LIFE HISTORY AND PATHOGEN SUSCEPTIBILITY: EFFECTS ON RISK TAKING BEHAVIORS

by

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Life History and Pathogen Susceptibility: Effects on Risk Taking Behaviors

When Tomorrow Rodriguez was picked to compete on the famous game show *Deal or No Deal*, she was living month to month. When interviewed, she admitted to being over a hundred thousand dollars in debt from bills, school loans, and bad choices (Winfrey, O. & Rodriguez, T., 2009). Although Tomorrow's risks taken in the past may have cost her, this time her big gamble paid off- Tomorrow walked off the show with the one million dollar grand prize. To win, a contestant must painstakingly eliminate 26 briefcases of money in the hopes that the one they have blindly chosen to hold contains a large cash prize, all the while turning down smaller but certain prize offers made by the show's banker. Many individuals give up early in the show and take the smaller, safe bet rather than risk leaving with nothing. Very few have the nerve to last to the end and even fewer have the luck to win the grand prize. What is it that separates those who are willing to put it all on the line in hopes of getting a big payoff from those who prefer a safer but smaller reward? Further, why are some able to delay gratification while others jump at the first offer made?

For decades, researchers have been interested in examining this question by gauging how people perceive and react to risky choices (see Yates, 1992 for a broad overview). While the factors that shape risk perception and decision making are numerous, a growing body of research grounded in principles from evolutionary biology suggests that some of these differences might emerge from ecological and developmental differences that impact the benefits available from earlier versus delayed reproduction (Daly & Wilson, 2005; Wang, Kruger, & Wilke, 2009; Wilson & Daly, 1997). Conditions favoring earlier reproduction promote fast life history strategies, characterized by rapid growth and more immediate mating effort. Conditions favoring later reproduction encourage the development of slower life history strategies, characterized by prolonged growth and delayed reproduction

(Griskevicius et al., 2013; Griskevicius, Tybur, et al., 2011; Kaplan & Gangestad, 2005). In general, faster strategies are favored in ecological contexts in which survivability into adulthood is less certain because it decreases the likelihood that a person will perish without first having had the chance to reproduce (Griskevicius, Tybur, et al., 2011; Liu, Feng, Suo, Lee, & Li, 2012). Indeed, past research finds that activating cues to a growing mortality threat alters one's sense of control over his or her future, which favors present-oriented decision-making characterized by a desire for sooner, smaller monetary rewards and greater financial risk-taking (Griskevicius, Tybur, et al., 2011; Liu, Feng, Suo, Lee, & Li, 2012; Mittal & Griskevicius, 2014).

In the following, I will build on this past research by investigating how one's vulnerability to illnesses and the perceived pathogen load impact behaviors that are consistent with faster and slower life history strategies. Specifically, I will examine how these cues impact temporal focus and risk taking behavior. I predict that individuals who are vulnerable to infection (indexed by a poorer health history) will respond to the threat of disease by becoming more present focused and engaging in greater levels of risky behavior than they would in control conditions. Results will provide new insights into the relationship between health and decision making. Further, this research will offer novel insights into factors that contribute to variations in behavioral patterns that characterize different life history pathways.

Life History Theory

Life history theory is an evolutionary biological framework that is used to predict how and when organisms will allocate effort to the differing demands of survival and reproduction that occur across the lifespan (Charnov, 1993; Kaplan & Gangestad, 2005;

Roff, 1992; Stearns, 1992). Because energy is inherently limited, an organism must make a chain of decisions about how to allocate energy between the competing areas of somatic growth, maintenance, reproduction, and parental care. For example, if one is ill he or she must devote energy to immune system function and bodily maintenance, rendering oneself less able to direct energy to mate attraction or to caring for an infant. Because investment in any one of these categories necessarily comes at the expense of investing those resources elsewhere, life history theory highlights that organisms should 'choose' to partition their limited energy budgets in ways that best promote fitness in their local ecology (Ellis, Figueredo, Brumbach, & Schlomer, 2009; Kaplan & Gangestad, 2005).

Each choice an organism makes about how to allocate its energy narrows the number of potential directions that the organism's future decisions can take across development. For example, investing energy in immune function to fight disease during early childhood comes at the cost of expending energy on somatic growth. Making this tradeoff decreases the availability of somatic capital to resist the chronic diseases of adulthood (Gluckman, Hanson, & Beedle, 2007; McDade, 2005; Rickards, Frankenhuis, & Nettle, 2014). Thus, an adult raised in a pathogen rich environment must make choices in how to allocate energy in ways that account for the resulting limitations in bodily resources. The decisions an individual makes during development group together to form a general strategy for allocating tradeoffs across the lifespan: one's *life history strategy* (Del Giudice, Ellis, & Shirtcliff, 2011; Ellis et al., 2009).

Organisms' life history strategies fall on a continuum of fast to slow. Those adopting a faster strategy tend to make trade-offs that prioritize mating effort. They mature relatively rapidly, begin reproducing early, and produce a relatively large number of offspring, in

whom they invest minimal parental effort. Those adopting a slower strategy, on the other hand, make energy allocation decisions that prioritize investment in prolonged somatic growth. They mature more slowly, have a later onset of reproduction, and have fewer offspring in whom they invest very heavily. Although originally developed as a framework for understanding differences between species, life history strategies also vary within species. For example, although humans tend to fall on the slower end of the life history spectrum when compared to other species, individual humans calibrate their strategies based on features in their local ecologies, producing some variation (Belsky, Steinberg, & Draper, 1991; Chisholm, 1993; Del Giudice et al., 2009; Ellis et al., 2009; Kaplan & Gangestad, 2005).

What ecological factors influence how individuals calibrate their life history strategies (LH)? Research indicates that human LH strategies are attuned to the degree of harshness and unpredictability in the local ecology (Daly & Wilson, 2005; Ellis et al., 2009; Simpson, Griskevicius, Kuo, Sung, & Collins, 2012; Stearns, 2000). Environments are considered harsh and unpredictable if they are rapidly changing or are characterized by factors that increase the risk of morbidity/mortality, such as resource scarcity, a high pathogen load, or poor maternal care (Kaplan & Gangestad, 2005). These environments favor the development of a faster life history strategy because the likelihood of surviving into adulthood is diminished (Del Giuduce, 2009; Ellis et al., 2009; Kaplan & Gangestad, 2005). For example, research finds that people living in violent, inner-city neighborhoods tend to reproduce earlier than individuals living in safer neighborhoods (Daly & Wilson, 2005; Wilson & Daily, 1997). Faster strategies are favored in these contexts because they minimize the likelihood that one will perish before having a chance to reproduce. Environments that

are relatively benign, on the other hand, tend to promote the development of slower strategies (Del Giuduce, 2009; Ellis et al., 2009; Kaplan & Gangestad, 2005). In these cases, it is adaptive to delay reproduction and allocate more resources to somatic growth, bodily robustness, and the development of skills and knowledge that will make one better able to compete for resources in adulthood. This strategy promotes fitness by giving one a highly competitive phenotype in a benign adult environment, where long term survival is more certain.

