STUDY 1 MATERIALS

Prescreen Questions

1. What most accurately describes your gender?
    a. Male
    b. Female
    c. Transgender
2. Are you currently using an oral hormonal contraceptive (i.e., "the pill")?
    a. Yes
    b. No
3. When you *most recently* took a dose of your oral contraceptive pill (today or yesterday), did you take an active pill or did you take a "reminder" pill (i.e., sugar or iron pill that coincides with menstruation)? For example, *Junel-Fe* contains 21 active pills containing norethindrone and ethinyl estradiol followed by 7 differently colored reminder pills with iron, whereas *Junel-21* contains 21 active pills containing norethindrone and ethinyl estradiol but does not contain any reminder pills.
    a. Active pill
    b. Reminder pill / No pill
    c. I'm not sure
4. [If Reminder Pill] Have you taken *at least* 2 days of reminder pill doses at this point today?
    a. Yes
    b. No

Demographics Information.

1. What is your age?
2. What most accurately describes your biological sex?
    a. Male
    b. Female
    c. Transgender
3. What most accurately describes your sexual orientation?
    a. Heterosexual
    b. Homosexual
    c. Bisexual
    d. Other
4. What most accurately describes your current relationship status?
    a. Single
    b. In an exclusive, romantic relationship with someone of the opposite sex
    c. In an exclusive, romantic relationship with someone of the same sex
    d. In a romantic relationship with more than one person

5. Are you of Hispanic, Latin, or Spanish origin? (Check ALL the boxes that apply)
    a. No, not of Hispanic, Latin, or Spanish origin
    b. Yes, Mexican, Mexican American, Chicano
    c. Yes, Puerto Rican
    d. Yes, Cuban
    e. Yes, another Hispanic, Latin, or Spanish origin
6. What is your Race/Ethnicity? (Check ALL the boxes that apply)
    a. White
    b. Black, African American
    c. American Indian or Alaska Native
    d. Asian Indian
    e. Chinese
    f. Filipino
    g. Japanese
    h. Korean
    i. Vietnamese
    j. Other Asian
    k. Native Hawaiian
    l. Guamanian or Chamorro
    m. Samoan
    n. Other Pacific Islander
    o. Other Race
    p. Prefer not to answer
7. In the last 7 days, have you experienced an illness such as a cold or the flu?
    a. Yes
    b. No
8. Have you ever menstruated?
    a. Yes
    b. No
9. Are you using hormone replacement therapy (HRT)? This does not include hormonal birth control (e.g., “the pill”).
    a. Yes
    b. No
10. Are you currently pregnant?
    a. Yes
    b. No
    c. Unsure
11. Have you given birth to a child in the last year?
    a. Yesm

b. No

12. [If yes] When was your child born? (MM/DD/YYYY)
13. Are you currently breast-feeding?
    a. Yes
    b. No

Hormonal Contraceptive Questionnaire (for participants who are using oral hormonal contraceptives)

Some of these questions may be similar to the questions you completed before signing the consent form. It is important for the questions below to be answered **honestly**. Your honest responses to the questions here **do not** impact your eligibility for SONA credit as long as you respond completely.

1. What TYPE of hormonal contraceptive are you currently using? Please select from the following (check all that apply):
    - Oral Contraceptive Pill
    - Vaginal Ring (e.g., NuvaRing)
    - Hormonal IUD
    - Skin Patch
    - Shot/Injection
    - Implant
2. Please select the brand of oral contraceptive you use. Match your pill pack *exactly* to the correct option below. Many brands may look similar, for example, Nortrel 1/35 21 is different from Nortrel 1/35 28. Additional details about tablets have been provided to differentiate between similarly named brands.
    - Alyacen 1/35 (21 peach + 7 light green tablets)
    - Alyacen 7/7/7 28 (7 white + 7 light peach + 7 peach + 7 light green tablets)
    - Apri
    - Aranelle
    - Aviane
    - Balziva
    - Blisovi 24 FE (24 white + 4 brown tablets)
    - Blisovi FE 1.5/30 (21 pink + 7 brown tablets)
    - Blisovi FE 1/20 (21 yellow + 7 brown tablets)
    - Camila
    - Elinest
    - Enpresse
    - Enskyce
    - Errin

