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Although frequently characterized as undesirable, risk-taking is an integral part of human social life. Research into the factors that influence risk-taking therefore represents an important area of study in the evolutionary sciences. The current research draws from research on humans' pathogen avoidance psychology, life history theory, and sickness behavior to examine the role that pathogen threat plays in modulating risk tolerance. Across 4 studies, we measured or manipulated pathogen threat cues and examined their effect on risk tolerance. Results revealed a consistent pattern whereby people preferred less risk when the threat of illness was high. This pattern was observed using both self-report measures and behavioral assays but was eliminated when the threat was experimentally minimized. The current research provides evidence of a conceptual link between pathogen threats and global tolerance for risk, demonstrating a tendency to play it safe when the threat of illness is high.

Public Significance Statement
The current research examined how pathogen threats influence the psychology governing risk-taking. Four experimental studies found that when pathogen threats are made salient, people exhibit decreased willingness to take risks across domains (e.g., financial, social, health) and take fewer risks during a behavioral task. Results provide preliminary evidence that humans' pathogen management psychology may play an important role in modulating risk preferences in a variety of domains.

Keywords: behavioral immune system, decision-making, life history theory, risk-taking, sickness behavior

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In the first week of October 2014, stock market prices in the United States began to plummet unexpectedly after a steady climb of significant gains relative to previous years. At one point on October 8, the Dow Jones Industrial Average fell a staggering 460 points in one day, closing at its lowest point in nearly two years (Long, 2014). This crash lasted several weeks before the markets began a slow climb back toward stability (Fox Business, 2014).

At the same time, a second crisis was unfolding nearly 1500 miles away in a north Texas hospital: panic over the possibility of an outbreak of the highly infectious and deadly Ebola virus (World Health Organization Ebola Response Team, 2014). A patient admitted to the ICU presented a strange and severe constellation of symptoms that was quickly diagnosed as a worst-case scenario: an Ebola infection. Soon

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after the patient succumbed to the disease, two local nurses who had treated him contracted the virus. Fearing a possible outbreak, 177 others were placed under quarantine until it could be determined whether they, too, would become ill (Burrough, 2015; Zavis, Dixon, Hennessy-Fiske, & Morin, 2014).

Although the Ebola scare was relatively short-lived and geographically contained, financial analysts believe that the resulting uncertainty may have had national implications: prompting the unexpected October market plunge (Burrough, 2015; Fox Business, 2014). Indeed, journalists have speculated about outbreak effects on financial markets since the time of the Great Spanish Influenza Epidemic, which was believed to have triggered a 4-year economic depression in the early 1900s (Barro & Ursúa, 2009). More minor outbreak effects are evidenced each year during flu season, which predicts decreased stock market trading activity, reduced returns, and higher ask–bid spreads (McTier, Tse, & Wald, 2013).

Although journalists and analysts have long speculated about the existence of such outbreak effects, this phenomenon and the psychological factors that precipitate it have not yet been explored experimentally. Here, we use insights from evolutionary models of pathogen avoidance, life history theory, and sickness behavior to examine the relationship between pathogen presence and one such factor: risk tolerance. By showing how and why pathogen cues influence risk tolerance, the current research contributes to theory and research on pathogen avoidance psychology, sickness behavior, and the psychology governing risk-taking.

The Adaptive Problem of Pathogen Prevalence and the Behavioral Immune System

Parasites and pathogens are both ubiquitous and deadly and have thus played a critical role in human longevity and evolution (Finch, 2010; Morens, Folkers, & Fauci, 2004). Although contemporary humans have developed practices and technologies to help manage the spread of pathogens, infectious diseases are still a leading cause of death, accounting for approximately one quarter of all deaths worldwide each year (World Health Organization [WHO], 2004). Given the salient survivability threats posed by pathogens and parasites, humans have evolved a set of elaborate defenses that serve to protect the body from infection.

One such defense involves the suite of biological mechanisms that comprise the immune system. This complex set of bodily systems identifies nonself molecules and organisms within the body and responds to these intruders with a cascade of biological defenses designed to eliminate them. Although this system is remarkably efficient at managing numerous pathogen threats, activation of the immune system is metabolically expensive and imperfect (Dantzer, Kent, Bluthé, & Kelley, 1991; Gurven et al., 2016). For example, the inflammatory response that occurs in response to bacterial stimulation increases metabolic activity up to 13% (Dantzer et al., 1991) and—because immunocompetence is often domain-specific—it is unlikely that any one individual will possess all immune-relevant genes necessary to protect the body against all types of encountered threat (see, e.g., Casanova, Abel, & Quintana-Murci, 2011; Delves, Martin, Burton, & Roitt, 2011; Horowitz et al., 2013; Murphy, 2011). In light of these costs and imperfections, researchers have hypothesized that individuals also possess a set of defenses that function to mitigate the threat of disease before it comes into contact with the body. Among these responses are the mechanisms that comprise what psychologists refer to as the *behavioral* immune system (see, e.g., Ackerman, Hill, & Murray, 2018; Murray & Schaller, 2016; Schaller, 2016; Schaller & Park, 2011) or the evolved pathogen avoidance system (Tybur & Lieberman, 2016).

Humans' pathogen avoidance psychology comprises a complex array of mechanisms that function, ultimately, to prevent illness and disease transmission (Neuberg, Kenrick, & Schaller, 2011). These mechanisms have been designed to detect pathogen relevant cues in the environment and then generate psychological and behavioral changes aimed at minimizing those threats. For example, research finds that exposure to pathogen cues leads to avoidant or prophylactic behaviors (Curtis, de Barra, & Aunger, 2011; Tybur, Lieberman, Kurzban, & DeScioli, 2013), such as avoidance of people possessing cues heuristically associated with illness (Park, Faulkner, & Schaller, 2003; Park, Schaller, & Crandall, 2007), expression of more restricted sexual attitudes (Duncan, Schaller, &

Park, 2009; Murray, Jones, & Schaller, 2013; Schaller & Murray, 2008), desire for self-improvement solutions (Ackerman, Tybur, & Mortensen, 2018), and reduced motivation to contact and interact with other people in general (Aarøe, Osmundsen, & Petersen, 2016; Mortensen, Becker, Ackerman, Neuberg, & Kenrick, 2010; Park, 2015; Sacco, Young, & Hugenberg, 2014). Psychological and behavioral shifts such as these function to prevent contamination or infection at times when pathogen transmission risk is heightened.

Much research on humans' pathogen avoidance psychology has focused on the relationship between pathogen cues and psychological and behavioral changes aimed at mitigating the infection risk posed by *specific* individuals or contexts. For example, when infection risk is made salient, research finds that people pay more attention to sick-looking, but not healthy-looking targets (Ackerman et al., 2009; Miller & Maner, 2012). However, prior work has not directly addressed whether pathogen threat motivates changes in *general* risk propensity. Integrating the existing research into the large body of research on sickness behavior (e.g., Dantzer, 2001; Dantzer & Kelley, 2007) offers a new theoretical lens through which to examine such domain general impacts of infection risk on behavior: life history theory. Life history theory predicts that—in addition to activating psychobehavioral mechanisms that reduce interpersonal contact with infected others—cues indicating a heightened risk of illness will lead individuals to shift energetic resources inward, leading to global motivational shifts that promote resource conservation. Next, we will expand on this theory and its premises.

Life History Theory and Pathogens as Agents of Intrinsic Mortality

Life history theory is an established biological framework used to predict how and when organisms will allocate effort among the various tasks needed for survival and reproduction (Charnov, 1993; Roff, 1992; Stearns, 1992). Because bodily resources are inherently limited, organisms face important trade-offs in how they distribute these resources toward the various (and sometimes competing) tasks inherent in survival and reproduction. For example, because energy invested in immune system functioning cannot be used concurrently to attract a mate, life history theory predicts that individuals will have to make trade-offs when choosing how and when to invest in various life tasks related to mating versus bodily maintenance. How and when an individual resolves life history trade-offs such as these constitute an individual's life history strategy (Ellis, Figueredo, Brumbach, & Schlomer, 2009).