In humans, the quality of one's early childhood environment plays an important role in calibrating life history strategies in adulthood (Belsky et al., 1991; Del Giudice et al., 2011; Ellis et al, 2009; Gluckman, Hanson, Cooper, & Thornburg, 2008; Kuzawa & Quinn, 2009; McDade, 2003; Rickards et al., 2014). Individuals from harsh early life environments, for example, show greater levels of oxidative stress (Griskevicius, 2013; Rickards et al., 2014), greater telomere degradation (i.e. more rapid aging; Rickards et al., 2014), and are more vulnerable to ecological stressors present in adulthood than are those from more benign early life environments (Belsky, & Pluess, 2009; Boyce, & Ellis, 2005; Ellis, Jackson, & Boyce, 2006; Mittal & Griskevicius, 2014). Accordingly, individuals from stressful environments have higher early adult mortality rates than those raised in benign environments (Doblhammer, & Vaupel, 2001; Flinn, 2006; Flinn, Nepomnaschy, Muehlenbein, & Ponzi, 2011; Gluckman et al., 2008) – effects that persist across levels of socioeconomic status achieved in adulthood (Hertzman, 1999; Kittleson et al., 2006). Given their greater vulnerability, those raised in harsh early life environments show heightened stress reactivity to ecological stressors encountered in adulthood (Belsky, 2008; Del Giuduce, 2009) and respond to such cues by exhibiting psychological and behavioral changes that

would promote earlier reproduction (Griskevicius, Delton, et al., 2011, Griskevicius, Tybur, et al., 2011; Hill, Rodeheffer, Delpriore, & Butterfield, 2013; Hill, DelPriore, Rodeheffer, & Butterfield, 2014). Indeed, both correlational and experimental research find that those sensitized to a faster strategy react to extrinsic stress by indicating (and pursuing) a younger desired age of first reproduction (Griskevicius, Delton, et al., 2011; Simpson et al., 2012), displaying more present focused financial decision making (Griskevicius, Tybur, et al., 2011; Griskevicius et al., 2013; White, Li, Griskevicius, Neuberg, & Kenrick, 2013), and preferring a heavier, more fertile female body ideal (Hill et al., 2014).

Vulnerability to Disease and Life History Strategies

To date, experimental research examining the impact of ecological stressors on the expression of life history strategies has focused almost exclusively on external, extrinsic mortality threats. An extrinsic threat is one that cannot be minimized through greater investment in one's own bodily resources (Stearns, 2000). For example, the high adult mortality rate seen in neighborhoods characterized by high rates of gang violence can be thought of as an extrinsic threat because increased investment in bodily robustness cannot itself decrease one's survivability in response to this type of threat (Ellis et al., 2009; Wilson & Daly, 1997). However, for humans, the risk of infectious disease – which is an intrinsic threat – has posed an even more substantial threat to human survivability than has the extrinsic threat posed by other dangerous humans. In the 1300s, for example, the plague killed up to 50% of the populations of Europe, Asia, and Africa (Gottfried, 2010). In the 1500s, European travelers to the Americas carried along diseases such as smallpox, measles, and typhoid fever, which killed over 75% of the population of Mexico (Acuna-Soto, Stahle, Cleaveland, & Therrell, 2002). Indeed, the World Health Organization estimates that

currently no fewer than 15 million humans die per year from infectious diseases, including influenza, tuberculosis, and AIDS (WHO, 2004). Biologists estimate that the threat of infectious disease has therefore played a critically important role in human evolution (Morens, Folkers, & Fauci, 2004).

Given that the threat of disease in one's environment has tremendous implications for one's expectations about survivability into adulthood, it is likely that these cues have implications for one's life history strategies. However, as with extrinsic threats, the way that people respond to intrinsic threats such as those posed by a growing risk of disease should differ depending on their developmental history. Exposure to environmental stress in the uterine and early life environments produces long-term, detrimental effects on immune system activity and immune-related hypothalamic pituitary axis (HPA) function (Kuzawa, 2005; Kuzawa & Quinn, 2009; McDade 2003, 2005; Miller, Chen, et al., 2009; Williams & Nesse, 1991). Although these physiological changes function to help combat pathogens and promote survival during childhood, they ultimately reduce immune system quality in adulthood (Miller, Chen, et al., 2009). Individuals with a history of low quality immune function would therefore generally benefit from being cautious and allocating their efforts toward somatic maintenance in adulthood (Rickards, 2014; Stearns, 1992; 2000; Waynforth, 2012). For example, if a person has a history of frequent illness, he or she can generally decrease his or her risk of illness by allocating effort toward immune function and maintenance and away from reproductive effort.

In the face of a rapidly increasing pathogen load, however, where one's survivability into adulthood becomes less certain, individuals with a history of low immune reactivity should shift their energy allocation toward decisions that promote more immediate

reproduction. Such a shift – although potentially increasing one's risk of infection in the short term – would be favored by selection because it would minimize the likelihood that an individual would die without having first had the chance to reproduce. Individuals with more robust immune systems, on the other hand, who have the bodily resources necessary to handle the threat of a rising pathogen load can best promote fitness in this context by allocating energy inward towards somatic maintenance. The way that individuals respond to a pathogen threat in their local ecology should therefore differ in predictable ways based on their developmental history and their immune functionality.

The Current Research

In the following, I present the results of two studies that examined the impact of vulnerability to illness on individuals' decision-making. Specifically, I predicted that exposing individuals to cues indicating a growing threat of disease in the environment would lead those with weaker immune systems to value present, immediate rewards (Study 1) and to be more willing to take financial risks (Study 2) compared to those in the control condition. I also explored possible mediators to this relationship (Study 2). Specifically, I examined whether the predicted effects would be mediated by changes in one's Health Locus of Control (Wallston, Wallston, & DeVellis, 1978) and one's uncertainty about their future health (Hill, Ross, & Low, 1997).

Study 1

The goal of Study 1 was to examine how disease threat impacts individuals' temporal preference (i.e. desire for immediate versus future rewards), based on individuals' vulnerability to illnesses. For individuals who are highly vulnerable to illnesses (i.e. those in poor health), the threat of disease heightens the risk of future mortality and thus, the

uncertainty of one's future environment (Hill, Prokosch, & DelPriore, under review). Therefore, we should expect to see that these individuals will react to disease threat by becoming more present focused: a signal consistent with a faster life history strategy (Griskevicius, Tybur, et al., 2011; Griskevicius et al., 2013). By hastening mating, it increases the chances of reproducing successfully before possibly succumbing to disease in the future. To this end, I hypothesized that individuals who are highly vulnerable to illness would react to a disease threat prime by picking more immediate rewards versus delayed rewards on a temporal preference task (Griskevicius et al., 2013).