- Estarylla
- Estrostep FE
- Generess FE
- Hailey 1.5/30 (21 green tablets only)
- Hailey 24 FE (24 white + 4 brown tablets)
- Hailey FE 1.5/30 (21 green + 7 brown tablets)
- Hailey FE 1/20 (21 white + 7 brown tablets)
- Heather
- Isibloom
- Junel 1.5/30 21 (21 pink tablets only)
- Junel 1/20 (21 yellow tablets only)
- Junel FE 1.5/30 (21 pink + 7 brown tablets)
- Junel FE 1/20 (21 yellow + 7 brown tablets)
- Junel FE 24 (24 yellow + 4 brown tablets)
- Kariva
- Larin 1.5/30 (21 yellow tablets only)
- Larin 1/20 (21 pale yellow tablets only)
- Larin 24 FE (24 pale yellow + 4 brown tablets)
- Larin FE 1.5/30 (21 yellow + 7 brown tablets)
- Larin FE 1/20 (21 pale yellow + 7 brown tablets)
- Larissia
- Leena
- Lessina
- Levora
- Lo Loestrin FE (24 blue + 2 white + 2 brown tablets)
- Loestrin 21 1.5/30 (21 pink tablets only)
- Loestrin 21 1/20 (21 yellow tablets only)
- Loestrin FE 1.5/30 (21 pink + 7 brown tablets)
- Loestrin FE 1/20 (21 yellow + 7 brown tablets)
- Lutera
- Microgestin 1.5/30 (21 yellow tablets only)
- Microgestin 1/20 (21 pale yellow tablets only)
- Microgestin 24 FE (24 pale yellow + 4 brown tablets)
- Microgestin FE 1.5/30 (21 yellow tablets + 7 brown tablets)
- Microgestin FE 1/20 (21 pale yellow + 7 brown tablets)
- Minastrin 24 FE
- Natazia
- Necon 0.5/35 (21 light peach + 7 white tablets)
- Necon 7/7/7 (7 white + 7 light peach + 7 peach + 7 green tablets)

- Nortrel 0.5/35 (21 light yellow + 7 white tablets)
- Nortrel 1/35 (21 yellow + 7 white tablets)
- Nortrel 7/7/7 (7 light yellow + 7 blue + 7 peach + 7 white tablets)
- Pimtrea
- Previfem
- Seasonique
- Setlakin
- Sprintec
- Sronyx
- Tarina FE 1/20 (21 yellow + 7 brown tablets)
- Taytulla
- Tri-Femynor
- Tri-Legest FE
- Tri-Lo-Mili
- Tri-Sprintec
- Trivora
- Tri-Vylibra
- Tri-Vylibra-Lo
- Yasmin
- Yaz
- Other/Not Listed

3. [IF participant selects Other: Please provide "Please specify which brand of oral contraceptive you use. It may be helpful to look at your pill pack for the brand information"
    - Afirmelle
    - Altavera
    - Amethia (84 white + 7 blue tablets)
    - Amethia Lo (84 orange + 7 yellow tablets)
    - Amethyst
    - Ashlyna
    - Aubra (21 yellow + 7 brown tablets)
    - Aubra EQ (21 white + 7 green tablets)
    - Aurovela 1.5/30 (21 white tablets only)
    - Aurovela 1/20 (21 yellow tablets only)
    - Aurovela 24 FE (24 yellow + 4 brown tablets)
    - Aurovela FE 1.5/30 (21 white + 7 brown tablets)
    - Aurovela FE 1/20 (21 yellow + 7 brown tablets)
    - Ayuna
    - Azurette

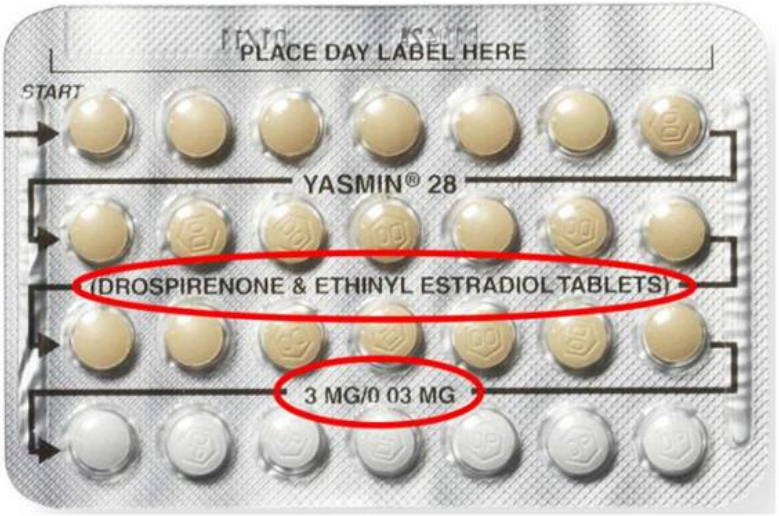
- Balcoltra
- Bekyree
- Beyaz
- Briellyn
- Camrese (84 blue-green + 7 yellow tablets)
- Camrese Lo (84 orange + 7 yellow tablets)
- Caziant
- Chateal
- Cryselle
- Cyclafem 1/35 (21 pink + 7 light green tablets)
- Cyclafem 7/7/7 28 (7 white + 7 light pink + 7 pink + 7 light green tablets)
- Cyonanz
- Dasetta 1/35 (21 orange + 7 white tablets)
- Dasetta 7/7/7 28 (7 light peach + 7 peach + 7 orange + 7 white tablets)
- Daysee
- Deblitane
- Delyla
- Dolishale
- Emoquette
- Falmina
- Fayosim
- Femynor
- Finzala
- Gemmily
- Iclevia
- Incassia
- Introvale
- Jaimiess
- Jencycla
- Jolessa
- Kaitlib FE
- Kalliga
- Kelnor
- Kurvelo
- Layolis FE
- Levonest
- Lojaimiess