Broadly speaking, life history theory predicts that individuals will favor trade-offs that best promote survival and reproduction in their local ecology. These trade-offs will vary across the life course based on one's life stage, bodily condition, and current local ecology. When one lives in an environment where the risk of mortality from uncontrollable causes is high, this favors resource allocation trade-offs that prioritize mating effort over prolonged somatic investment (Chisholm, 1999; Chisholm et al., 1993; Stearns, 1992). For example, research finds that individuals who live in dangerous or unpredictable environments experience expedited sexual development, express a preference for a greater number of sexual partners, and are more likely to pursue higher-risk, higher-reward outcomes than those who develop in ecologies where the risk of extrinsic mortality is low (Belsky, Steinberg, & Draper, 1991; Ellis, 2004; Ellis et al., 2009; Griskevicius et al., 2013; Griskevicius, Tybur, Delton, & Robertson, 2011; Mata, Josef, & Hertwig, 2016; Mittal & Griskevicius, 2014; Wilson & Daly, 1997). Heavy investment in mating effort—even though it often comes at the expense of bodily growth and development (Campbell, Lukas, & Campbell, 2001; Rickard, Frankenhuis, & Nettle, 2014)—is favored in these contexts because effort invested in somatic development and maintenance will not significantly diminish one's risk of being killed when the risk of death from uncontrollable causes is high. The best evolutionary bet in such conditions is to invest heavily in behaviors aimed at more immediate reproduction to decrease the likelihood of perishing without having first had the chance to reproduce.

Although environments with a high risk of morality from uncontrollable causes (what researchers call an *extrinsic* mortality risk) favor faster life history strategies, a different set of strategies is favored when a person is confronted with ecological threats that pose a risk

of mortality from *intrinsic* causes. An intrinsic mortality threat is one that can be mitigated by increased energy allocation to one's own somatic development/maintenance (Stearns, 1992). In an environment that has a high rate of mortality from intrinsic causes, an individual's best evolutionary bet is to favor behaviors that promote conservation of resources for somatic development and repair, consistent with slower life history strategies. Such resource allocation decisions—although they come at the expense of more immediate reproduction—increase an individual's chances of survival, saving reproduction for a later date. However, because the majority of pathogen threats can be mitigated by increased energy allocation to one's own somatic development/maintenance (i.e., are intrinsic in nature: Rickard et al., 2014; Stearns, 1992; Waynforth, 2012), individuals may also exhibit more global psychological shifts in response to pathogen cues that function to help conserve bodily and financial resources that can be used to help minimize infection risk. For example, individuals who are chronically (e.g., such as living in a high pathogen load environment) or temporarily primed with pathogen cues show shifts toward a more restricted sociosexuality and greater endorsement of prophylactic use, congruent with a slower life history strategy (Murray et al., 2013; Schaller & Murray, 2008; Tybur, Bryan, Magnan, & Hooper, 2011).

Perhaps the most well-established example of increased somatic maintenance in the face of infection risk is the well-studied phenomenon of *sickness behavior* (Dantzer, 2001; Shattuck & Muehlenbein, 2015). Sickness behavior describes a global set of behavioral changes that occur when an organism is fighting illness or infection to minimize the risk of morbidity and mortality in its wake (Dantzer, 2001; Lopes, 2014). For example, sickness behavior is characterized by anhedonia, social withdrawal, and decreases in gain-seeking behaviors, such as foraging and mating (Dantzer & Kelley, 2007; Larson & Dunn, 2001). Although originally believed to be a maladaptive byproduct of infection, sickness behavior is now understood to reflect a constellation of responses orchestrated by the host's immune system that function to conserve energy, mitigate bodily damage from infection, and avoid injury or predation while the organism is in a weakened state (Aubert, Vega, Dantzer, & Goodall, 1995; Dantzer, 2001; Dantzer & Kelley, 2007; Harden, Kent, Pittman, & Roth, 2015; Kluger & Rothenburg, 1979; Medzhitov, Schneider, & Soares, 2012). These psychological and behavioral shifts come at the expense of a temporary reduction in investment in gain seeking pursuits, but they help the body conserve resources that can be redirected to immunological functioning and somatic repair, promoting survival in the face of an internalized, intrinsic pathogen threat (Dantzer, 2001; Dantzer & Kelley, 2007).

The Current Research

Here, we integrate insights from work on human pathogen detection systems with those from research on sickness behavior to hypothesize that externally occurring pathogen cues will lead individuals to exhibit decision-making shifts that would help the body conserve resources that can be redirected to immunological functioning and somatic repair even *before* becoming infected (Dantzer, 2001; Dantzer & Kelley, 2007). That is, individuals exposed to pathogen cues may exhibit global shifts in decision-making marked by withdrawal and decreased gain-seeking behaviors, even before becoming ill, as a means of conserving bodily and financial resources for healing and recovery—a form of *preemptive* sickness behavior.

We examined this possibility by focusing specifically on individuals' risk-taking psychology. Risk-taking is characterized by the possibility of achieving larger gains than can be realized from being cautious (Figner & Weber, 2011; Wang, Kruger, & Wilke, 2009), but come with a potential for loss (Hill, Ross, & Low, 1997; Kahneman & Lovallo, 1993; Weber & Johnson, 2008). The relative state model of risk-taking (Mishra, Barclay, & Sparks, 2017) posits that an organism's embodied capital and present goals interact with its current environment to create a relative state of deprivation or advantage that, in turn, promotes risky versus conservative behavior. For example, although animals who choose to forage outside their home range may find more bountiful food patches than those they typically graze from, roaming also comes at the risk of finding nothing at all. Risk-taking in this context is thus modulated by taking into consideration both the benefits associated with achieving a larger gain

than are available from their home ranges, as well as their ability to withstand a loss (Kacelnik, & Bateson, 1996). For animals that are near to starving—and are in state of relative *deprivation*—the potential benefits of finding life-saving food outweigh the costs of not finding any, as holding back from foraging guarantees starvation—a situation that Mishra and colleagues (2017) operationalize as *need* based risk-taking. Alternately, the benefits of foraging may be less valued in contexts where an animal's *ability*—or relative *advantage*—to successfully take foraging risks is diminished by other relevant energetic needs (Houston, Mc Namara, Barta, & Klasing, 2007).

In the context of a pathogen threat, the ability to withstand a loss is decreased because the body is in greater need of somatic resources with which it can use to mount an immune response should one become necessary (Houston et al., 2007; Lopes, 2014, 2017). As such, organisms that invest more in risk-taking may potentially bear more costs of infection (Barber & Dingemanse, 2010). For example, research finds that nonhuman animals who engage in risk-taking behavior—because of its association with energy expenditure and the potential for resource loss—experience more frequent and intense infections than more cautious conspecifics (Horváth et al., 2016; Marinov et al., 2017). Additionally in humans, gender differences in disgust sensitivity (a behavior-motivating BIS mechanism; Tybur et al., 2013) indicating lower disgust for men on average may help to explain men's higher rates of risk-taking and in turn, higher rates of infection-related mortality (Sparks, Fessler, Chan, Ashokkumar, & Holbrook, 2018). Thus, pathogen threats may lower tolerance for ability-based risk taking by raising the costs of potential losses. Accordingly, when individuals are confronted with pathogen cues, they may benefit from prioritizing smaller, more certain gains over larger, probabilistic ones. Here, we tested this hypothesis in a series of four exploratory studies using a variety of methods and measures. In each of our studies, we manipulated pathogen threat and then measured participants' general proclivity for situations involving risk and tolerance for risk in their decision-making. We will refer to these measures collectively as risk tolerance—which we operationalized as the amount of risk people are willing and able to accept during decision-making. We chose to use this term because it broadly encompasses each of the risk measures used in the current research. Risk tolerance was assessed by examining self-reported interest in risk-taking behaviors across a range of domains (Studies 1 and 2) and behavioral assays of risk-taking (Studies 3 and 4).

Study 1: Do Experimentally Primed Pathogen Threats Decrease Risk Tolerance?

Results of a pilot study found that greater feelings of situational pathogen threat were related to lower risk-tolerance (please see Appendix A of the online supplemental materials for pilot study methods and results). Specifically, the pilot study was conducted during a nationwide panic over Ebola in 2014 and found that participants who reported high worry about the outbreak also tended to report lower risk tolerance. Study 1 was designed to experimentally examine whether pathogen threat would lead to a reduction in risk tolerance. We predicted that participants exposed to pathogen threat cues would exhibit less risk tolerance than those in a comparably valenced control condition.

Method

Participants. Participants were 109 undergraduates (M_{age} = 19.49 years, SD = 1.14; 52 in the pathogen threat condition and 57 in the academic failure condition; 77 women). All students received partial course credit in exchange for their participation. We based initial sample size estimates for all studies in this paper, which were planned in Fall 2013 and conducted in Spring 2014 through Spring 2015, on the past convention of collecting a minimum of 30 participants within each experimental condition to achieve 80% power for a medium to large effect size (Cohen, 1988).

