PRIMING OPTIMISM
THE AUTOMATICITY OF HEALTH RISK PERCEPTIONS

Sherine El-Toukhy

A dissertation submitted to the faculty of the University of North Carolina at Chapel Hill in partial fulfillment of the requirements for the degree of Doctor of Philosophy in the School of Journalism and Mass Communication.

Chapel Hill
2012

Approved by:

Jane D. Brown

Rajiv Rimal

Rhonda Gibson

Sri Kalynaraman

Tanya Chartrand

ABSTRACT

SHERINE EL-TOUKHY: Priming optimism: The automaticity of health risk perceptions
(Under the direction of Jane D. Brown)

Communication initiatives frequently are used to inform people about various risks in an attempt to improve their decision making and behavior. People, however, do not readily accept personal risk information, often underestimating their susceptibility to and the severity of the risks. This dissertation draws on psychological theory and research to explain why people underestimate their risks.

Three experiments were designed to test whether people have an automatic tendency to underestimate self-relevant risks - the default option that occurs without conscious awareness of underestimation of health risks as a bias and of self-schema activation as a source of this bias and is likely to occur when people are mentally taxed. Rationale for the theoretical propositions advanced here is derived from research documenting that health risk information is (a) processed as self-relevant, (b) inconsistent with the positivity bias and self-threatening, and that (c) the activation of the self-schema is automatic.

Experiment 1 tested the hypothesis that people underestimate their likelihood of experiencing negative health conditions and overestimate their likelihood of experiencing positive ones. Experiment 2 examined whether underestimation of health risks is an efficient process, one that occurs when people are mentally taxed. Finally, Experiment 3

tested the effects of health risk controllability and psychological reactance on health risk perceptions.

All three experiments relied on priming to activate the self-schema and measured the effects of this activation on reaction time, an implicit measure of risk perceptions. Several individual difference variables (e.g., self efficacy) and health risk characteristics (e.g., prevalence) that influence risk perceptions were controlled for.

Results show that people automatically underestimate their health risks, that is, without awareness, intention, or effort. The studies provided conclusive evidence of (a) underestimation of susceptibility to negative health conditions and overestimation of susceptibility to positive ones and (b) efficiency of underestimation of health risks. Results regarding self-schema activation as a source of underestimation of risks were not as conclusive.

Individual difference variables did not affect implicit measures of risk perceptions. Health risk characteristics, on the other hand, influenced risk perceptions. The experiments showed an inverted relationship between susceptibility and severity dimensions of health risk perceptions.

ACKNOWLEDGEMENTS

This dissertation marks the end of a long journey and the birth of another. Throughout this journey, it was only because of the people in my life that I got through rough times and rejoiced in happy ones. To them, I owe the completion of this work.

I am wholeheartedly grateful to my chair, Dr. Jane Brown, to whom I always turned for guidance throughout my tenure in the doctoral program. Her unwavering support, encouragement, and dedication were the bedrock of my successful completion of this dissertation.

I am indebted to Dr. Tanya Chartrand, Dr. Sri Kalyanaraman, Dr. Rhonda Gibson, and Dr. Rajiv Rimal, my committee members, whose suggestions were invaluable.

I have been honored to receive a William R. Kenan, Jr. University Fellowship from the Graduate School at the University of North Carolina at Chapel Hill, which allowed me to pursue a doctorate in health communication. This material is based upon work supported by the National Science Foundation under Grant No. (NSF 06-605).

Thanks are also due to faculty, staff, and my colleagues in the School of Journalism and Mass Communication who supported me in many ways.

Thousands of miles away, my family in Egypt believed in me and supported me beyond words. Words of gratitude are not enough to thank my parents, my husband, and my brothers. Finally, it is my son, Omar, to whom I owe the most. His unconditional love and patience with his busy mom have been the fuel that kept me going. To him I dedicate this work.