- Loryna
- LoSeasonique
- Low-Ogestrel
- Lo-Zumandimine
- Marlissa
- Merzee
- Mibelas 24 FE
- Mili
- Mono-Linyah
- Nextstellis
- Nikki
- Nora-be
- Norlyda
- Nylia 1/35 (21 peach + 7 green tablets)
- Nylia 7/7/7 (7 white + 7 light peach + 7 peach + 7 green tablets)
- Ocella
- Orsythia
- Philith
- Pirmella 1/35 (21 peach + 7 green tablets)
- Pirmella 7/7/7 (7 white + 7 light peach + 7 peach + 7 green tablets)
- Portia
- Quartette
- Reclipsen
- Safyral
- Sharobel
- Simpesse
- Slynd
- Syeda
- Tarina FE 24 (24 yellow + 4 brown tablets)
- Tilia FE
- Tri-Estarylla
- Tri-Linyah
- Tri-Lo Estarylla
- Tri-Lo Marzia
- Tri-Lo Sprintec
- Tri-Mili
- Tri-Nymyo
- Tri-Previfem
- Twirla

- Tyblume
- Tydemy
- Velivet
- Vestura
- Vienva
- Viorele
- Volnea
- Vyfemla
- Vylibra
- Wera
- Wymzya FE
- Xulane
- Zovia
- Zumandimine
- Other/Not Listed

4. [If participant selects OTHER]

Please look at your pill pack, there will be information about the dose of medication on the front. For example, Yasmin 28 contains 3 mg of Drospirenone and 0.03 mg of Ethinyl Estradiol



Use the drop-down and write-in portions below to enter the medications and strengths listed on your pill pack (e.g., .15 mg Levonorgestrel/30 mcg ethinyl estradiol). IMPORTANT: Do not write the manufacturer (e.g., Teva, Bayer, Mylan). Please provide enough information for the research team to locate the oral contraceptive that you use.

	Progestin type	Progestin strength in mg or mcg (if there are multiple, list them in order)
Progestin		

	Estradiol Type	Estradiol strength in mg or mcg (if there are multiple, list them in order)
Estradiol		

5. For how long have you been using your **current** brand of hormonal contraceptive?
   ____ YEARS, _____ MONTHS
6. In your current pill pack, which week are you on? For example, if you are taking a pack that lasts for four weeks, there are four rows of pills in your pack. If you are taking pills from the third row, you are in the third week of your pack.
7. When you *most recently* took a dose of your oral contraceptive pill (today or yesterday), did you take an active pill or did you take a "reminder" pill (i.e., sugar, placebo, or iron pill)? For example, *Junel-Fe* contains 21 active pills containing norethindrone and ethinyl estradiol followed by 7 differently colored reminder pills with iron, whereas *Junel-21* contains 21 active pills containing norethindrone and ethinyl estradiol but does not contain any reminder pills.
   - Active pill
   - Reminder pill / No pill
   - I'm not sure
8. If you are reading this, answer "4" for this question.
   - 1
   - 2
   - 3
   - 4
   - 5

9. Which day of your oral contraceptive pill are you currently on? For example, if you started a new pill pack yesterday then you are on Day 2 today. If you start a new pill pack tomorrow, then you might be on Day 28 today (this varies depending on the brand of oral contraceptive that *you* use!).
10. How many hours has it been since you took your most recent oral contraceptive pill?

Perceived Vulnerability to Disease scale (Duncan, Schaller, & Park, 2009)

[Instructions] 1 = Strongly Disagree … 7 = Strongly Agree

1. It really bothers me when people sneeze without covering their mouths.
2. If an illness is 'going around', I will get it.
3. I am comfortable sharing a water bottle with a friend. (R)
4. I don't like to write with a pencil someone else has obviously chewed on.
5. My past experiences make me believe I am not likely to get sick even when my friends are sick. (R)
6. I have a history of susceptibility to infectious diseases.
7. I prefer to wash my hands pretty soon after shaking someone's hand.
8. In general, I am very susceptible to colds, flu, and other infectious disease.
9. I dislike wearing used clothes because you don't know what the past person who wore it was like.
10. I am more likely than the people around me to catch an infectious disease.
11. My hands do not feel dirty after touching money. (R)
12. I am unlikely to catch a cold, flu, or other illness, even if it is going around. (R)
13. It does not make me anxious to be around sick people. (R)
14. My immune system protects me from most illness that other people get. (R)
15. I avoid using public telephones because of the risk that I may catch something from the previous user.

The Disgust Propensity and Sensitivity Scale–Revised (DPSS-R; van Overveld et al., 2010)

This questionnaire consists of 12 statements about disgust. Please read each statement and think how often it is true for you, then select the response that is closest to this.