Method

Participants

A priori power analyses were conducted using G Power software (version 3.1; Faul, Erdfelder, Buchner, & Lang, 2009; Faul, Erdfelder, Lang, & Buchner, 2007), basing estimates on the small to medium effect sizes typically found in disease threat related research (.10-.14; Terrizzi, Shook, & McDaniel, 2013). Using Cohen's recommendations (1992) as a guide, and basing my calculations upon an alpha of .05, and power at .80, I determined that least 100 participants would be needed. As such, One hundred and eighteen TCU undergraduates (87 female) aged 18 to 26 (M = 19.23, SD = 1.36; 58 in the disease condition, 60 in the control condition) participated in this study in exchange for partial course credit.

Procedures

Participants were seated at individually partitioned computers in a group experiment room. All survey measures were distributed using Qualtrics Research Suite (Qualtrics, 2013). Participants were informed that they were participating in a study about how current body

state impacts mood, memory, and judgment. Participants viewed (randomly assigned) either a sickness or a failure prime as part of an ostensibly related memory task at the beginning of the study. Participants then completed a task designed to assess participants' temporal reward preferences and a self-report measure of participants' abilities to delay gratification. Participants next answered measures about their personal health, and their family health, followed by a standard set of demographic questions. Once finished, participants were debriefed, given credit, and dismissed.

Materials

Priming manipulation. In order to manipulate disease threat perceptions, participants viewed one of two primes. The experimental prime was a slideshow portraying the increasing level of infectious diseases in America (Hill et al., under review). Past studies have reliably used disease slideshows as a manipulation to cause functional changes in behavior (Miller, & Maner, 2011, 2012; Mortensen, Vaughn Becker, Ackerman, Neuberg, & Kenrick, 2010; Schaller, Miller, Gervais, Yager, & Chen, 2010). But, these slideshow manipulations have primarily used images that evoke immediate, disgust-related, disease threat. For example, slides in one manipulation used pictures of people covered in contagious sores (Schaller et al., 2010).

Because I was primarily interested in cues of pathogen load rather than immediate disease threat, participants were shown a slideshow designed to prime the threat of a rising disease threat without evoking a visceral disgust reaction (Hill et al., under review). In a separate study, 80 female participants (26 in the disease threat condition, 27 for each of the other slideshows) pre-rated the disease slideshow. There was no significant difference for the amount of disgust elicited by the disease threat slideshow (M = 3.52, SD = 1.53) than by the

other two control slideshows (*M*s = 3.29, 3.78, *SD*s = 1.88, 1.40), $F(2, 79) = .64$, $p = .53$. However, the disease threat slideshow did lead participants to believe that: infectious disease is everywhere these days ($M_{disease} = 5.00$, $SD = 1.36$; $M_{scarcity} = 3.30$, $SD = 1.64$; $M_{failure} = 3.37$, $SD = 1.36$; $F(2, 77) = 9.90$, $p < .001$, $n^2_p = .03$), disease is a greater problem than it used to be ($M_{disease} = 4.38$, $SD = 2.00$; $M_{scarcity} = 3.30$, $SD = 1.56$; $M_{failure} = 3.33$, $SD = 1.52$; $F(2, 77) = 3.46$, $p = .04$, $n^2_p = .01$), they are more likely to get sick that year ($M_{disease} = 4.65$, $SD = 1.67$; $M_{scarcity} = 3.15$, $SD = 1.59$; $M_{failure} = 3.26$, $SD = 1.75$; $F(2, 77) = 3.22$, $p = .002$, $n^2_p = .02$), and they are more likely to catch a serious illness in their lifetime ($M_{disease} = 3.38$, $SD = 1.84$; $M_{scarcity} = 2.52$, $SD = 1.51$; $M_{failure} = 2.30$, $SD = 1.75$; $F(2, 77) = 3.22$, $p = .05$, $n^2_p = .02$) relative to the other slideshows presented. The control slideshow used in Study 1 featured a story about how college education is getting tougher in Texas, and the likelihood of failure is rising (Hill, Rodeheffer, Griskevicius, Durante, & White, 2012).

Temporal preference measures. Previous research has used measures of delayed discounting as a proxy for the tradeoff between mating effort versus somatic effort (Griskevicius et al., 2013). Discounting the future serves as a proxy measure for engaging in a faster life history strategy because shifting to a preference for immediate reward shows a prioritization towards gaining resources for use in the present, in response to a vulnerable individual perceiving a decreased shadow of the future in face of threat (Belsky, 2008; Daly & Wilson, 2005; Griskevicius et al., 2013; Griskevicius, Tybur, et al., 2011).

To measure present versus future focus in decision making (and indirectly, mating versus somatic effort), participants made series of rapid financial choices (Green & Myerson, 2004; Griskevicius et al. 2013; Kirby & Maracovic, 1996; Wilson, & Daly, 2004). Participants were instructed to complete the questions quickly, without spending too much

time on any one, in order to assess split second decision making (Griskevicius, Tybur, et al., 2011). As further precaution, questions were presented in a randomized order to prevent any ordering or anchoring effects. Most research directly comparing hypothetical versus real rewards has not found any significant differences in rate of discounting (Baker, Johnson, & Bickel, 2003; Madden, Begotka, Raiff, & Kastern, 2003; Green, & Myerson, 2004), thus, participants were not paid any real money for this task. During the task, participants chose between receiving a specified amount of money on the day after the experiment and receiving a larger amount of money 33 days after the experiment (for example: "Do you want to get $30 tomorrow or get $41 33 days from now?"). The monetary amounts varied systematically from $9 to $86 for the immediate reward, and from $47 to $99 for the delayed reward options. The dependent variable was measured as the sum of the immediate reward options a participant selected out of the 20 questions (range = 0-20, α =.87).

Additional measures of impulsivity. Research suggests that there are multiple, distinct components of impulsivity (Copping, Campbell, & Muncer, 2013, 2014). Accordingly, participants filled out a secondary measure in addition to the delayed discounting task. Participants completed the 10 item Delaying Gratification Inventory (DGI; Hoerger, Quirk, & Weed, 2011) as another measurement of impulsive versus planned decision making. The DGI measures one's ability to delay reward, asking questions like "I have always felt like my hard work would pay off in the end." It measures ability to delay gratification across physical, social, monetary, and status related domains on a Likert scale of 1-5 (1 = *completely disagree*, 5 = *completely agree*). Appropriate items were reverse scored

and a mean composite was calculated (α = .56)[1], with higher scores indicating a greater ability to delay gratification. So, someone who is more impulsive would have a lower DGI score. Mean DGI score was used as a dependent variable in analyses.