TABLE OF CONTENTS

LIST OF TABLES

Table

LIST OF FIGURES

Figure

CHAPTER 1

INTRODUCTION

"Are you greater than AIDS?" (Kaiser Family Foundation [KFF], 2010/11) and "This is your brain on drugs" (Partnership for Drug-Free America [PDFA], 1987) are examples of health messages that people often encounter in the media. Such messages are designed to inform people about personal health risks in an attempt to improve health decision making and promote positive behavioral change, the ultimate goals of health education and promotion efforts (Glanz, Rimer, & Viswanath, 2008). Common across these messages is a self-relevant health risk. Dual process theories of persuasion posit that self-relevance induces elaborate information processing, which results in stable and strong attitudes that predict behaviors (Brinol & Petty, 2006). A number of behavioral change theories posit that perceptions of susceptibility to a severe risk motivate people to adopt positive behaviors to reduce or eliminate the risk altogether (Ajzen, 1991; Becker, 1974; Rogers, 1975; Witte, 1992).

Studies have shown, however, that people do not readily accept personal risk information (Liberman & Chaiken, 1992) and most often underestimate their susceptibility to and severity of health risks (e.g., Bränström, Kristjansson, & Ullén, 2005 re: skin cancer). This dissertation draws on psychological theory and research to explain why people underestimate personal health risks. It posits that people have an automatic tendency to underestimate self-relevant risks. Rationale for the theoretical propositions advanced here is derived from research documenting that health risk information is (a)

processed as self-relevant, (b) inconsistent with the positivity bias and self-threatening, and that (c) the activation of the mental representation of the self (i.e., self-schema) is automatic, a process that occurs without awareness, intention, control, or much cognitive resources.

Risk perceptions reflect a subjective probability that a negative health-related incident could happen in a specific time (Menon, Raghubir, & Agrawal, 2008). Risk perceptions have two dimensions, susceptibility and severity. Susceptibility is the likelihood of experiencing a health risk (e.g., breast cancer) whereas severity is the seriousness, harmfulness, or dangerousness of the risk (e.g., stage of breast cancer) (Brewer et al., 2007).

Risk perceptions are a cornerstone of health-specific (e.g., health belief model) and general (e.g., theory of planned behavior) behavioral change theories. These individual-level theories underline the importance of risk perceptions as necessary, yet insufficient, for positive behavioral change. The theories generally posit that people who feel susceptible to a severe health risk will be motivated to reduce or eliminate the risk (Ajzen, 1991; Becker, 1974; Rogers, 1975; Witte, 1992).

These behavioral change theories, however, fail to recognize that people rarely estimate their risks accurately (Zeckhauser & Viscusi, 2000). People's risk perceptions are subjective and often deviate from objective risk estimates (e.g., medical tests) of susceptibility to and severity of a health risk (Gerend, Aiken, West, & Erchull, 2004). Nonetheless, subjective risk perceptions influence behavior more than objective risks estimates (Brewer & Hallman, 2006).

Although risk perceptions can be overestimated or underestimated, underestimation of risks is more common (Brewer & Hallman, 2006). Studies show that risk perceptions are underestimated for health risks such as breast cancer (Woloshin, Schwartz, Black, & Welch, 1999), HIV (Raghubir & Menon, 1998) and smoking-related problems (Strecher, Kreuter, & Kobrin, 1995). This bias is consistent across age groups (Weinstein, 1987), time (Shepperd, Helweg-Larsen, & Ortega, 2003) and cultures (Chang, Asakawa, & Sanna, 2001). The pattern has been documented whether risk perceptions are compared to those of an average other (Weinstein, 1980) or to objective measures of risk (Brewer & Hallman, 2006). When risk perceptions are not compared to any standard, similar patterns have been documented as evidenced by below mean estimates for negative events (Bränström et al., 2005).

Underestimation of risk prevents people from attending to health messages about risks to which they are susceptible and influences how they process such messages (Radcliffe & Klein, 2002). Furthermore, people who underestimate their risks are satisfied with their current behaviors and do not plan positive behavioral changes (e.g., quitting smoking, engaging in protected sex, using contraceptives, getting a swine flu vaccine) (Burger & Burns, 1988; Dillard, Mccaul, & Klein, 2006; Larwood, 1978; Sheer & Cline, 1994).