Procedure and materials. Participants completed the study in small groups while seated at privately partitioned computer terminals, using Qualtrics web-based experimental software. Participants were randomly assigned to view either a slideshow designed to prime pathogen threat, or a slideshow priming a negative control threat. Following the priming procedure, participants filled out a survey on risk

tolerance followed by a short battery of demographic questions. At the conclusion of the study, participants were debriefed, given credit, and then dismissed.

Priming slideshows. Pathogen threat was primed by having participants view a slideshow that ostensibly summarized a recent news story from *Newsweek* magazine. The slideshow was titled, "The Growing Problem of Disease in America: A Sick Future Ahead." This slideshow included captioned photos detailing how the rate of serious illness in America is increasing at a steady pace, despite modern advances in medicine (Hill, Prokosch, & DelPriore, 2015). Photos in the pathogen threat slideshow depicted scenes like doctors surrounding a patient on a hospital bed, and a crowd of people in a train station wearing hospital masks to avoid deadly, contagious strains of the flu. Participants in the control condition saw a slideshow titled "Making the Grade: No Longer a Walk in the Park," about the escalating threat of failing out of college, which contained photos portraying stressed looking college students, abandoned extracurricular activities, and papers marked with big, red *F* grades (see Hill, DelPriore, Rodeheffer, & Butterfield, 2014). This prime suggested that college would become harder, in general, without evoking individualized competitive disadvantage. Past research has shown that this control prime produces similar levels of anxiety, hopelessness, worry and disgust as the pathogen threat slideshow, but does not change one's beliefs about the threat of intrinsic mortality or of the threat of pathogens in the environment (Hill et al., 2015). Each slide was shown for 15 seconds.

Risk tolerance. To measure risk tolerance, participants completed the Domain Specific Risk-taking Scale, Part 1 (DOSPERT: Weber, Blais, & Betz, 2002), a measure of risk attitudes. We did not use the optional Part 2 of this scale, which asks people to rate perceptions of risk magnitudes and benefits, as we were primarily interested in measuring people's proclivity toward risk-taking, rather than people's outcome perceptions (Weber et al., 2002). The DOSPERT (Part 1), asks participants to indicate their likelihood of engaging in a series of risky behaviors on a five-point rating scale (endpoints: 1 = *very unlikely,* 5 = *very likely*). These risky behaviors cross a series of domains, such as health (e.g., "binge drinking"), financial (e.g., "taking a day's income to play the slot machines at a casino"), social (e.g., "dating someone you are working with"), ethical (e.g., "cheating on an exam"), and recreational (e.g., "exploring an unknown city or section of town"). Higher endorsement of each of these items indicates a more favorable attitude toward risk taking in that particular domain. In addition to assessing attitudes toward risk-taking across a series of distinct domains, the DOSPERT has shown good validity in predicting real-world risk-taking outcomes (Hanoch, Johnson, & Wilke, 2006; Szrek, Chao, Ramlagan, & Peltzer, 2012). We chose this scale because it would allow us to assess whether cues to pathogen threat produce lowered risk tolerance across multiple domains (as indexed by less favorable attitudes toward risk-taking in those domains), as predicted by our theory, or whether the exhibited shifts would only occur in domains directly related to health.

Results

Given the domain-specificity of the DOSPERT, we first tested whether there was an interaction between priming condition and risk domain by creating composites for each of the domain subscales (see Weber et al., 2002). We entered these scores into a 2 (Priming condition) × 5 (Domain; within-subjects) mixed model ANOVA. Results revealed no interaction between priming condition and risk domain ($p = .46$, $d = .18$), but did reveal a main effect of priming condition on risk tolerance across all domains, $F(1, 107) = 4.31$, $p = .04$, $d = .40$ (see Table 1 for descriptive statistics and Figure 1). Participants in the pathogen threat condition reported significantly lower risk tolerance ($M = 2.41$, $SE = .06$) than did participants in the academic threat condition ($M = 2.58$, $SE = .06$). The same pattern emerged when we analyzed the data using a single-factor ANOVA on the combined, full-scale composite of risk-taking likelihood ($\alpha = .84$), $F(1, 107) = 5.07$, $p = .03$, $d = .44$. Participants in the pathogen threat condition demonstrated lower risk tolerance ($M = 2.42$, $SD = .39$) than did participants in the academic threat condition ($M = 2.59$, $SD = .41$).

Because women have been found to be more disease cue sensitive (e.g., disgust sensitive; Sparks et al., 2018), and more risk averse than

Table 1
Descriptive Statistics for Study 1 (DOSPERT Scale)

	Academic threat				Pathogen threat			
Likelihood of taking	*M*	*SD*	Min	Max	*M*	*SD*	Min	Max
Ethical risks	1.87	0.57	1.00	3.25	1.75	0.58	1.00	3.50
Financial risks	1.88	0.60	1.00	4.20	1.80	0.63	1.00	3.80
Health risks	2.54	0.64	1.22	3.89	2.29	0.58	1.00	3.78
Recreational risks	3.51	0.77	1.20	5.00	3.25	0.58	1.80	4.60
Social risks	3.09	0.46	2.10	4.10	2.97	0.45	1.90	3.80
Total risk	2.59	0.41	1.76	3.43	2.42	0.39	1.68	3.43

Note. DOSPERT = Domain Specific Risk-taking.

men in general—and for the DOSPERT specifically (Byrnes, Miller, & Schafer, 1999; Weber et al., 2002)—we reran the above analyses and controlled for gender. We first entered DOSPERT scores, controlling for gender, into a 2 (Priming condition) × 5 (Domain; within-subjects) mixed model ANCOVA. Results again revealed no interaction between prime and DOSPERT risk domain ($p = .46$, $d = .18$). Gender was, however, a significant covariate, $F(1, 106) = 7.81$, $p = .01$, $d = .54$, and there was still a main effect of priming condition, $F(1, 106) = 4.46$, $p = .04$, $d = .41$, with the mean DOSPERT scores in each priming condition remaining virtually identical to the model where gender was not controlled. The same pattern emerged when gender was controlled for using a single factor ANOVA on the collapsed DOSPERT composite. Gender was again a significant covariate, $F(1, 106) = 9.39$, $p = .003$, $d = .59$. Even controlling for gender, priming condition remained significant with virtually identical results to the first model, $F(1, 106) = 5.32$, $p = .02$, $d = .43$.

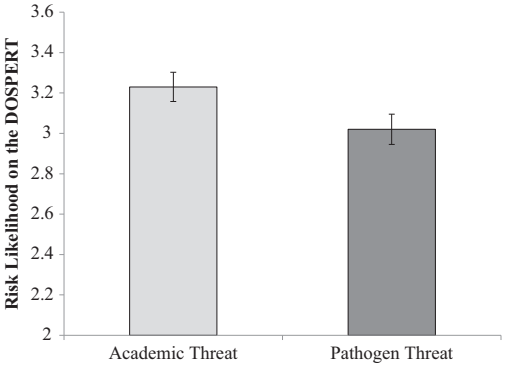

Figure 1. Participants' likelihood of risk-taking on the Domain Specific Risk-taking (DOSPERT) by condition (Study 1). Error bars reflect the standard error.

Discussion

The results of Study 1 found experimental support for the hypothesis that pathogen threats decrease risk tolerance. More specifically, the results of this experiment found that manipulated pathogen threat cues led people to report being less likely to participate in risky activities relative to people exposed to cues of an alternative type of threat (the threat of academic failure). Importantly, this pattern extended across domains of risk, rather than being specific to domains of risk that might increase one's immediate infection risk (e.g., health risks). Although the small sample size and number of comparisons made in this study limit the strength of conclusions that a reader can draw, these results provide preliminary support for the hypothesis that pathogen threats diminish global risk tolerance.

Study 2: Does Prophylaxis "Treat" Preemptive Sickness Behavior?

Study 2 was designed with two purposes in mind. First, we wanted to examine whether we would conceptually replicate the results of Study 1 (where participants reported a less favorable attitude toward taking a series of hypothetical risks) while using a different priming procedure: a detailed vignette about a situation connoting a pathogen threat. Second, we wanted to examine whether introducing a prophylactic intervention intended to reduce the potential for infection—hand washing—would

eliminate the impact of pathogen threats on risk tolerance. Research finds that performing simple, everyday disease avoidance behaviors such as hand washing mitigates downstream defensive psychological responses to pathogen threat cues (Huang, Sedlovskaya, Ackerman, & Bargh, 2011). Accordingly, if the decrease in risk tolerance found in Study 1 results from feeling vulnerable to pathogens, we should find that this decrease is attenuated by the hand-washing manipulation.