History of illness and vulnerability to disease. Next, participants filled out a series of questions that assessed health history and immune reactivity. This measure included items measuring history of childhood illness (Hill et al., under review). This set included questions like, "When I was growing up, I missed a lot of school due to illness." I also included questions assessing overall health during the last year adapted from the 12 item Short Form Health Survey (Ware, Kosinski, & Keller, 1996). These items measured the frequency with which poor health affected their day-to-day lives over the past year (e.g. "How frequently did health problems interfere with your daily activities"). Finally, I included items that measure participants' familial health (e.g. "People in my family are generally healthy"). To get an overall measure of adult health quality and genetic resilience to disease, these measures were all transformed into z-scores and aggregated into a mean composite of vulnerability to illnesses (α = .85), with higher scores indicating greater vulnerability. This composite was included as moderating variable in analyses.

[1] Although the DGI is a published scale with reliable validity (Hoerger et al., 2011), I found in Study 1, that it had below acceptable levels of reliability in our sample (α = .56). Reliability analyses revealed three questions that did not load well with the other questions (e.g. "I would be willing to give up physical pleasure or comfort to reach my goals") I formed a subsequent composite variable without these questions included that yielded a more acceptable reliability (α = .66). I next conducted multiple regression analyses using this higher reliability composite. The analyses revealed results nearly identical to those obtained using the original, full composite variable. Namely, there was a significant interaction between priming condition and sickness history on ability to delay gratification b = .33 (SE = .13), $t(114) = 2.48$, p = .01, semipartial r^2 = .05, (CI: .07, .59). Simple slope tests and regions of significance tests also revealed similar results to those reported for the full composite variable.

Results

To exclude the possibility that priming condition caused a change in participants' perceived vulnerability to illnesses, I conducted a univariate ANOVA with vulnerability to illnesses (indexed by the composite measure of health described above) as the dependent variable and priming condition (disease threat vs. academic failure) as the grouping variable. The ANOVA did not reveal an effect of priming condition on participants' vulnerability to illnesses (p = .38). Therefore, I used the composite measure of perceived vulnerability to illnesses as a moderator in both of my subsequent models.

Temporal preference

I next examined the impact of disease threat cues and perceived vulnerability to illnesses on temporal preference using multiple regression. Condition (dummy coded), vulnerability to illnesses (centered), and their interaction were entered as predictors of temporal preference (more present versus more future focus). In addition to significance testing, I used Hayes' Process Model (2013) during all procedures to perform bootstrapping (1000 samples) and generate confidence intervals (set at 95 percent certainty). Results revealed a significant interaction between priming condition and vulnerability to illnesses (see *Table 1* for descriptive statistics), b = -3.50 (SE = 1.22), $t(114)$ = -2.87, p = .005, semipartial r^2 = .07, (CI: -5.91, -1.09).

Table 1. Descriptive Statistics for Study 1.

	Priming Condition			
	Control		Disease	
	M	SD	M	SD
Adult Sickness	2.63	0.69	2.51	0.60
Family Sickness	2.66	1.22	2.59	1.09
Preference for Immediate Rewards	9.07	4.27	9.31	3.89
Ability to Delay Gratification	3.93	0.41	3.86	0.41

I next probed the interaction by conducting simple slope and regions of significance testing (Aiken & West, 1991). Simple slope tests revealed that, for those in the disease threat condition, higher vulnerability to illness predicted a greater preference for immediate reward, b = 2.21 (*SE* = .91), $t(114) = 2.43$, $p = .02$, semipartial $r^2 = .05$, (CI: .41, 4.02). There was not a significant influence of vulnerability to illness upon reward preference for those in the control condition ($p = .11$). I also examined the impact of priming condition on those who were relatively high or low in vulnerability to illness by conducting regions of significance tests (at 1 *SD* above and below the mean). As predicted, results revealed that those who were most vulnerable to illness (1 *SD* above mean vulnerability) showed a greater preference for immediate rewards relative to participants in the academic failure condition (see *Figure* 1), b = -2.44 (*SE* = 1.05), $t(114) = -2.31$, $p = .02$, semipartial $r^2 = .04$, (CI: -4.52, -.35). Participants low in vulnerability to illnesses (1 *SD* below the mean) preferred marginally more delayed reward in the disease threat condition relative to the academic failure condition (b = 1.84, $p = .08$).

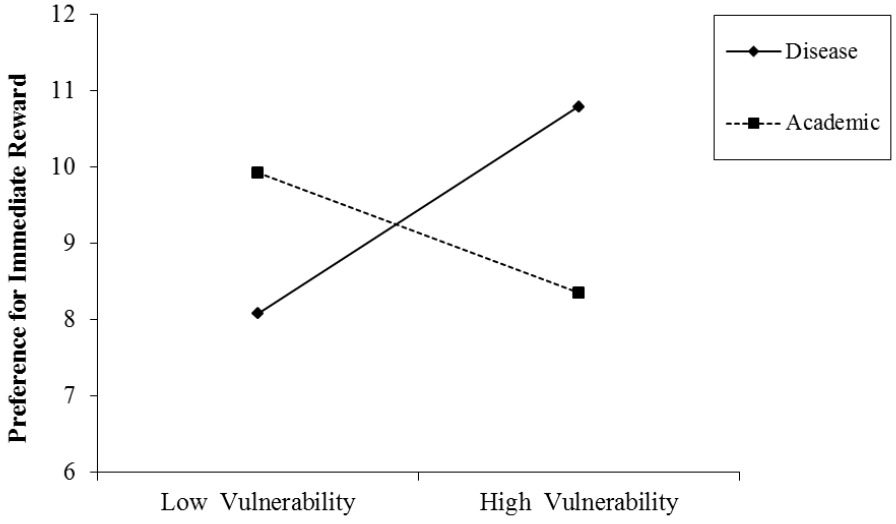

Figure 1. Effect of priming condition by vulnerability to illness on temporal reward preference. Higher scores indicate greater preference for immediate reward.