Never
Rarely
Sometimes
Often
Always

1. I avoid disgusting things.

2. When I feel disgusted, I worry that I might pass out.
3. It scares me when I feel nauseous.
4. I feel repulsed.
5. Disgusting things make my stomach turn.
6. I screw up my face in disgust.
7. When I notice that I feel nauseous, I worry about vomiting.
8. I experience disgust.
9. It scares me when I feel faint.
10. I find something disgusting.
11. It embarrasses me when I feel disgusted.
12. I think feeling disgust is bad for me.

The Disgust Scale–Revised (DR-R; Haidt et al., 1994; Modified by Olatunji et al., 2007)

Please indicate how much you agree with each of the following statements, or how true it is about you. Please select a number (0-4) to indicate your answer:

0 = Strongly disagree (very untrue about me)
1 = Mildly disagree (somewhat untrue about me)
2 = Neither agree nor disagree
3 = Mildly agree (somewhat true about me)
4 = Strongly agree (very true about me)

1. I might be willing to try eating monkey meat, under some circumstances.
2. It would bother me to be in a science class, and to see a human hand preserved in a jar.
3. It bothers me to hear someone clear a throat full of mucus.
4. I never let any part of my body touch the toilet seat in public restrooms.
5. I would go out of my way to avoid walking through a graveyard.
6. Seeing a cockroach in someone else's house doesn't bother me.
7. It would bother me tremendously to touch a dead body.
8. If I see someone vomit, it makes me sick to my stomach.
9. I probably would not go to my favorite restaurant if I found out that the cook had a cold.
10. It would not upset me at all to watch a person with a glass eye take the eye out of the socket.
11. It would bother me to see a rat run across my path in a park.
12. I would rather eat a piece of fruit than a piece of paper.
13. Even if I was hungry, I would not drink a bowl of my favorite soup if it had been stirred by a used but thoroughly washed flyswatter.
14. It would bother me to sleep in a nice hotel room if I knew that a man had died of a

heart attack in that room the night before.

How disgusting would you find each of the following experiences? Please select a number (0-4) to indicate your answer:

0 = Not disgusting at all
1 = Slightly disgusting
2 = Moderately disgusting
3 = Very disgusting
4 = Extremely disgusting

15. You see maggots on a piece of meat in an outdoor garbage pail.
16. You see a person eating an apple with a knife and fork
17. While you are walking through a tunnel under a railroad track, you smell urine.
18. You take a sip of soda, and then realize that you drank from the glass that an acquaintance of yours had been drinking from.
19. Your friend's pet cat dies, and you have to pick up the dead body with your bare hands.
20. You see someone put ketchup on vanilla ice cream, and eat it.
21. You see a man with his intestines exposed after an accident.
22. You discover that a friend of yours changes underwear only once a week.
23. A friend offers you a piece of chocolate shaped like dog-doo.
24. You accidentally touch the ashes of a person who has been cremated.
25. You are about to drink a glass of milk when you smell that it is spoiled.
26. As part of a sex education class, you are required to inflate a new unlubricated condom, using your mouth.
27. You are walking barefoot on concrete, and you step on an earthworm.

Attention Check

Sometimes people move through surveys quickly without reading the questions and answering honestly. If you are paying attention and responding honestly, please respond "Strongly Agree".

- Strongly Disagree
- Disagree
- Agree
- Strongly Agree

Culpepper Disgust Image Set (C-DIS; Culpepper et al., 2018)

The stimuli that will be used to rate feelings of disgust. Participants will view *either* the pathogen-salient stimuli or (2) pathogen-free stimuli. The 20 images will be presented to

participants one-at-a-time and participants will be asked to rate each image for disgust on a 7-point scale (0 = *not disgusting at all*, 6 = *extremely disgusting*).

State Disgust

How disgusted do you feel right now? (rated on a sliding scale)

0 ----------100

Situational Pathogen Avoidance scale (SPA; Makhanova et al., 2021)

For this task, you will answer a questionnaire about your attitudes. For the following questions, try to imagine yourself being placed in these situations right now, that is at this moment in time. Please indicate how much you agree or disagree with each of the following statements on a scale of 1 (Strongly Disagree) to 7 (Strongly Agree).

1. Right now, if I was standing next to a person who sneezed I would feel disgusted.
2. Right now, I would be grossed out if I shook a stranger's hand.
3. Right now, if someone coughed next to me without covering their mouth, I would move away from them.
4. Right now, it would make me uncomfortable to touch a door handle in a public restroom.
5. Right now, if I heard that a friend had the flu, I would avoid going to their house or apartment.
6. Right now, I would be bothered by sitting in a seat (that is still warm) that a stranger just got up from.
7. Right now, if a person looked like they were sick, I would be willing to shake their hand.
8. Right now, I would be happy to sit close to someone who just finished an intense workout.
9. Right now, I would try to sit on the opposite side of the room if I walked into the room where there was a person blowing their nose.
10. Right now, I would want to wash my hands immediately, if I shook hands with a person with sweaty palms.