Participants in Study 2 were exposed to pathogen threat (or control) cues and were then asked to either wash their hands or complete a behaviorally similar, but disease-irrelevant control activity. Participants were then asked to report their attitudes toward risk-taking using the same measures used in Study 1. We predicted that participants exposed to pathogen cues would exhibit lower risk tolerance than those in the control condition. Additionally, we predicted that the hand-washing procedure would reduce or eliminate this effect.

Method

Participants. Participants were 178 men and women (94 female, M_{age} = 38.46 years, SD = 12.12; 90 in the pathogen condition, 88 in the control condition; 94 in the hand washing group, 84 in the no hand washing control group) recruited from Amazon Mechanical Turk to complete a 2 (Prime: control, pathogen threat; between-subjects) × 2 (Intervention Task: paper examination, hand washing; between-subjects) online study involving "person and product impressions" for a nominal payment.

Procedure and materials. Participants completed the study online. Upon providing consent, participants were randomly assigned to read a pathogen threat or control vignette. After reading the vignette, participants were randomly assigned to one of two intervention activities (pathogen avoidance or control activity). Participants then completed two measures of risk tolerance. The study closed with participants being asked to recall the content of the original vignette, how worried they felt when reading this vignette (1 = *not at all*, 7 = *extremely*), and to recall what activity they completed earlier in the study (hand washing, imagining hand washing, examining paper, imagining examining paper, or none), and the amount of time they spent engaging in this activity. These measures acted as manipulation checks. Finally, participants completed demographics, a suspicion probe, were debriefed and compensated.

Priming procedure. Pathogen threat was primed by having participants complete a guided imagery procedure in which they read a detailed vignette about their time volunteering in a hospital gerontology ward, where numerous cues of pathogens and infection were present. In the control vignette, participants completed a guided imagery procedure of comparable length and time, where they instead read about organizing their desk. Previous research has established that the pathogen vignette is effective at increasing pathogen concerns relative to the control vignette (Ackerman et al., 2018; White, Kenrick, & Neuberg, 2013).

Intervention procedure. Participants were randomly assigned to wash their hands with soap and water for 10 seconds (pathogen avoidance activity) or examine a blank piece of paper closely for 10 seconds (control activity). Both tasks involved comparable use of one's hands, but the washing activity was intended to interfere with and mitigate active pathogen threat (e.g., Huang et al., 2011), whereas the paper examination activity was not intended to induce any particular effect. Because the study was administered online, participants were told that if they did not have access to a piece of paper or a sink, they should instead imagine performing the specific activity in detail for the same amount of time. Participants were instructed to: "Think about each step in the process of wetting your hands, applying soap, rubbing your hands together, rinsing off the soap, and then drying them" [control: "Think about each step in the process of holding the paper, feeling it, holding it up to your eyes, turning it over, and examining it closely"]. Participants were instructed to: "try to imagine this as if it is really happening." Literature bearing upon mental simulation and grounded cognition support that mental simulation activates similar sensorimotor neural circuitry as actually performing the act (Barsalou, 2010; Gallese, Keysers, & Rizzolatti, 2004). Thus, we expected similar results for participants reporting that they actually washed their hands and those who simulated this procedure mentally.

To help ensure that participants completed the assigned task, we restricted participants from continuing to the remainder of the study for 5 s and, at the end of the study, we asked people to report the specific activity (or imagined activity) they performed earlier and the amount of time they performed this. Participants who reported a different activity than the assigned one or who reported performing this for less than the requested 10 seconds were excluded from the analyses.[1]

Risk tolerance. After the priming vignette, instructions informed participants that they would next complete several ostensibly unrelated tasks. The first task was a one-item measure of risk tolerance (Dohmen et al., 2011): "How do you see yourself? Are you generally a person who is fully prepared to take risks, or do you try to avoid taking risks?" Participants indicated their answer on a 10-point scale, (endpoints: 0 = *not at all willing to take risks*, 10 = *very willing to take risks*). Although only one-item, this measure has been found to be a good predictor of actual risky behaviors across different domains including stock investment, sports participation, sexual activities, and a variety of health-relevant actions such as smoking and drinking (Dohmen et al., 2011; Szrek et al., 2012). Participants also completed the DOSPERT (Weber et al., 2002), a measure of attitudes toward risk-taking across domains, as in Study 1.

Results

One-item risk measure. We first analyzed the one-item risk tolerance measure as a dependent variable by using a 2 (Priming condition) × 2 (Intervention condition) ANOVA. Results revealed no main effects of priming or intervention condition (ps ≥ .29, ds ≤.16), but did reveal a significant interaction between priming condition and intervention activity on reported risk tolerance, $F(1, 174) = 9.59$, $p = .002$, $d = .47$ (see Table 2 and Figure 2). To unpack the interaction, we first conducted planned comparisons examining the impact of priming condition at each level of the follow-up manipulation (washing hands vs. no hand washing). As expected given our earlier results, for those in the no hand washing group, participants in the pathogen threat condition exhibited diminished risk tolerance relative to what was observed among participants in the control condition, $F(1, 174) = 5.35$, $p = .02$, $d = .35$. For participants in the hand-washing intervention condition, however, pathogen threats increased risk tolerance relative to participants who read the control vignette, $F(1, 174) = 4.25$, $p = .04$, $d = .31$. We next examined the difference between the two intervention manipulations within each level of pathogen threat condition. Results revealed that, within the pathogen threat condition, participants who washed their hands reported greater willingness to take risks than participants who did not wash their hands, $F(1, 174) = 8.83$, $p = .003$, $d = .45$. No effect of the hand-washing manipulation was present in the control vignette condition, $F(1, 174) = 2.02$, $p = .16$, $d = .21$.

DOSPERT. To analyze participants' DOSPERT scores, composites were first created for each of the domain subscales as was done in Study 1 (Weber et al., 2002). A 2 (Priming condition) × 2 (Intervention condition) × 5 (Domain; within-subjects) mixed-model ANOVA revealed no main effect of risk domain, nor any interaction between risk domain and either of our other manipulated factors (ps ≥ .28, ds ≥ .17). These scores were therefore collapsed into a single, global risk tolerance composite ($\alpha = .92$), which served as the dependent measure in a 2 (Prime) × 2 (Intervention) ANOVA.

Results of the ANOVA again revealed no significant main effects of prime or intervention (ps ≥ .48, ds ≤.12), but did reveal a significant priming condition by intervention interaction (see Figure 3), $F(1, 174) = 5.71$, $p = .018$,

[1] The original MTurk sample included 191 men and women (99 female, $M_{age} = 37.73$ years, $SD = 12.18$; 95 in the pathogen threat condition, 95 in the control condition; 103 in the hand wash vignette, 88 in the control vignette) recruited from Amazon Mechanical Turk. Six participants were excluded for failing the vignette recall manipulation check. Seven other participants reported performing the intervention activity either incorrectly or for fewer than the 10 seconds required. This left 178 total participants to be included in our final analysis. We also ran the results without excluding any participants. The results were nearly identical in pattern and significance.