Ability to delay gratification

Next, I examined the effect of condition and vulnerability to illnesses on participants' ability to delay gratification (DGI score) using multiple regression. Condition (dummy coded), vulnerability to illnesses (centered), and their interaction were entered as predictors of ability to delay gratification (mean composite DGI score). Results revealed a significant interaction between condition and vulnerability to illnesses, b = .25 (SE = .12), t(114) = 2.04, p = .04, semipartial r^2 = .03, (CI: .01, .50). Simple slope tests revealed that in the disease condition, those with higher vulnerability to illnesses reported experiencing more difficulty delaying gratification than those with lower vulnerability to illnesses, b = -.22 (SE = .09), t(114) = -2.36, p = .02, semipartial r^2 = .05, (CI: -.40, -.03). There was no effect of vulnerability to illnesses for participants in the control condition (p = .68). I also probed the interaction by examining the impact of priming condition on those who were relatively high or low in vulnerability (1 *SD* above and below the mean). Highly vulnerable individuals who viewed the disease threat prime reported greater difficulty delaying gratification relative to those in the academic failure condition, b = .24 (SE = .11), t(114) = 2.22, p = .03, semipartial r^2 = .04, (CI: .03, .45) (see *Figure 2*). There was no effect of priming condition for those low in vulnerability to illnesses (p = .51).

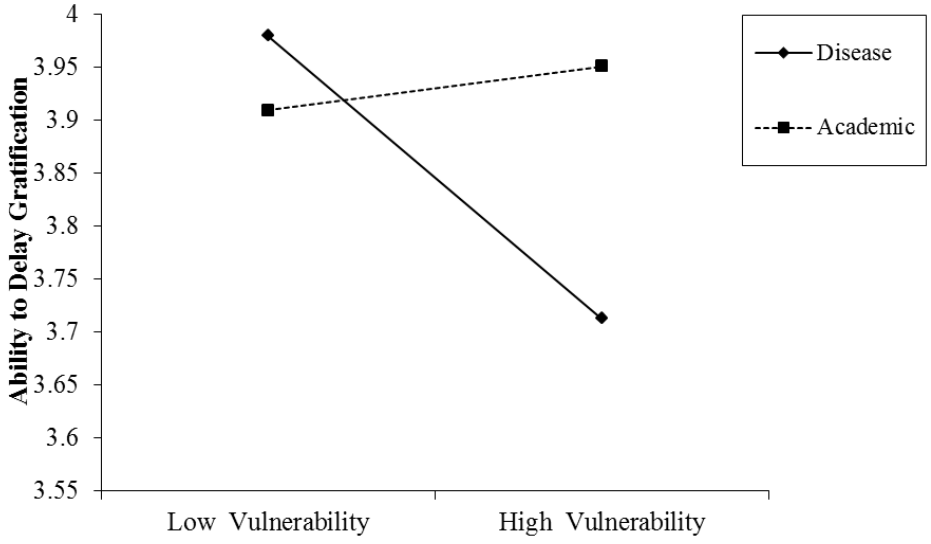

Figure 2. Effect of priming condition by vulnerability to illnesses on ability to delay gratification. Higher scores indicate greater ability to delay gratification.

Discussion

As predicted, cues of growing disease threat influenced participants' temporal focus. Specifically, I found that individuals who were most vulnerable to illnesses (due to extensive adulthood and family history of poor health) responded to the threat of growing disease prevalence by shifting their decision making towards a preference for immediate gratification. Vulnerable individuals chose the quicker reward, even though making this choice came at the cost of getting an absolutely larger reward. They also reported greater difficulty delaying gratification in order to reap longer-term benefits. No such priming effect was shown for vulnerable individuals in the control condition or for healthy individuals in either priming condition.

These shifts in temporal focus are consistent with the hypothesis that when the shadow of the future is made shorter by a relevant ecological threat, such as heightened pathogen load, vulnerable individuals shift to desiring present rewards as a way to promote immediate survival and mating. When the odds of future survival are uncertain, it is not adaptive to waste time and energy holding out for later rewards that one might not live long enough to reap into fitness benefits.

Study 2

Study 1 measured the effects of disease threat and vulnerability to illnesses (as indexed by health history) on impulsive decision making by measuring temporal discounting, and reported ability to delay gratification. Shifts in these two markers are indicative that vulnerable individuals may speed up their life history strategy by prioritizing mating effort in face of an uncertain, harsh future. Study 2 was designed to examine the effects of disease threat and vulnerability to illness on another studied behavioral marker of a faster life history strategy: risk taking (Copping et al., 2014; Griskevicius et al., 2013). I predicted that a disease threat would lead vulnerable individuals to take more risks than they would in the control condition. Taking more financial risks, although a gamble, can lead to the rapid accrual of resources needed to successfully reproduce (Griskevicius, Tybur et al., 2011). Further,study 2 also included a set of possible mediators to help further elucidate possible mediating mechanisms to the relationship between disease threat, personal vulnerability, and decision making.

One such mediating mechanism might be a shift in individuals' perceptions of their future lifespan following the threat of disease. For example, Hill and colleagues (1997) found that individuals who found the future to be more uncertain, and who predicted they would

have a shorter lifespan took more risks on a risk taking task than those with more stable, certain futures. For Study 2 of the current research, I predict that disease threat might lead people who are highly vulnerable to illness to perceive their likelihood of getting a major disease as higher than someone with a healthy history, and therefore, that their future is more uncertain. The prospect of an uncertain future (and shortened longevity due to disease) should lead these individuals to speed up their life history strategy and take the risks needed to secure resources for mating while they still can. The prime should not affect healthier people's perceptions of their future. These individuals have enough resources to adequately devote towards somatic needs (such as buckling down to fight an infection) as an effective self-preservation strategy. Thus, I do not predict that disease threat should not produce any significant effect on robust individuals' perceptions of the future, or their subsequent risk-taking.

Another possible mediator between disease threat, vulnerability and risk taking is the sense of control that one feels that they have over their health outcomes (Wallston et al., 1978). Recent research has linked low childhood SES to a diminished sense of control (Mittal & Griskevicius, 2014), which in turn leads to increased impulsivity and risk taking. Extending this logic, it is possible that people who are in poor health (and thus, vulnerable to illnesses) should feel less in control of their health than individuals who have more robust bodies. Alternately, viewing a disease prime could lead people who are vulnerable to illness to attribute their health to chance, fate, or the actions of others (scale scores would indicate a more external health locus of control). I predicted that these increased external attributions of health control following disease prime, should lead people who are vulnerable to illness to engage in more risk taking behaviors. On the other hand, someone with an internal locus of

control should believe that through his or her own actions, they can keep themself from getting sick. These individuals should therefore feel less vulnerable to future disease than someone with poorer immune function. I predicted that a disease prime should not alter healthy individuals' sense of control over their health fate. In turn, it is possible that these internally focused people might even show a shift to a slower strategy when faced with focused disease threat, as playing it safe and taking care of themselves can be effective in helping to survive a period of heightened pathogen load.

Method

Participants

One hundred and five TCU undergraduates (67 female) aged 18 to 43 (M = 20.10, SD = 2.96; 50 in the disease condition, 55 in the control) participated in this study in exchange for partial course credit.