APPENDIX D

STUDY 2 MATERIALS

Participant ID Number

What is your participant ID number that is labelled on your study kit? This will be found on the clear plastic bags and on the card containing the link to this survey.

Demographics Information.

1. What is your age?
2. What most accurately describes your biological sex?
    a. Male
    b. Female
    c. Transgender
3. What most accurately describes your sexual orientation?
    a. Heterosexual
    b. Homosexual
    c. Bisexual
    d. Other
4. What most accurately describes your current relationship status?
    a. Single
    b. In an exclusive, romantic relationship with someone of the opposite sex
    c. In an exclusive, romantic relationship with someone of the same sex
    d. In a romantic relationship with more than one person
5. Are you of Hispanic, Latin, or Spanish origin? (Check ALL the boxes that apply)
    a. No, not of Hispanic, Latin, or Spanish origin
    b. Yes, Mexican, Mexican American, Chicano
    c. Yes, Puerto Rican
    d. Yes, Cuban
    e. Yes, another Hispanic, Latin, or Spanish origin
6. What is your Race/Ethnicity? (Check ALL the boxes that apply)
    a. White
    b. Black, African American
    c. American Indian or Alaska Native
    d. Asian Indian
    e. Chinese
    f. Filipino
    g. Japanese
    h. Korean
    i. Vietnamese
    j. Other Asian
    k. Native Hawaiian
    l. Guamanian or Chamorro

m. Samoan
n. Other Pacific Islander
o. Other Race
p. Prefer not to answer

7. In the last 7 days, have you experienced an illness such as a cold or the flu?
    a. Yes
    b. No
8. Have you ever menstruated?
    a. Yes
    b. No
9. Are you using hormone replacement therapy (HRT)? This does not include hormonal birth control (e.g., "the pill").
    a. Yes
    b. No
10. Are you currently pregnant?
    a. Yes
    b. No
    c. Unsure
11. Have you given birth to a child in the last year?
    a. Yes
    b. No
12. [If yes] When was your child born? (MM/DD/YYYY)
13. Are you currently breast-feeding?
    a. Yes
    b. No

Menstrual Cycle Questionnaire (for participants who are not using hormonal contraceptives):

1. What was the FIRST day of menstrual flow of your last period?
2. How certain are you of this estimate?
3. What was the FIRST day of menstrual flow for your period before last?
4. How certain are you of this estimate?
5. When do you expect your next menstrual period to start?
6. How certain are you of this estimate?
7. What is the usual length of your entire menstrual cycle? That is, **what is the typical number of DAYS between the <u>first day</u> of menstrual blood flow in one month to the day before the <u>first day</u> of menstrual blood flow in the next month?** The average menstrual cycle length is 28 days. Please think carefully about this and provide the most accurate answer you can:

8. How certain are you of this estimate?
9. If you are reading this, answer “4” for this question.
    a. 1
    b. 2
    c. 3
    d. 4
    e. 5

Hormonal Contraceptive Questionnaire (for participants who are using oral hormonal contraceptives):

1. What TYPE of hormonal contraceptive are you currently using? Please select from the following (check all that apply):
    a. Oral Contraceptive Pill
    b. Vaginal Ring (e.g., NuvaRing)
    c. Hormonal IUD
    d. Skin Patch
    e. Shot/Injection
    f. Implant
2. Please specify which brand of oral contraceptive you use. It may be helpful to look at your pill pack for the brand information.
    - Alesse®
    - Apri®
    - Aviane®
    - Camila®
    - Desogen®
    - Errin®
    - Estrostep FE®
    - Heather®
    - Jolivette®
    - Junel FE®
    - Junel-21®
    - Kariva®
    - Levora®
    - Lo/Ovral®
    - Loestrin FE 1/20®
    - Lutera™
    - Microgestin 1.5/30®
    - Micronor®
    - Mononessa®

- Necon 1/35®
- Nor-Q.D.®
- Ortho Tri-Cyclen Lo®
- Ortho Tri-Cyclen®
- Ortho-Cept®
- Ortho-Cyclen®
- Ortho-Novum 1/35®
- Ortho-Novum 7/7/7®
- Ovrette®
- Seasonale®
- Seasonique®
- Sprintec®
- Trinessa®
- Tri-Sprintec®
- Trivora®
- Yasmin®
- Yaz®

3. [If participant selects OTHER] Please provide the brand name of your oral contraceptive. If you are taking a generic version, please refer to your pill pack. There should be information about the dose of medication on the front of your pill pack. Please enter that information below (e.g., .15mg Levonorgestrel/30 mcg ethinyl estradiol, .4mg Norethindrone/35 mcg ethinyl estradiol, etc.). Please do not write the Manufacturer of your pill (e.g., Teva, Bayer, Mylan, etc.)
4. Is your oral contraceptive progestin-only (i.e., contains **only** norethindrone/norethisterone) or is it a combined oral contraceptive (i.e., contains norethindrone/norethisterone **and** ethinyl estradiol)?
    a. Progestin-only
    b. Combined oral contraceptive
    c. I'm not sure
5. For how long have you been using your **current** brand of hormonal contraceptive?