Table 2
Descriptive Statistics (Study 2)

Measure	Control threat				Pathogen threat			
	Hand washing		Control intervention		Hand washing		Control intervention	
	M	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
One-item risk tolerance measure	5.49	2.57	6.33	2.99	6.67	2.50	4.93	3.03
Likelihood of taking								
Social risks	3.40	0.52	3.47	0.70	3.43	0.64	3.26	0.77
Recreational risks	2.17	0.87	2.18	1.04	2.40	0.98	1.97	0.90
Financial risks	2.30	0.75	2.42	0.77	2.45	0.75	2.16	0.74
Health risks	2.21	0.67	2.45	0.89	2.60	0.78	2.34	0.74
Ethical risks	1.62	0.66	1.91	0.97	1.79	0.77	1.63	0.64
Total risk	2.34	0.48	2.49	0.66	2.53	0.57	2.27	0.56

$d = .36$.[2] We first conducted planned comparisons examining the impact of priming condition at each level of the follow-up manipulation (washing hands vs. no hand washing). Results revealed that, for participants who did not hand wash, being cued with pathogen threat did not reduce their risk tolerance relative to participants in the control condition, though the direction of the effect did conceptually mimic that of the effect found in Study 1, $F(1, 174) = 3.00$, $p = .085$, $d = .26$. This effect was not found for those in the hand-washing condition (in fact, the pattern was reversed from that in the control condition), $F(1, 174) = 2.71$, $p = .10$, $d = .25$. We also examined the differences between the two intervention manipulations within each level of pathogen threat condition. Results revealed that, within the pathogen threat condition, participants who washed their hands reported a greater likelihood of engaging in risky behavior compared with participants who did not wash their hands, $F(1, 174) = 4.84$, $p = .03$, $d = .33$. This effect did not occur for participants in the control priming condition, $F(1, 174) = 1.41$, $p = .24$, $d = .18$.[3]

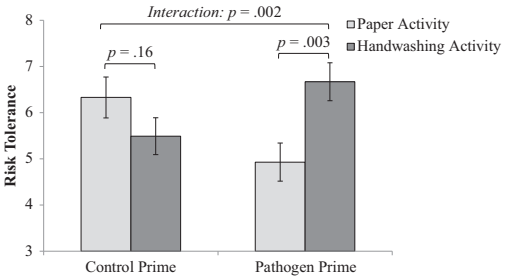

Figure 2. Participants' reported one-item risk tolerance by priming condition (pathogen threat vs. control prime) and vignette condition (Study 2). Plotted means represent the means for each condition. Error bars reflect the standard error.

Discussion

The results of Study 2 found mixed support for the hypothesis that pathogen threats diminish risk tolerance. Specifically, Study 2 found that exposure to pathogen cues led participants to report being less willing to take risks compared with participants in the control condition. This pattern of results was not conventionally

[2] Because past studies have found that women tend to score lower in risk tolerance when taking the DOSPERT and display greater risk aversion than men (Byrnes et al., 1999; Weber et al., 2002), we re-ran the analyses for priming condition by intervention activity on each dependent risk measure while controlling for gender. Results revealed for the one-item risk measure model, that although gender was a significant covariate, $F(1, 172) = 13.60$, $p < .001$, $d = .54$, the interaction between priming condition and intervention activity remained significant, $F(1, 172) = 7.30$, $p = .01$, $d = .39$. For the DOSPERT model, gender was also significant, $F(1, 172) = 9.66$, $p = .002$, $d = .46$, while the interaction between prime and intervention remained significant, $F(1, 172) = 1.37$, $p = .04$, $d = .31$.

[3] All analyses for both dependent measures were re-run including a variable coding for whether participants performed the intervention activity or merely imagined performing it, but no interaction effects for this variable emerged, Fs < 1. Thus, actually engaging in the behavior or mentally simulating this behavior each produced the same effects. This result is consistent with research demonstrating that simulation acts on many of the same mental processes as actual behavior (e.g., Ackerman, Goldstein, Shapiro, & Bargh, 2009; Decety & Grezes, 2006).

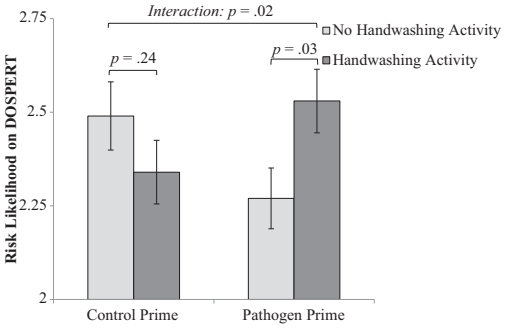

Figure 3. Participants' reported likelihood of risk-taking (Domain Specific Risk-taking [DOSPERT] score) by priming condition (pathogen threat vs. control prime) and vignette condition (Study 2). Plotted means represent the means for each condition. Error bars reflect the standard error.

significant when using the DOSPERT scale used in Study 1 but was significant for a single-item measure of risk tolerance. Although the DOSPERT effect was not significant, the overall pattern was consistent with predictions. Furthermore, the results of Study 2 found that an intervention activity intended to reduce the threat posed by pathogens—hand washing—eliminated or reversed this effect. Interestingly, this latter result was found regardless of whether the intervention activity was actually performed or merely simulated. Because hand washing is conceptually linked with prophylaxis and germ avoidance (Huang et al., 2011), this result lends convergent support for the hypothesis that pathogen threat manipulations reduce risk tolerance specifically because they increase one's feelings of current vulnerability to infection and disease.

An unanticipated result of Study 2 was that people who originally read the pathogen prime demonstrated *more* risk tolerance after washing their hands than did people who read the control prime and washed their hands. It is possible that feeling buffered from the salient threat of pathogens (through committing an act of prophylaxis) may lead one to take more risks than if that threat is not salient because the act of protecting oneself from a threat may be emboldening and therefore prompt greater risk tolerance (for an analogous crossover effect occurring in response to perceived invulnerability, see Huang, Ackerman, & Bargh, 2013). This interpretation would be consistent with research showing that people who feel more able to overcome the costs of risky behavior (here, participants engaging in the protective experience) take more risks (Mishra et al., 2017). Additionally, it is possible that once the threat is removed by cleaning, the residual arousal created by the potential danger of infection may strengthen consequent actions, such as risk-taking behavior more generally (Mano, 1992).

Study 3: The Impact of Pathogen Threat on Behavioral Risk-Taking

Studies 1 and 2 found convergent support for the hypothesis that pathogen threats decrease risk tolerance. Additionally, these studies have demonstrated an important boundary condition for this effect (an illness-prevention intervention). Study 3 was designed to build on these results by testing whether we could conceptually replicate the preceding results using a behavioral assay of risk tolerance. To test this possibility, we measured risk tolerance using a well-established behavioral risk-taking task that incentivizes risk-taking with the chance to win growing monetary payoffs (the Balloon Analogue Risk Task [BART]).

Method

Participants. Participants were 65 undergraduates (47 female; M_{age} = 19.30 years, SD = 1.39; 33 in the pathogen threat condition). Participants received partial course credit in exchange for their participation.

Procedure and materials. The experimental laboratory set up and priming procedure were the same as those used in Study 1. Participants were randomly assigned to view a slideshow on the growing threat of pathogens in their environment or a control slideshow about the increasing risk of academic failure. Participants were then asked to report on their affective response to the slideshow, followed by the BART (Lejuez et al., 2002), which was administered using Inquisit experimental software (lab Version 4.0). After completing the BART, participants answered a standard battery of demographic questions. Finally, they were thanked, debriefed, paid the amount of money earned during the BART, and dismissed.

Balloon Analogue Risk Task. The BART is a widely used, validated computerized risk-

taking task that consists of a series of virtual balloons that a participant must inflate one pump at a time to earn money (White, Lejuez, & de Wit, 2008). Participants inflate each balloon by clicking a mouse button. Each mouse click serves as a single pump. The participants earn a set increment of money for each pump (accruing more money as they continue to pump), but the balloon may pop at any given time. The participant earns as much money as he/she pumps into a balloon, so long as he/she ceases pumping and cashes that balloon in for payment before it pops. If the participant does not cash in before the balloon pops, he/she makes no money. The participant must therefore balance the competing desire to earn more money by pumping the balloon with the possibility that the balloon may pop at any moment and he/she will earn nothing. Greater persistence at pumping up a balloon is therefore a riskier strategy than administering fewer pumps. All participants were notified before the start of the task that they would be paid the amount of money they earned from the task at the end of the lab session. Participants participated in 10 trials. To minimize chances of any practice trial balloon breaks setting participants' expectations for the task, no practice trials were administered. Each pump was worth (.02 cents) and balloon breakpoints ranged from 1–64 pumps (Lejuez et al., 2002).

Results

To score participants' performance on the BART, we used participants' average number of pumps, adjusted so that all popped balloons were removed from the average, which is the scoring procedure recommended by the creators of this task (Lejuez et al., 2002). In our resulting mean composite, a higher score on the BART indicated greater risk-taking behavior (See Table 3 for descriptive statistics). We next analyzed the effect of priming condition on BART scores using a one-way between subjects (condition) ANOVA. Results revealed a significant effect of priming condition on risk-taking (see Figure 4), $F(1, 63) = 5.35$, $p = .02$, $d = .58$, demonstrating that participants in the pathogen threat condition delivered fewer pumps than participants in the control condition.[4]

Discussion

The results of Study 3 conceptually replicated the pattern of results observed in Studies 1 and 2 using a behavioral measure of risk tolerance. Specifically, Study 3 found that participants who were exposed to cues indicating a growing pathogen threat made fewer pumps in the BART task than did participants in the control condition. This shift occurred despite the fact that risky behavior in this context offered participants the possibility of receiving a higher monetary payoff than could be realized from playing it safe. This suggests that participants' perceptions of the benefits available from resource conservation in the face of a pathogen threat were sufficiently large to outweigh the opportunity cost of a larger reward gain, even in a case where risk-taking was explicitly incentivized. However, the small sample size of this study warrants some caution when interpreting the results on their own.