Procedure

Participants completed Study 2 using the same experimental room and computers as Study 1. In addition, Inquisit experiment-building software version 4.0.3 (Millisecond Software, 2013) was used to run the behavioral measure of risk taking. As in Study 1, participants viewed either the slideshow about rising disease threat in America, or the control slideshow about rising levels of academic failure. Participants next completed a Balloon Analog Risk Task (BART: Lejuez et al., 2002) on the computer, as a behavioral measure of risk taking. Next, participants filled out two potential mediator scales (administered immediately after the risk taking dependent variables) that could potentially mediate the relationship between priming condition, childhood illness, and risk taking. Participants filled out the same health assessments and demographic measures that were administered in Study

1. Once finished, an experimenter debriefed the participants, paid them their final earnings from the BART, assigned credit, and dismissed the session.

BART. The BART was chosen as a proxy for risk taking in this study because it is a widely used risk task in both clinical and social research, that demonstrates good test retest reliability (r = .77 ; White, Lejuez, & de Wit, 2008). The BART task consists of a series of balloons that a participant must pump up to earn money. Each pump earns a small, set increment of money. However, the balloon may pop at any given time, and the participant is unaware of when it will do so. The participant earns as much money as he or she pumps into a balloon, as long as he or she submits his or her earnings before the balloon pops. Therefore, the person must balance the chance to earn more money with the possibility that the balloon will pop and he or she gains nothing. Thus, greater persistence at pumping up a balloon represents a riskier strategy (that the participant is favoring the greater risk of the bigger, riskier payoff over the smaller, safer bet).

To realistically mimic real-life risk-taking behavior, participants were notified before the start of the task that they would be paid the final remaining balance from this task (Lejuez et al., 2002).Participants engaged in 10 balloon blowing trials (for reliability data, see Wallsten, Pleskac, & Lejuez, 2005), where each pump was worth .02 cents (for a discussion on the effects of varying incentive amounts see Dahne, Richards, Ernst, MacPherson, & Lejuez, 2013; Lejuez, & Mathias, 2013) and each balloon had a moderate, 1-64 breakpoint (see Lejuez et al., 2002). Risky behavior was coded in two different ways (Griskevicius et al., 2013). First, total number of pumps was recorded as a dependent variable (M = 181.60, SD = 72.98), with a larger number of pumps indicating more risk taking. Second, total number of balloons popped out of the 10 trials was recorded (range = 0-9, M = 3.90, SD =

1.85). Here, a higher number of balloons popped indicating that participants made the tradeoff towards taking a risky "go big or go home" strategy relative to a safer "small but safe" strategy during trials. Because the two measures were moderately to highly correlated (r = .58, p < .001), I standardized and averaged scores of the two measures to form a single measure of riskiness (α = .73), consistent with past coding of BART results (Griskevicius et al., 2013).

Potential mediators. Participants next completed the Multidimensional Health Locus of Control (Wallston et al., 1978), which measures the amount of control someone believes to have over his or her own health outcomes, as a potential mediator (e.g. "I am in control of my health." 1 = *strongly disagree*, 7 = *strongly agree*). Participant's answers were recoded and summed into a composite, with higher scores indicating a greater sense of personal control over health outcomes (α = .74)

As an additional mediator, participants then completed the Future Lifespan Assessment (Hill et al., 1997), which asks participants to estimate the likelihood of a variety of outcomes, (e.g. financial stability, death) happening to them at each decade in life (e.g. "how likely is it that you will have suffered a major disease by this point in your life?"; 1 = *very unlikely*, 7 = *very likely*). I modified this scale to include several measures of major disease and health, which I used to form composites where higher scores indicated a greater predicted chance of an outcome happening during the lifespan (αs ≥ .89).

Results

As in Study 1, I ran a univariate ANOVA with vulnerability to illness entered as the dependent variable and priming condition (disease threat vs. academic failure) as the grouping variable, to ensure that there was no effect of priming condition on reported

vulnerability. Results revealed that there was no effect of priming condition on participants' reported vulnerability (p = .70). Therefore, I used the composite measure of vulnerability to illness as a moderator in subsequent models.

Risk taking on the BART

Next, I examined the interaction (c path) between disease threat and vulnerability to illnesses on risk taking using multiple regression. In two separate models, condition (dummy coded), vulnerability to illnesses (centered), and their interaction were entered as predictors of BART performance (using the z-score composite of risky behavior). I again used Process (Hayes, 2013) during all analyses to generate 95% confidence intervals (using 1000 bootstrapped samples). Results of the regression revealed a significant interaction of condition and vulnerability to illnesses on risky behavior during the BART (see *Table 2* for descriptive statistics by condition), b = -.65 (SE = .28), t(101) = -2.36, p = .02, semipartial r^2 = .05, (CI: -1.20, -.10) (see *Figure 3*).

Table 2. Descriptive Statistics for Study 2.

	Prime Condition			
	Control		Disease	
	M	SD	M	SD
Adult Sickness	2.43	0.66	2.46	0.74
Family Sickness	2.56	1.12	2.68	0.98
Total Pumps on BART Task	181.20	80.00	182.04	65.21
Total Number of Balloons Popped	3.76	1.90	4.06	1.80

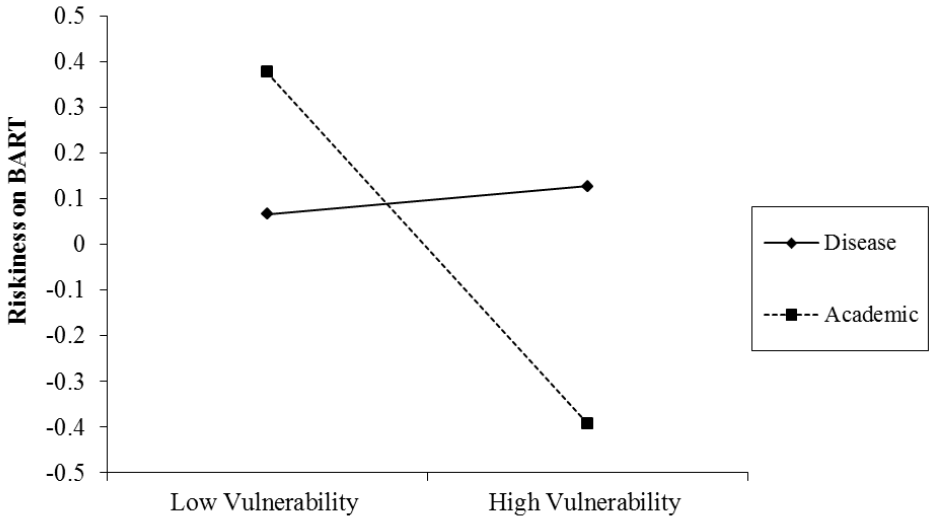

Figure 3. Effect of priming condition by vulnerability to illnesses on risk taking during a BART task. Higher scores indicate riskier behavior.