    ____ YEARS, _____ MONTHS
6. In your current pill pack, which week are you on? For example, if you are taking a pack that lasts for four weeks, there are four rows of pills in your pack. If you are taking pills from the third row, you are in the third week of your pack.
7. When you *most recently* took a dose of your oral contraceptive pill (today or yesterday), did you take an active pill or did you take a "reminder" pill (i.e., sugar, placebo, or iron pill)? For example, *Junel-Fe* contains 21 active pills containing norethindrone and ethinyl estradiol followed by 7 differently colored

reminder pills with iron, whereas *Junel-21* contains 21 active pills containing norethindrone and ethinyl estradiol but does not contain any reminder pills.
    a. Active pill
    b. Reminder pill / No pill
    c. I'm not sure
8. If you are reading this, answer "4" for this question.
    a. 1
    b. 2
    c. 3
    d. 4
    e. 5
9. Which day of your oral contraceptive pill are you currently on? In other words, if you are currently taking your active pills, how many days of active pills have you taken? If you are currently taking your reminder pills, how many days of reminder pills have you taken?
10. At what time of day did you take the most recent dose of your oral contraceptive pill (e.g., 8:00 am)?

Perceived Vulnerability to Disease scale (Duncan, Schaller, & Park, 2009)

[Instructions] 1 = Strongly Disagree … 7 = Strongly Agree

16. It really bothers me when people sneeze without covering their mouths.
17. If an illness is 'going around', I will get it.
18. I am comfortable sharing a water bottle with a friend. (R)
19. I don't like to write with a pencil someone else has obviously chewed on.
20. My past experiences make me believe I am not likely to get sick even when my friends are sick. (R)
21. I have a history of susceptibility to infectious diseases.
22. I prefer to wash my hands pretty soon after shaking someone's hand.
23. In general, I am very susceptible to colds, flu, and other infectious disease.
24. I dislike wearing used clothes because you don't know what the past person who wore it was like.
25. I am more likely than the people around me to catch an infectious disease.
26. My hands do not feel dirty after touching money. (R)
27. I am unlikely to catch a cold, flu, or other illness, even if it is going around. (R)
28. It does not make me anxious to be around sick people. (R)
29. My immune system protects me from most illness that other people get. (R)
30. I avoid using public telephones because of the risk that I may catch something from the previous user.

TIME #1 Saliva Sample

It is now time to provide your TIME #1 saliva sample. Use the vial labeled "TIME 1" for this first sample. To reduce bubbling, place your straw into the provided TIME 1 vial and passively drool (you may want to tilt your head forward and to one side) through the straw into the vial without spitting your sample. If you are having trouble producing saliva, it is often helpful to rub your lower jaw or think about biting into a sour lemon or your favorite food. We need at least 2 mL of fluid saliva, not including bubbles, to properly analyze your sample. 2 mL is indicated on your vial, but we have emphasized this with a sharpie mark. When your fluid saliva reaches at least the 2 mL line, please remove your straw and tightly cap your TIME 1 vial.

IMPORTANT REQUIREMENTS: Use the provided disinfectant wipes to wipe and disinfect the sides of your vial after it is capped and place your disinfected vial into the provided plastic specimen bag labelled TIME 1. Move your bagged sample to your freezer until it can be returned to the research team. It is important to keep your sample frozen until it can be returned to Pryale Hall. To return the saliva sample, please transport the sample in a cooler with ice or gel packs and bring it directly to the researcher in Pryale Hall Room 102 at the agreed upon time.

The Disgust Scale–Revised (DR-R; Haidt et al., 1994; Modified by Olatunji et al., 2007)

Please indicate how much you agree with each of the following statements, or how true it is about you. Please select a number (0-4) to indicate your answer:

0 = Strongly disagree (very untrue about me)
1 = Mildly disagree (somewhat untrue about me)
2 = Neither agree nor disagree
3 = Mildly agree (somewhat true about me)
4 = Strongly agree (very true about me)

1. I might be willing to try eating monkey meat, under some circumstances.
2. It would bother me to be in a science class, and to see a human hand preserved in a jar.
3. It bothers me to hear someone clear a throat full of mucus.
4. I never let any part of my body touch the toilet seat in public restrooms.
5. I would go out of my way to avoid walking through a graveyard.
6. Seeing a cockroach in someone else's house doesn't bother me.
7. It would bother me tremendously to touch a dead body.
8. If I see someone vomit, it makes me sick to my stomach.
9. I probably would not go to my favorite restaurant if I found out that the cook had a cold.