Study 4: Does an Internal Pathogen Threat Mimic the Effects of External Pathogen Threat on Risk-Taking?

The results of Studies 1–3 found that externally primed pathogen threats predict safer, more conservative decision-making. Study 4 sought to build upon these results in three key ways. First, we sought to examine whether we would conceptually replicate the results of Study 3 using a second behavioral measure of risk-taking: a gambling task consisting of a series of choices between a safer, smaller reward and a larger but uncertain reward (Griskevicius et al., 2011, 2013). Second, we tested whether this effect is specific to pathogen threats—as we have hypothesized—or whether it extends to any survivability threat. To this end, we introduced a novel control condition that portrayed a different, but equally manageable type of threat to one's survival: a slideshow news story about

[4] Because men tend to make more pumps on the BART than women (Lejuez et al., 2002), we tested the effect of priming condition on BART scores, while controlling for gender. We found that gender was not a significant covariate, F (1, 62) = .32, p = .57, d = 005. Further, after controlling for gender, the previous pattern remained- albeit less significant—where participants in the pathogen condition made fewer pumps than those in the control condition, $F(1, 62) = 4.18$, $p = .045$, $d = .51$.

Table 3
Descriptive Statistics (Study 3)

Measure	Academic threat				Pathogen threat			
	M	*SD*	Min	Max	*M*	*SD*	Min	Max
Average number of pumps on BART	21.04	9.69	5	37.5	15.89	8.20	0	31.14
Total payout for BART task	1.04	0.51	0.41	2.75	1.26	0.61	0	2.20

Note. BART = Balloon Analogue Risk Task.

the growing threat of deadly household accidents. Lastly, we designed Study 4 to examine whether internal cues of pathogen presence—the catalyst of classic sickness behavior—produce a decrease in risk tolerance similar to external pathogen cues. If diminished risk tolerance is capturing a strategic resource allocation shift toward somatic maintenance and repair (i.e., "preemptive" sickness behavior), we should find that individuals recovering from illness—and who are exhibiting actual sickness behavior—should exhibit similar patterns of decision-making as those exposed to external cues of pathogen threat. To examine this possibility, we included measures assessing recent illness. We predicted that participants who were healthy would respond to pathogen threat cues by choosing fewer risky gambles compared with those in the control condition. For those whose immune systems were currently being challenged by illness, however, we predicted a preference for fewer risky options regardless of priming condition.

Method

Participants. Participants were 72 undergraduates (M_{age} = 19.89 years, *SD* = 1.70; 42 in the pathogen threat condition and 30 in the accident failure condition; 54 women). All students received partial course credit in exchange for their participation.

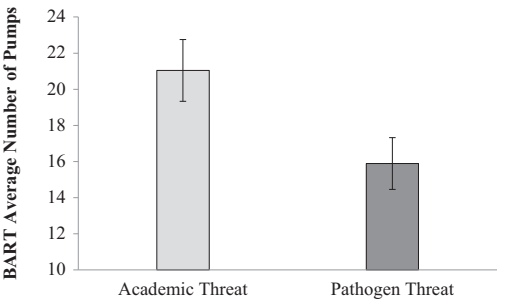

Figure 4. Participants' average number of pumps on the Balloon Analogue Risk Task (BART) as a function of priming condition (Study 3). Error bars reflect the standard error.

Procedure and materials. Participants completed the same ruse and testing paradigm as in Studies 1 and 3. After viewing their assigned slideshow (pathogen threat or household accidents), participants completed a gambling task, followed by a measure of current/recent illness, and a short battery of demographic questions. At the end of the study, participants were thanked, debriefed, and dismissed.

Priming manipulation. Our experimental priming procedure and pathogen threat prime were the same as Studies 1 and 3. Again, participants viewed each slide in their assigned prime for 15 seconds. The control survivability threat was primed by having participants view a similarly formatted slideshow titled, "Home Dangerous Home: Serious Household Accidents on the Rise." This slideshow told a story about a recent rise in deadly household accidents. The photos in the slideshow depicted accident scenes like an injured man sprawled on the ground after a fall, a car flipped over in someone's driveway, and a kitchen on fire from defective cooking equipment. We chose this control topic because past research has found that the threat of household accidents is comparably negative and elicits comparable concerns with morbidity/mortality relative to pathogen threats (Park et al., 2007) and it would allow us to identify whether reductions in risk tolerance are specific to the risk posed by pathogens (similar to sickness behavior) or whether they occur in response to any type of potentially controllable mortality threat.

To ensure that our two priming slideshows (pathogen threats and household accidents) were comparable with one another in the degree to which the depicted threat was perceived as controllable but differed in the degree to which

they raised concern with pathogens, we conducted a pretest. We recruited a separate sample of 19 participants (four male, 15 female), aged 18–22 (M_{age} = 20.21, SD_{age} = 1.08). Participants viewed both primes presented in random order by the computer.

After viewing each slideshow, participants responded by indicating their agreement or disagreement with statements regarding how the slideshow made them feel. The first set of questions were related to how the slideshow impacted participants' perception of control over the future: (a) I cannot do anything to avoid the problems that I will run into in the future, (b) I have no control over my future, (c) whether I live a long life or not has more to do with luck than the decisions that I make, and (d) my health and well-being is determined by factors out of my control. The set of statements yielded good reliability (α = .88 after pathogen prime; α = .77 after accident prime) and were summed to create a composite measure of perceived control. Next, participants responded to statements regarding their concern about pathogens after each prime: (a) I am very likely to get sick later this year, (b) infectious disease is everywhere this year, (c) even with medical advances; disease is a greater problem than it used to be, and (d) I am likely to get a serious disease sometime in my life. These statements also yielded good reliability (α = .86 after pathogen prime; α = .94 after accident prime) and were formed into a composite measure of pathogen concern. We predicted that the distinct threats presented in the primes would not differ in their impact on perceived control, but the pathogen threat prime would elicit significantly greater pathogen concern than the household accidents prime.

We next conducted a one-way within-subjects ANOVA to test for differences in perceived ability to control the future and disease concern. The results of the analyses revealed that the slideshows did not differ in the degree to which they elicited feelings of lacking control over the future (p = .31). However, the slideshows differed in the degree to which they made participants concerned about pathogens, $F(1, 18) = 73.75$, $p < .001$, $d = 2.31$, with more illness concerns being reported after the pathogen prime (M = 4.70, SD = 1.10) than after the accident prime (M = 2.08, SD = 1.17). Thus, deadly household accidents represented a threat that is comparably controllable to the threat of pathogens. However, the pathogen prime increased pathogen concern to a greater extent than the control prime.

Risk tolerance. To measure risk tolerance, participants completed a behavioral choice task (Griskevicius et al., 2013). The task consisted of 20 choices between a small, certain amount of money or a chance at gaining a larger amount of money (e.g., "Do you want $37 for sure, or a 54% chance to get $54?"). The certain rewards ranged from $10 to $61, and the risky bet amounts ranged from $47 to $99. Participants' preference for risky versus safe options was calculated by summing the number of time participants chose the probabilistically risky versus safe (certain) outcome (Griskevicius et al., 2013).

Recent illness. The body's immune response is active both during infection and for several days after recovering from illness (LeVine, Koeningsknecht, & Stark, 2001; Van der Sluijs et al., 2004). Accordingly, we measured participants' recent experiences with illness by asking them about their health in the last week (e.g., "I have felt sick within the past week"; see Miller & Maner, 2011). All ratings were made on 7-point rating scales (end points: 1 = *strongly disagree*, 7 = *strongly agree*), with higher scores corresponding to a greater likelihood of immune system activation (the driver of sickness behavior; see, e.g., Dantzer, 2001).

Results

Preliminary analyses. First, we created a mean composite score of participants' reported recent illness experience (α = .91). Twenty-three people scored as being at midpoint or higher (4 or higher) on the scale for experiencing recent illness. Next, to confirm that the priming procedure did not influence participants' reported experience with recent illness, we conducted a univariate ANOVA with illness experience as the dependent variable and priming condition (pathogen threat vs. accident threat) as the grouping variable. The results revealed that there was no effect of priming condition on participants' reported experience with recent illness (p = .48, d = .17).