This dissertation posits that the principles of automaticity apply to how people process personal health risk messages and may explain why people are biased to underestimate personal risks. Three experiments examined the extent to which underestimation of self-relevant health risks is automatic -- the default option that occurs without conscious awareness of the bias (i.e., underestimation of health risks) and its

source (i.e., activation of self-schema); and is likely to occur when people are mentally taxed (i.e., under cognitive load) (Bargh, 1994). Additional attention was given to reactance, an individual difference variable (Dillard & Shen, 2005), and perceived risk controllability, a health risk characteristic (Klein & Larsen, 2002), which have been shown to affect perceptions of personal risks in previous studies. Reactance is a motivational state to restore or maintain one's freedom if it is perceived to be taken away by restriction of choices (Brehm, 1996). Controllable health risks are "preventable by personal action" (Weinstein, 1984, p. 431); that is, health conditions that people believe they can control either by not encountering the risk, or by controlling its severity, rate of growth and/or treatment outcomes.

The experiments relied on priming to activate the self-schema and measured the effects of this activation on health risk perceptions reaction time, the main dependent variable. Participants were primed with self-identity words (e.g., *I*, *me*) and then were asked to make judgments about their personal susceptibility to and the severity of health risks. Based on the premise that any mental process takes time (Cameron & Frieske, 1994), and that faster reaction times reflect more accessible, automatic, less thoughtful, and efficient concepts and processes (Bargh & Chartrand, 2000), reaction time was assessed. Reaction time was defined as the time in milliseconds that elapses between a participant's exposure to a health condition (e.g., "cancer," "broken bone") on a computer screen and her response (*yes*/*no*) using pre-assigned buttons on a buttons box.

Controlling for several individual differences (e.g., self-efficacy, dispositional optimism) and health risk characteristics (e.g., prevalence), the primary hypothesis was that, when asked about likelihood of experiencing a health condition, participants primed

with self-identity words would underestimate their health risks as evidenced by faster reaction times responding *no* to negative health conditions (e.g., heart attack) and *yes* to positive health conditions (e.g., healthy gums).

In Experiment 1, it was hypothesized that participants primed with self-identity words (e.g., *I*, *me*) would be more likely to respond *no* to negative health conditions and *yes* to positive ones when asked about their personal susceptibility to these conditions. They would make these decisions faster than those primed with third-person (e.g., *he*/*she*, *him*/*her*) or neutral (e.g., *it*) words as measured by reaction time. Thus, Experiment 1 examined lack of conscious awareness of underestimation of risk and of self-schema activation as its source as a defining feature of automatic health risk perceptions.

Experiment 2 examined efficiency as a defining feature of health risk perceptions. The hypothesis tested was that participants would exhibit faster reaction times responding *no* when asked about personal susceptibility to and severity of health conditions when they were mentally taxed (i.e., memorizing an eight-digit number) compared to when they were not (i.e., memorizing a two-digit number). These effects were predicted to be stronger for participants primed with self-identity words compared to those primed with third-person and neutral words. Underestimation of risk perceptions when people's cognitive resources are taxed would be a manifestation of an efficient automatic process; a process that occurs when people lack sufficient mental capacity to engage in elaborative processing of information.

The third experiment examined the effects of perceived risk controllability, a health risk characteristic, and psychological reactance, an individual difference variable, on perceptions of a health risk. The hypothesis tested was that highly reactant people (i.e.,

those who react negatively to restrictions on personal choice) when primed with self-identity words would be more likely to exhibit an automatic tendency to underestimate their susceptibility to and severity of a negative health condition when compared to participants who score low on this individual difference variable. This effect was predicted to be stronger if a health condition was perceived to be highly controllable (i.e., is preventable if personal action is taken).

The series of experiments conducted here are important on theoretical, practical, and methodological grounds. Theoretically, they are one of the first to make the case for the automaticity of risk perceptions. Rather than measuring risk perceptions as outcomes of consumption of health messages (Nabi & Oliver, 2009), the *processes* governing self-relevant health risk perceptions are identified. Bridging research on self, automatic processes, health communication and behavior change could resolve some of the inconsistencies in the literature (e.g., when and why personal health risk information is effective) and expand the focus of behavioral change theories beyond rational decisions to include automatic influences on health behavior (Ajzen & Fishbein, 2000; Brewer & Rimer, 2008).