Subsequently, I used simple slope tests and regions of significance tests (Aiken & West, 1991) to unpack this interaction. Simple slope tests revealed that in the control condition, highly vulnerable individuals took significantly less risks than their healthier counterparts, b = -.60 (SE = .19), $t(101)$ = -3.11, p = .002, semipartial r^2 = .09, (CI: -.99, -.21). However, there were no significant differences between high and low sickness-vulnerable individuals in the disease threat condition, p = .81. Further analysis analyzing the effects of condition at relatively high and low levels of sickness vulnerability (1 *SD* above and below the mean) revealed that those who were highly vulnerable to illness in the disease threat condition made significantly more pumps than vulnerable individuals in the control condition, b = -.52 (SE = .25), $t(101)$ = -2.09, p = .04, semipartial r^2 = .04, (CI: -1.01, -.03). There was no difference in the riskiness of healthy individuals (those 1 *SD* below mean sickness vulnerability) between conditions (p = .21).

Mediators

To examine the role of my proposed mediators, a mediated moderation model was examined using the Hayes' Process Model macro for SPSS (2013). Condition (dummy coded), vulnerability to illness (centered), and their interaction were entered as direct predictors of BART risk taking. The Future Lifespan Assessment and Multidimensional Health Locus of Control scales were be entered in separate models as mediators between condition, vulnerability to illnesses, and risk taking.

Future Lifespan Assessment. Results examining the role of Future Life Assessment found no interactions of condition by vulnerability to illnesses on future assessments of grave illness or health (*p*s ≥ .18). However, there was a main effect of vulnerability to illnesses, with participants who were most vulnerable to illness reporting a higher expectation to become seriously ill during their lifespan, $b = .84$ ($SE = .24$), $t(101) = 3.53$, $p = .006$, and trending lower expectations to have good health across their lifespan, $b = -.34$ ($SE = .20$), $t(101) = -1.68$, $p = .10$. However, neither of these assessments mediated the relationship between condition, vulnerability to illness, and risk taking during the BART (*p*s ≥ .15).

Multidimensional Health Locus of Control. There were no significant effects of sickness or condition on the MHLoC scale as a dependent variable (*p*s ≥ .07), or as a mediator between condition, vulnerability to illness, and BART risk taking (*p*s ≥ .17).

Discussion

The results of Study 2 lend additional evidence to support the hypothesis that decision-making (especially that of vulnerable individuals) is functionally attuned to changes in environmental disease threat. Specifically, while Study 1 demonstrated that disease vulnerable individuals change the temporal focus of their decision making towards

immediate gratification. In Study 2, disease threat did not lead vulnerable individuals to take more risks than healthy individuals, contrary to my predictions. Rather, disease threat prompted vulnerable individuals to take more risks than they would under non-disease related threats, which erased differences in levels of risk taking between individuals of high and low illness vulnerability. Although Study 1 and Study 2 show different patterns of results, both sets of results demonstrate that vulnerable individuals react to heightened disease threat by speeding up their life history strategy. Disease threat leads those who are most vulnerable to catching an illness to shift their decision making in ways (such as taking more risks than they normally would) that promote immediate survival and resource acquisition needed to facilitate hastened reproduction in the face of a threat.

General Discussion

Past research has found that extrinsic environmental stressors lead people to behave differently based on their childhood environment (Griskevicius, Tybur, et al., 2011; Griskevicius et al., 2013; Hill et al., 2013; Mittal & Griskevicius, 2014). For example, heightened mortality threat leads people from harsher backgrounds to behave more impulsively, while people from wealthier backgrounds behave more cautiously (Griskevicius, et al., 2013). For a vulnerable individual (such as someone from a harsh socioeconomic background) such behaviors favor earlier reproduction and heightened genetic fitness in face of an uncertain future.

Across two experiments, I found evidence that disease threat can lead to divergent decision-making based on a more intrinsic factor: individuals' vulnerability to illness. Specifically, the experiments found that people who were most vulnerable to illness reacted to the threat of heightened morbidity by making impulsive, risky choices.

This shift towards less cautious, more impulsive behavior suggests that developmentally compromised immune quality is another cue that can lead individuals to pursue a faster life history strategy under times of uncertainty and duress.

Taken together, these studies add to a growing literature that demonstrates the impact of disease threat on shaping social cognitions (Duncan & Schaller, 2009; Duncan, Schaller, & Park, 2009; Hill et al., under review; Miller & Maner, 2011, 2012). My studies extend this work by providing a novel conceptual link between the threat of heightened morbidity/mortality caused by disease and decision making behaviors. These studies provide the first experimental evidence in humans that aspects of decision making like impulsivity and riskiness may be functionally plastic in response to ecological cues of pathogen load, such that vulnerable individuals engage in behaviors that might facilitate present gains and earlier opportunities to reproduce.

These results also add to a growing body of research in evolutionary developmental psychology suggesting that childhood conditions may interact with adulthood conditions to influence decision-making (Belsky et al., 1991; Ellis et al., 2009; Rickards et al., 2014). This extends recent experimental research that has demonstrated that various markers of early environmental harshness (like lower socioeconomic status) in early childhood influence how people respond to primed stressors in adulthood (Hill et al., 2014; Hill et al.,2013). The current experiments provide evidence that differences in one's developmental history with disease (indexed here by adulthood and family sickness history) can predict a pattern of temporal preference and risk taking under conditions of heightened pathogen load. This shift in present focused, risky decision making would have helped to facilitate fitness by encouraging quicker resource acquisition. This would help to guarantee that an

immunocompromised individual would have the resources needed to reproduce before possibly succumbing to illness. The present studies suggest that immune quality (based upon childhood history and forecasted threat) is an important factor to consider when examining adults' decision making behaviors.

Lastly, this research is important because it sheds light on how individual differences in sensitivity to disease threat may be a factor that predicts the types of decisions people make at crucial periods of their life. To be financially successful in current Western society, one must often make tradeoffs in early adulthood towards somatic investment (such as delaying starting a family to go to college and gain more financial earning potential). For example, those who are able to think long term and allocate money into retirement savings in early adulthood reap tens of thousands more dollars when they retire than those who choose to forego saving that money, in favor of immediate gratification (Financial Services Institute, 2012). As such, there is an abundance of research that looks at improving self-regulation (e.g. helping people to delay gratification more) as a way to improve favorable life outcomes (Vohs & Baumeister, 2011). While self-regulation research makes a valuable contribution to improving life outcomes in today's long-term focused culture, the current studies suggest that present focused, risky decision making is not necessarily a mal-adaptive trait. Rather, these shifts in behavior might be reflective of evolved adaptive strategies that increase fitness in harsh and unpredictable environments.