Risk-taking. First, we created our composite measure of preference for risky versus safe monetary outcomes by summing the number of

risky options chosen ($\alpha = .88$). Next, to test our primary predictions we regressed participants' risk-taking scores on priming condition (dummy coded) and reported experience with recent illness (centered; each in the first step) and the interaction between these two variables (in the second step). Results did not reveal a significant main effect of priming condition, $b = -.57$ ($SE = .31$), $t(68) = -1.70$, $p = .07$, semipartial $r^2 = .04$, although the conceptual pattern of results trended toward participants picking fewer risky options in the pathogen threat condition compared with the control condition (see Table 4 for descriptive statistics). There was, however, a significant two-way interaction between priming condition and experience with recent illness on participants' risk-taking, $b = -1.32$ ($SE = .64$), $t(68) = -2.05$, $p = .04$, semipartial $r^2 = .05$ (see Figure 5).[5] Simple slope tests revealed that, for participants in the control condition, higher scores on our measure of experience with recent illness (higher scores reflecting a more recent incidence of infection) were predictive of choosing fewer risky monetary options, $b = -1.44$ ($SE = .52$), $t(68) = -2.76$, $p = .007$, semipartial $r^2 = .10$. There was no effect of experience with recent illness on risk-taking among people in the pathogen threat condition ($b = -.12$, $p = .76$, semipartial $r^2 = .001$), however.

We next examined the impact of the pathogen threat prime on people relatively high (1 *SD* above the mean) and low (1 *SD* below the mean) in experience with recent illness (e.g., infections). The pathogen prime led healthy people (i.e., people who reported 1 *SD* below the sample mean of experience with recent illness) to report a significantly lower likelihood of risk-taking relative to similar participants in the control condition, $b = 4.16$ ($SE = 1.56$), $t(68) = 2.67$, $p = .01$, semipartial $r^2 = .09$. We did not observe a priming effect for those who reported high incidence of recent health problems (i.e., participants who reported 1 *SD* greater experience with recent illness than the sample mean), however ($b = -.37$, $p = .81$, semipartial $r^2 < .001$).

Discussion

The results of Study 4 found continued support for the hypothesis that pathogen cues—whether externally or internally primed—predict more cautious, conservative decision-making. Importantly, the biggest shift in risk tolerance was seen in those who had been healthy over the prior week. Individuals who were currently recovering from illness—and thus, were already exhibiting real sickness behavior—exhibited cautious decision-making regardless of priming condition. These results lend continued evidence for a conceptual link between pathogen threats and reduced risk tolerance. Study 4 also replicated the pattern of lowered risk-taking seen in the previous studies using a novel control prime—the threat of deadly household accidents. Although the main effect was not conventionally significant, the direction of the pattern that emerged was consistent with the pattern in Studies 1–3, suggesting that the demonstrated preference for safe decisions is not driven by a general decrease in risk tolerance that occurs in response to any threat. Further, the small sample size of this study lowers the power needed to test results, so the unique results of this study should be interpreted with caution.

Internal Meta-Analysis

Given that there were some inconsistencies in the results across studies (e.g., the lack of main effect in Study 2) and some of our studies had relatively small sample sizes, we conducted an internal meta-analysis (as recommended by Maner, 2014) to examine the reliability of the main effect of pathogen cues on risk tolerance across the current exploratory studies (Rosenthal, 1991).

Following procedures outlined by Goh, Hall, and Rosenthal (2016), the effect sizes of the main effect of pathogen threat on risk tolerance across our 5 studies (the pilot and studies 1 – 4: total $N = 500$) were first examined using a full random-effects model, in which mean effects

[5] Because women have been found to behave more risk aversely on the gambling task we chose to use for the current study (Griskevicius et al., 2011), we re-ran our regression analysis with gender dummy coded and added into block one of our model as a covariate. Results revealed that gender was not a significant covariate, $b = .41$ ($SE = 1.24$), $t(67) = .33$, $p = .74$, semipartial $r^2 = .001$. The inclusion of gender as a covariate to the model did not change the pattern of the interaction between priming condition and recent illness, $b = -1.39$ ($SE = .66$), $t(67) = -2.12$, $p = .04$, semipartial $r^2 = .06$.

Table 4
Descriptive Statistics (Study 4)

Measure	Accident threat				Pathogen threat			
	M	*SD*	Min	Max	*M*	*SD*	Min	Max
Recent illness	3.24	1.56	1	7	2.95	1.81	1	7
Number of risky financial choices	10.30	4.74	1	18	8.67	4.45	0	17

were unweighted because of differences in our control prime and our dependent risk variable across studies. Because Study 2 included two dependent measures (a one-item risk measure and the DOSPERT), we created a mean effect size composite of the *d* across both risk measures and used this composite as the effect size for Study 2 going forward (Goh et al., 2016).

We next converted each study's effect size for the main effect of priming condition upon risk tolerance from Cohen's *d* into Pearson's *r* to facilitate the next step of calculating a standardized measure of effect size. The resulting *r* values were as follows: $r_{pilot} = .2376$, $r_{Study1} = .2126$, $r_{Study2} = .0069$, $r_{Study3} = .2241$, $r_{Study4} = .1758$. We next applied Fisher's z transformations to each study effect size *r* then conducted our analysis on the standardized values. Following this, results were converted back to Pearson's *r* and reported as *r* for ease of interpretation. Results of a random effects test of the mean effect size against 0 revealed a small but significant effect of pathogen threat on risk tolerance, with participants exhibiting lower risk tolerance in the pathogen threat conditions of our studies than the control conditions, $r = -.17$, $t(4) = -4.04$, $p = .02$ (two-tailed), 95% CI [−.29, −.05].

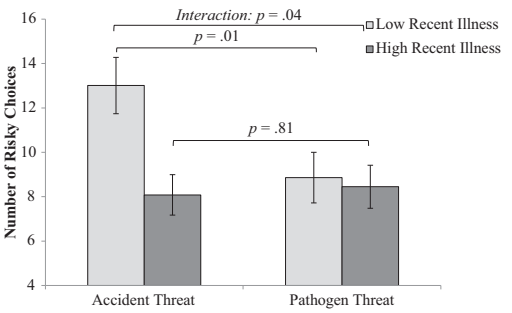

Figure 5. Participants' gambling behaviors as a function of priming condition (pathogen threat vs. accident) and recent illness (Study 4). Plotted means represent one standard deviation above and below mean illness. Error bars reflect the standard error.

Next, to test for heterogeneity, Cochran's Q was computed (Cochran, 1954). Results of a chi-squared test on the Cochran's Q value revealed no evidence of heterogeneity in our study effects, $\chi^2(4) = 5.05$, $p = .28$. As a second measure of heterogeneity/inconsistency between studies, we also calculated an I^2 statistic (Higgins, Thompson, Deeks, & Altman, 2003). For interpretation of I^2, a value of 0 reflects little to no inconsistency (e.g., little to no heterogeneity impacting the results of this mini meta-analysis), whereas values above 0 indicate increasing percentage of heterogeneity (up to 100%). The I^2 revealed a small to moderate amount of heterogeneity in effect sizes across our studies, $I^2 = 20.83$ (Higgins et al., 2003). A small to moderate amount of heterogeneity in our effect size across studies is not unexpected, because of the small number of studies included in our mini meta, and because Studies 2 and 4 included extra moderating variables (like a handwashing intervention and recent illness history) in addition to a main priming condition. Given that both heterogeneity and inconsistency measures yielded only low to moderate heterogeneity, we also reran the mini meta-analysis using a fixed effects model. Results were similar to those of the random effects model, revealing again that the effect was small but significant, $r = -.14$, $Z = -3.44$, $p = .0006$ (two-tailed). Although the authors urge readers to exercise caution when drawing implications about the current research given the limited sample size of several studies in this paper (more replication is needed), results of the meta-analysis suggest that the impact of pathogen threats on risk tolerance is robust, though small. Because of the small sample sizes of the original studies, it is possible that the original effect sizes pooled for this meta-analysis may be somewhat inflated and that future studies

may find similar or smaller effect sizes than the ones reported in this paper.