A theoretical model is proposed to describe how people arrive at biased health risk perceptions. The model maps out how automatic self-schema activation can bias risk perceptions through indirect and direct routes of information processing. Self-schema activation takes different forms: pre-conscious, post-conscious and goal-dependent automaticity. The effects of post-conscious and goal-dependent automatic activation are most relevant to health communication. In the indirect route, the active self-schema guides elaborate/controlled processing of health messages consistent with people's

illusory perceptions of control, optimism, and positive self-views. In the direct route, people lack the motivation and/or ability to process risk messages. Thus, the active self-schema solely guides information processing in an automatic fashion.

Practically, understanding how risk perceptions work has both individual-level and public policy implications. On an individual level, understanding risk perceptions and the psychological processes responsible for them should promote more informed personal and proxy health decisions. On a public policy level, risk perceptions affect how a society responds to a health risk (e.g., promoting certain behaviors as healthy; initiating medical interventions; expending public funds on certain treatment options and therapies for chronic diseases) (Chapman, 2004; Eddy, 1984; Peters, McCaul, Stefanek, & Nelson, 2006). Knowing more about how health risk perceptions are formed should result in more effective public health initiatives.

Methodologically, the experiments conducted here demonstrate the value of priming and reaction time to advance our understanding of risk perceptions and health-related attitudes and behaviors. These methods uncover implicit cognitions and processes of which people are not aware, and override their unwillingness to report these judgments (Bargh & Chartrand, 2000; Nisbett & Wilson, 1977; Wegner & Wheatley, 1999).

In sum, health communication research and practice should move beyond the misplaced emphasis on the accuracy of risk perceptions given increasing evidence in support of intuitive/automatic decisions rather than rational decision making. We need to understand how automaticity applies to health risk information processing to improve the predictive power of theories of behavioral change. This, in turn, can improve the content,

implementation and evaluation strategies for interventions and communication campaigns.

In the next chapter, a review of the literature is presented. The chapter presents evidence regarding reasons for and implications of underestimated health risk perceptions. The study's theoretical propositions are outlined along with supporting evidence. It is posited that underestimation of personal risks is driven by a positivity bias and exhibits two features of automatic processes: lack of conscious awareness (of the bias and its source) and efficiency. Support for these propositions is derived from research documenting that health risk information is processed as self-relevant; is inconsistent with the positivity bias and self-threatening; and that self-schema activation is automatic. In the third chapter, the study design, hypotheses, procedures, and measures, along with pilot work, are presented. The fourth chapter reports the results of three experiments testing the main hypotheses. Finally, the fifth chapter includes a summary of the main results and a discussion of the theoretical, practical, and methodological implications for health risk communication research and practice.

CHAPTER 2

LITERATURE REVIEW

Risk Perceptions and Behavioral Change

Risk perceptions reflect a subjective probability that a negative health-related incident could happen in a specific time (Menon et al., 2008). Risk perceptions have two dimensions, susceptibility and severity, which, either additively or multiplicatively, capture one's perceptions of risk. Susceptibility, usually used interchangeably with words such as likelihood, vulnerability, and probability, is the possibility of experiencing a health risk. Severity, on the other hand, is the seriousness, harmfulness, or dangerousness of the risk (Brewer et al., 2007).

Risk perceptions are a cornerstone of individual-level behavioral change theories. As a crucial dependent variable in health education programs (Glanz et al., 2008; Hornik, 2002), theorists seek to understand the determinants of health behavior, and the processes governing its change and/ or maintenance, which, in turn, can inform interventions aiming to improve health behaviors (Glanz et al., 2008). A number of theories of behavior change consider risk perceptions a necessary, yet insufficient step, toward behavioral change (Ajzen, 1991; Becker, 1974; Rogers, 1975; Witte, 1992).

Risk perceptions determine behaviors directly, as in the health belief model (HBM), or indirectly through attitudes, as in theories of reasoned action (TRA), planned behavior (TPB), and the integrated behavioral model (IBM).