Limitations and Future Directions

One limiting factor of the current research is that the pattern of behavior participants exhibited is divergent between my two studies. In Study 1, disease threat produced different levels of present versus future focus in high versus low illness-vulnerable individuals; but in

Study 2, disease threat did not produce any differences in risk-taking across people of any illness vulnerability. Instead, high and low illness vulnerable individuals showed divergent risk taking in the control condition; those in poorer, more vulnerable health took *less* risks than their healthier counterparts. One such possibility for this mismatch in results is that temporal focus and risk taking are two distinct, psychological mechanisms. For example, the pattern of results in Study 2 may be better explained using principles of optimal foraging theory (see McNamara & Houston, 1992 for a review). Sickly individuals may be chronically more cautious because their weaker body makes it less profitable to be risky on a regular basis (since somatic capital is low). Healthy individuals can afford to take more risks in general. When disease threat is heightened, it may pay more in fitness benefits for sickly individuals to take the risks necessary to hasten mating- and this mating need is enough that these individuals take as many risks as healthier individuals normally do. Although temporal focus and risk taking have both been found to be reliable markers of the tradeoff people are making between somatic versus mating effort (Griskevicius et al., 2013), impulsivity has been poorly operationally defined in the literature- which can lead to distinct processes being combined when they should not be (Copping et al., 2013). The present studies may lend support to that notion. Future studies may want to investigate temporal preference and risk taking separately as measures of life history strategy.

Although the current studies are an important first step in determining the influence of immunocompetance on decision making processes, more work is needed to examine the role of different types of disease threat. This series of studies utilized a prime that heightened levels of forecasted disease threat. Would a more immediate disease threat (such as being near a contagious individual) produce similar results? Past research has shown that

immediate disease threat activates a prophylactic behavioral response (Miller & Maner, 2011, 2012). For example, individuals primed with immediate disease threat become more conservative and withdrawn towards other people (Mortenson et al., 2010), and less sexually risky (Murray, Jones, & Schaller, 2013; Tybur, Bryan, Magnan, & Hooper, 2011). The present studies found that forecasted disease threat led vulnerable individuals to act impulsively, while healthy individuals buckled down and played it safe. However, prophylactic concerns might lead people to become more conservative in their decision making as a way to facilitate temporary disease avoidance. It is possible that immediate disease threat might cause all individuals to focus on securing immediate rewards, and safe bets relative to larger gambles, regardless of their developmental history of disease. Or, the most disease vulnerable individuals might even show a greater switch towards making conservative, immediate decisions, compared to healthier individuals. Future research is needed to examine the effects of immediate disease threat- both primed (Mortenson et al., 2010; Murray et al., 2013; Neuberg, Kenrick, & Schaller, 2011) and unprimed (due to immunosuppression: Miller & Maner, 2011) - on decision making.

Another limitation of this project is that I did not ask participants to give a detailed medical history. Instead, I relied primarily on short, self-answer questionnaires to assess childhood, adulthood, and family sickness history. It is possible that because I used subjective measures, I neglected to examine a more direct link between actual childhood disease exposure and stress reactivity in adulthood. Instead, I primarily indexed peoples' perceptions of their immune system quality. It is possible that some participants might have better/worse immunocompetance than they reported they do, and that it was participants' perceptions of their immune quality (and not actual quality, per se) that drove the results seen

in these studies. More work needs to be done that attempts to replicate the results of these studies using physiological markers of immune system quality (e.g. mitogen stimulated cytokine proliferation, NK cell counts, flow cytometry panels of various immune cell types). This will help to elucidate the roles that actual immune quality, and self-perceptions of immune quality play in how people react to disease threats in adulthood.

Finally, while past research has suggested that sense of control acts as a mediating mechanism to bridge the relationship between childhood SES, ecological stress, and impulsive decision making (Mittal & Griskevicius, 2014), I did not find any effects of Health Locus of Control in my research. It is conceivable that similar to Mittal's findings (2014), it is perceptions of lack of general control (rather than health specific sense of control) that mediate the relationship between sickness history, disease threat, and impulsivity. Future research should include a non-health related Locus of Control scale to see if this is the case. There is also the potential that an entirely different mediating mechanism is at work when individuals are faced with intrinsic threats rather than the extrinsic threats previously studied by evolutionary researchers. More studies are needed to explore the possible mechanisms by which developmental history of sickness interacts with disease threat to produce impulsivity and risk taking.

Conclusion

Recent research has experimentally tested the impacts of early developmental conditions by ecological stress in adulthood on behavior. Fast strategists' preferences for earlier reproduction, and behaviors that facilitate earlier reproduction (e.g. impulsive decision making) have been well documented in research using extrinsic stressors (such as violent crime, and resource scarcity) as a manipulation (Griskevicius, Delton, et al., 2011;

Griskevicius, Tybur, et al., 2011; Griskevicius et al., 2013; Hill et al., 2013; Hill et al., 2014). In the current research, I demonstrated that stressors that are more intrinsic in nature may similarly affect vulnerable individuals' decision making processes. Specifically, disease threat prompted individuals with poor immunocompetance to favor both impulsive and risky choices. The current set of studies, provide some of the first evidence of the influence of immune quality and pathogen load upon decision-making in adulthood. In conclusion, vulnerability to disease (indexed by exposure history) may be a pertinent and largely unexplored factor that promotes a functional shift towards impulsive, risky choices.

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ABSTRACT

LIFE HISTORY AND PATHOGEN SUSCEPTIBILITY: EFFECTS ON RISK TAKING BEHAVIORS

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Life history theory predicts that cues in the environment that influence one's mortality risk should have important implications for decision-making. One factor that is known to impact one's mortality risk is the threat of disease, particularly among those most vulnerable to infection. Two studies examined the impact of the perceived disease load on people's preference for immediate versus delayed rewards and financial risk-taking. In both studies, participants were primed with cues indicating a growing disease threat or control cues. In Study 1, this was followed by a temporal discounting task and measures of one's ability to delay gratification. In Study 2, participants completed a risk taking behavioral measure. Results revealed that individuals with a history of health problems respond to cues of a growing disease threat by reporting more difficulty delaying gratification, valuing smaller, immediate rewards over larger, delayed rewards, and exhibiting a greater preference for risky rewards compared to controls. This research provides the first experimental evidence indicating that vulnerability to disease – both chronically occurring and experimentally manipulated - have implications for life history strategies and economic decision-making.