10. It would not upset me at all to watch a person with a glass eye take the eye out of the socket.
11. It would bother me to see a rat run across my path in a park.
12. I would rather eat a piece of fruit than a piece of paper.
13. Even if I was hungry, I would not drink a bowl of my favorite soup if it had been stirred by a used but thoroughly washed flyswatter.
14. It would bother me to sleep in a nice hotel room if I knew that a man had died of a heart attack in that room the night before.

How disgusting would you find each of the following experiences? Please select a number (0-4) to indicate your answer:

0 = Not disgusting at all
1 = Slightly disgusting
2 = Moderately disgusting
3 = Very disgusting
4 = Extremely disgusting

15. You see maggots on a piece of meat in an outdoor garbage pail.
16. You see a person eating an apple with a knife and fork
17. While you are walking through a tunnel under a railroad track, you smell urine.
18. You take a sip of soda, and then realize that you drank from the glass that an acquaintance of yours had been drinking from.
19. Your friend's pet cat dies, and you have to pick up the dead body with your bare hands.
20. You see someone put ketchup on vanilla ice cream, and eat it.
21. You see a man with his intestines exposed after an accident.
22. You discover that a friend of yours changes underwear only once a week.
23. A friend offers you a piece of chocolate shaped like dog-doo.
24. You accidentally touch the ashes of a person who has been cremated.
25. You are about to drink a glass of milk when you smell that it is spoiled.
26. As part of a sex education class, you are required to inflate a new unlubricated condom, using your mouth.
27. You are walking barefoot on concrete, and you step on an earthworm.

Attention Check

Sometimes people move through surveys quickly without reading the questions and answering honestly. If you are paying attention and responding honestly, please respond “Strongly Agree”.

- Strongly Disagree

- Disagree
- Agree
- Strongly Agree

Culpepper Disgust Image Set (C-DIS; Culpepper et al., 2018)

The stimuli that will be used to rate feelings of disgust. Participants will view *either* the pathogen-salient stimuli or (2) pathogen-free stimuli. The 20 images will be presented to participants one-at-a-time and participants will be asked to rate each image for disgust on a 7-point scale (0 = *not disgusting at all*, 6 = *extremely disgusting*).

State Disgust

How disgusted do you feel right now? (rated on a sliding scale)

0 ----------100

TIME #2 Saliva Sample

It is now time to provide your TIME #2 saliva sample. Use the vial labeled "TIME 2" for this first sample. To reduce bubbling, place your straw into the provided TIME 2 vial and passively drool (you may want to tilt your head forward and to one side) through the straw into the vial without spitting your sample. If you are having trouble producing saliva, it is often helpful to rub your lower jaw or think about biting into a sour lemon or your favorite food. We need at least 2 mL of fluid saliva, not including bubbles, to properly analyze your sample. 2 mL is indicated on your vial, but we have emphasized this with a sharpie mark. When your fluid saliva reaches at least the 2 mL line, please remove your straw and tightly cap your TIME 2 vial.

IMPORTANT REQUIREMENTS: Use the provided disinfectant wipes to wipe and disinfect the sides of your vial after it is capped and place your disinfected vial into the provided plastic specimen bag labelled TIME 2. Move your bagged sample to your freezer until it can be returned to the research team. It is important to keep your sample frozen until it can be returned to Pryale Hall. To return the saliva sample, please transport the sample in a cooler with ice or gel packs and bring it directly to the researcher in Pryale Hall Room 102 at the agreed upon time.

Situational Pathogen Avoidance scale (SPA; Makhanova et al., 2021)

For this task, you will answer a questionnaire about your attitudes. For the following questions, try to imagine yourself being placed in these situations right now, that is at this moment in time. Please indicate how much you agree or disagree with each of the

following statements on a scale of 1 (Strongly Disagree) to 7 (Strongly Agree).

11. Right now, if I was standing next to a person who sneezed I would feel disgusted.
12. Right now, I would be grossed out if I shook a stranger's hand.
13. Right now, if someone coughed next to me without covering their mouth, I would move away from them.
14. Right now, it would make me uncomfortable to touch a door handle in a public restroom.
15. Right now, if I heard that a friend had the flu, I would avoid going to their house or apartment.
16. Right now, I would be bothered by sitting in a seat (that is still warm) that a stranger just got up from.
17. Right now, if a person looked like they were sick, I would be willing to shake their hand.
18. Right now, I would be happy to sit close to someone who just finished an intense workout.
19. Right now, I would try to sit on the opposite side of the room if I walked into the room where there was a person blowing their nose.
20. Right now, I would want to wash my hands immediately, if I shook hands with a person with sweaty palms.