General Discussion

Research on the behavioral immune system has linked disease threat to behaviors aimed at reducing specific known pathogen contamination risks (e.g., reducing sexual behavior; Murray et al., 2013). In the current research, we extended this research by hypothesizing that pathogen cues in one's external environment would promote psychological modifications similar to sickness behavior that function to help one avoid becoming infected, and to conserve bodily and financial resources in face of potential infection. We examined this possibility by focusing specifically on individuals' risk-taking psychology, predicting that pathogen cues would decrease tolerance for risk-taking broadly across domains of decision-making.

We found support for this hypothesis across four exploratory studies. In Study 1, diminished risk tolerance in response to an experimentally primed pathogen threat occurred across multiple decision domains (e.g., financial, recreational, social). Study 2 found that performing physical actions which help to minimize infection risk (using a hand washing intervention) ameliorated participants' reduction in risk tolerance following a pathogen threat, though results were mixed across measures. Study 3 found evidence for diminished behavioral risk-taking in response to a pathogen threat, even in a case when risk-taking was financially incentivized. Lastly, Study 4 replicated this effect using a different behavioral risk-taking task and demonstrated that lowered risk tolerance was also found among individuals who were currently experiencing an internal pathogen threat (i.e., who were currently ill or had been recently ill at the time of the study). Across these studies, controlling for gender did not change the pattern of results. However, because of the limited sample sizes employed in the studies of this paper, we caution readers against making any firm conclusions about the presence or absence of gender-differences in risk tolerance following a pathogen prime. Larger scale replication of these studies is needed before any firmer conclusions can be reached.

Taken together, our results provide preliminary supportive evidence of a conceptual link between pathogen threats and risk tolerance. Further, the current results suggest that information about pathogen load in the environment may be used to motivate preemptive sickness behaviors, whose function is similar to that of actual sickness behaviors that occur once one becomes ill. To our knowledge, this is some of the first empirical research to combine research on sickness behavior (Dantzer & Kelley, 2007; Larson & Dunn, 2001) and the behavioral immune system (Schaller & Park, 2011) to make these predictions. This research therefore adds to a growing literature on risk-reward psychology (Figner & Weber, 2011; Kahneman & Lovallo, 1993; Mishra et al., 2017; Weber & Johnson, 2008), the impact of pathogen threat on cognition (Ackerman et al., 2009; Miller & Maner, 2011; Murray & Schaller, 2016; Neuberg et al., 2011), and to the growing body of research investigating ecological impacts on risk-taking strategies (Ellis et al., 2009; Griskevicius et al., 2011, 2013; Hill & Buss, 2010; Mata et al., 2016; Wang et al., 2009).

Limitations and Future Directions

The current studies found that pathogen threats reduced risk tolerance—a strategic resource allocation shift that can help minimize both the risk of infection and the costs of potential illness. As this area of research is largely unexplored in humans, future research is warranted into examining how pathogen threats impact resource allocation in specific domains critical to fitness, such as mating, parenting and foraging (Ellis et al., 2009; Houston et al., 2007), and how these resource allocation shifts are moderated by features of the person and the situation (Figner & Weber, 2011; Mishra et al., 2017). Because sickness behavior describes a relatively short-term motivational state that functions to promote long-term fitness interests, it is possible that sickness behavior (and risk tolerance more generally) can be up- and down-regulated based on other features in the environment that have consequences for fitness (see Lopes, 2014 for a review). For example, research on nonhuman animals finds that males that are made sick from injections with lipopolysaccharide (LPS) exhibit significantly less sickness behavior when presented with an attractive mating opportunity than when alone (Lopes et al., 2013; Yirmiya, Avitsur, Donchin,

& Cohen, 1995). Additionally, female sparrows injected with LPS were less likely to abandon their nest and brood as a result of sickness behavior when the fitness costs of eluding parenting responsibilities were highest (e.g., when offspring brood size was large; Bonneaud et al., 2003). Future inquiry could examine how the presence of competing threats and opportunities in other fitness-related domains attenuate human risk psychology in response to pathogens (Kenrick, Griskevicius, Neuberg, & Schaller, 2010; Neel, Kenrick, White, & Neuberg, 2016; Sacco et al., 2014). For example, being presented with a mating opportunity while exposed to a pathogen threat may prevent the shifting of resources from mating to somatic effort by diverting resources back to mating effort. Similarly, we should expect increased mating effort in cases where the pathogen threat changes from being a manageable, intrinsic threat to an extrinsic threat that one has little hope of being able to combat (Hill et al., 2015).

The results of the current research pave the way for several lines of future research examining the biological mechanisms guiding these effects and their downstream consequences for other areas of research. For example, research shows that sickness behavior is largely mediated by the release of small weight signaling proteins called cytokines (Kent et al., 1992; Vollmer-Conna et al., 2004). Recent experimental social psychology research finds that these same cytokines are released in response to external pathogen cues (Schaller, Miller, Gervais, Yager, & Chen, 2010), making it possible that they play a role in modulating the types of 'preemptive sickness behaviors' observed in the current research, as well as classical sickness behaviors.

Future research would also benefit from examining how various behaviors that are influenced by global changes in risk tolerance change in response to pathogen cues. For example, one prediction that falls from this framework is that the endowment effect (people's tendency to overvalue their own property) may be exaggerated in areas of chronically high pathogen prevalence or when future chances of disease contraction are high (Li et al., 2016; McTier et al., 2013). Alternately, it is possible that when disease threat is imminent, emotions like disgust might lead to devaluation of [potentially contaminated] resources (Huang, Ackerman, & Newman, 2017; Huang, Ackerman, & Sedlovskaya, 2017; Lerner, Small, & Loewenstein, 2004). As an extension of this possibility, future studies may also examine the role that individual differences in sensitivity to pathogen cues play in altering the influence of pathogen threats on decision-making. For example, recent research has found that people high in disgust sensitivity report lower risk tolerance across multiple risk domains, compared with people less sensitive to pathogen cues (Sparks et al., 2018). It is possible that a future replication and extension of the current research may find that people who are most sensitive to pathogens (e.g., have high germ aversion or high pathogen disgust) are lower in risk tolerance across multiple priming conditions. Alternately, it is possible that shifts in disgust or germ aversion might mediate the relationship between pathogen threat and risk tolerance—though this possibility was not tested in the current set of studies. In light of these possibilities, it is clear that researchers have only begun to scratch the surface of the relationship between pathogen threat and decision-making.

Additionally, future research would benefit from tying in insights from life history theory into individuals' reactions to pathogen concerns. For example, research finds that a variety of environmental stressors present in early childhood, such as lower socioeconomic status and fluctuating nutrition quality in utero, can lead to reduced immune system functioning in adulthood (McDade, 2003, 2005; Miller et al., 2009), leaving these individuals more vulnerable to infectious diseases than individuals who grew up in more benign environments. Although illness typically represents an intrinsic threat (i.e., a threat that can be forestalled by allocating energy toward somatic needs), individual differences in vulnerability determine one's ability to mitigate the threat, and as a result, the appropriate adaptive response (Hill et al., 2015; Rickard et al., 2014). Therefore, for those who are chronically sick (or vulnerable to getting sick), and thus less able to mitigate pathogen threat with somatic effort, future survivability in face of pathogen threats is more uncertain and the needs to accrue resources prior to forestall potential death may outweigh the benefits of being cautious and prompt greater risk taking—an adaptive behavioral response consistent with a faster life history strat-

egy (Hill, Boehm, & Prokosch, 2016; Mishra, 2014; Mishra et al., 2017; Waynforth, 2012).

Conclusion

The threats posed by infectious illness activate the behavioral immune system, which promotes disease avoidance behaviors (Schaller, 2016). Pathogens, like other intrinsic threats, also demand allocation of energetic resources toward somatic effort to mitigate damage and promote recovery (Rickard et al., 2014; Stearns, 1992). Sickness behavior involves a constellation of symptoms that together function to conserve resources for somatic maintenance and repair in response to cues of pathogen presence (Dantzer & Kelley, 2007; Larson & Dunn, 2001). In humans, this process of avoiding the depletion of resources that are vital to enduring a period of sickness may involve forgoing risky decisions altogether. Allocating energy away from riskier gain-seeking behaviors and effort in other domains—such as mating—can promote survival and recovery when the costs posed by infection outweigh the potential benefits of risk-taking (Ellis et al., 2009; Haselton & Nettle, 2006; Lopes, 2014). In the current research, we find preliminary support across four exploratory studies for the hypothesis that pathogen threat leads to a domain general shift toward cautious decision-making. As such, disease concern may be an important and largely unexamined factor that influences risk perception, decision-making, and energetic investment.

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