Participant ID Code generation

Thank you for completing your first session. In order to link today's session with your upcoming Session 2, we must generate a participant ID code that will be unique to you. This will be entered once more at the beginning of your follow-up session to allow the research team to match your participation today with your next session. To create your unique participant ID code, please enter the following:

- First letter of middle name (e.g., Steve ***M***. Smith = "M"; If no middle name use X)
- First letter of mother's first name (e.g., ***A***nne Smith = "A")
- Last two digits of your phone number (e.g., (XXX) XXX–58***65*** = "65")
- The number of the month you were born in (e.g., March = "***03***")
- First letter(s) of your self-identified ethnicity (e.g., ***A***frican ***A***merican, ***H***ispanic, ***I***ndigenous ***A***merican, ***C***aucasian, ***O***ther)

APPENDIX E

STUDY 3 MATERIALS

Symbolic Racism 2000 Scale (Henry & Sears, 2002; Sears & Henry, 2005).

Will be administered twice to assess xenophobia towards immigrant minority groups and minority groups that are not necessarily immigrants.

[To assess attitudes towards immigrant minority groups]

1. It's really a matter of some people not trying hard enough; if immigrants would only try harder they could be just as well off as whites.
    a. Strongly agree
    b. Somewhat agree
    c. Somewhat disagree
    d. Strongly disagree
2. Irish, Italian, Jewish, and many other minorities overcame prejudice and worked their way up. Modern immigrants should do the same.
    a. Strongly agree
    b. Somewhat agree
    c. Somewhat disagree
    d. Strongly disagree
3. Some say that immigrant leaders have been trying to push too fast. Others feel that they haven't pushed fast enough. What do you think?
    a. Trying to push very much too fast
    b. Going too slowly
    c. Moving at about the right speed
4. How much of the racial tension that exists in the United States today do you think immigrants are responsible for creating?
    a. All of it
    b. Most
    c. Some
    d. Not much at all
5. Generations of discrimination have created conditions that make it difficult for immigrants to work their way out of the lower class.
    a. Strongly agree
    b. Somewhat agree
    c. Somewhat disagree
    d. Strongly disagree
6. Over the past few years, immigrants have gotten less than they deserve.
    a. Strongly agree
    b. Somewhat agree
    c. Somewhat disagree
    d. Strongly disagree

7. Over the past few years, immigrants have gotten more economically than they deserve.
    a. Strongly agree
    b. Somewhat agree
    c. Somewhat disagree
    d. Strongly disagree

[To assess attitudes toward minority groups that aren't necessarily immigrants]

1. It's really a matter of some people not trying hard enough; if people of color would only try harder they could be just as well off as whites.
    a. Strongly agree
    b. Somewhat agree
    c. Somewhat disagree
    d. Strongly disagree
2. Irish, Italian, Jewish, and many other minorities overcame prejudice and worked their way up. people of color should do the same.
    a. Strongly agree
    b. Somewhat agree
    c. Somewhat disagree
    d. Strongly disagree
3. Some say that leaders of color have been trying to push too fast. Others feel that they haven't pushed fast enough. What do you think?
    a. Trying to push very much too fast
    b. Going too slowly
    c. Moving at about the right speed
4. How much of the racial tension that exists in the United States today do you think people of color are responsible for creating?
    a. All of it
    b. Most
    c. Some
    d. Not much at all
5. Generations of slavery and discrimination have created conditions that make it difficult for people of color to work their way out of the lower class.
    a. Strongly agree
    b. Somewhat agree
    c. Somewhat disagree
    d. Strongly disagree
6. Over the past few years, people of color have gotten less than they deserve.
    a. Strongly agree

    b. Somewhat agree
    c. Somewhat disagree
    d. Strongly disagree
7. Over the past few years, people of color have gotten more economically than they deserve.
    a. Strongly agree
    b. Somewhat agree
    c. Somewhat disagree
    d. Strongly disagree

Revised Ethnocentrism Scale (Neuliep & McCrosky, 2013).

Rated on a 5-point scale from "Strongly Agree" to "Strongly Disagree".

1. Most other cultures are backward compared to my culture.
2. My culture should be the role model for other cultures
3. People from other cultures act strange when they come to my culture. (Dropped)
4. Lifestyles in other cultures are just as valid as those in my culture. (R)
5. Other cultures should try to be more like my culture.
6. I am not interested in the values and customs of other cultures. (D)
7. People in my culture could learn a lot from people in other cultures. (R)
8. Most people from other cultures just don't know what's good for them.
9. I respect the values and customs of other cultures. (R)
10. Other cultures are smart to look up to our culture.
11. Most people would be happier if they lived like the people in my culture.
12. I have many friends from different cultures. (D)
13. People in my culture have just about the best lifestyles of anywhere.
14. Lifestyles in other cultures are not as valid as those in my culture.
15. I am very interested in the values and customs of other cultures. (D)
16. I apply my values when judging people who are different. (D)
17. I see people who are similar to me as virtuous. (D)
18. I do not cooperate with people who are different.
19. Most people in my culture just don't know what is good for them. (D)
20. I do not trust people who are different.
21. I dislike interacting with people from different cultures.
22. I have little respect for the values and customs of other cultures.