According to the HBM, for example, a person's perceptions of threat, which include susceptibility to and severity of (both medical/ clinical and social consequences such as stigma) a health condition, determine the likelihood of taking action (Becker, 1974). The model also underlines other determinants of behavior such as benefits of engaging in the health behavior promoted in the message, barriers to behavior, cues to action, which are environmental cues that serve as reminders to act (e.g., a mass media campaign, a close friend being sick, advice from others), and self-efficacy, a concept borrowed from Bandura's (1977, 1998) social cognitive theory (SCT), which refers to a person's capabilities of executing a specific course of action to achieve desired outcomes (Becker, 1974; Champion & Skinner, 2008).

In the TRA, TPB and IBM theories, risk perceptions are some of the underlying beliefs that determine attitudes. Attitudes, in turn, determine – among other variables such as perceived norms – intentions and, subsequently, behavior (Ajzen, 1991; Ajzen & Fishbein, 1973; Montaño & Kasprzyk, 2008). Beliefs reflect the probability that a specific outcome will have certain consequences or attributes multiplied by the positive or negative evaluation of these consequences and attributes. Behavioral beliefs are not determined *a priori* in the TRA, TPB, and IBM. Rather, pilot work elicits the beliefs people regard most important in determining their attitudes. Risk perceptions (concerns about susceptibility to and severity of a health risk) are considered determinants of attitudes, intentions, and behavior only if mentioned in the pilot phase of developing interventions guided by the TRA, TPB, or IBM (Ajzen, 1991; Ajzen & Fishbein, 1973; Eagly & Chaiken, 1993; Kaspryzk, Montaño, & Fishbein, 1998; Liska, 1984; Montaño & Kasprzyk, 2008; O'Keefe, 2002).

In another class of theories, acceptance of a health message rests on recipients' assessment of risk and response- and self-efficacy elements in the message. Both protection motivation theory (PMT) (Rogers, 1975) and the extended parallel process model (EPPM) (Witte, 1992), which are subsumed under the umbrella of fear appeals,[1] address the use of susceptibility and severity as a persuasive strategy. Both theories posit that when the rewards of engaging in a maladaptive behavior outweigh perceptions of susceptibility and severity, the message is rejected. When the effectiveness of a recommended response and the ability to engage in the promoted behavior outweigh the costs of engaging in that behavior, the message is accepted.

According to the PMT (Rogers, 1975), fear appeal messages are composed of susceptibility, severity, response efficacy, and self-efficacy. These perceptions produce protection motivation for initiating, sustaining, and driving action. Protection motivation results in message rejection if one's perceived susceptibility and severity do not outweigh the rewards of engaging in a maladaptive behavior (i.e., threat appraisals). If, on the other hand, the effectiveness of a recommended behavior and one's ability to engage in it exceed the costs of engaging in that behavior, the message is accepted (i.e., coping appraisals) (Rogers, 1975; Rogers & Prentice-Dunn, 1997).

According to Witte's EPPM (Witte, 1992), people engage in two appraisal processes after encountering a fear appeal message: threat appraisal and efficacy appraisal. In threat appraisal, they assess their susceptibility to a threat and the seriousness of the threat. If the threat is significant and personally relevant, they engage

[1] Fear appeals are characteristics of messages designed to scare people by stressing negative consequences if they do not engage in the promoted behavior. Fear is a negatively valenced emotion, which is accompanied by high levels of arousal when a threat is perceived as significant or serious and personally relevant.

in the second appraisal process, efficacy appraisal, in which they assess two aspects of the message: response efficacy and self-efficacy.[2]

Threat and efficacy appraisals result in one of three outcomes. If the threat is low, people lack the motivation to process the message and, thus, no further processing ensues (i.e., they do not engage in efficacy appraisals). If the threat is high, then the efficacy appraisal defines the nature of the outcome. Under high efficacy appraisals, people engage in danger control processes where they are likely to think about the recommended action and usually adopt the action promoted in the message as a way to eliminate or reduce the threat. Under low efficacy appraisals, people engage in fear control processes in which they are likely to engage in defensive processing (motivated resistance to the message), reactance, and/or denial to avoid experiencing a fear they cannot do anything about.

In sum, risk perceptions are at the core of behavioral change theories: central in some and secondary in others. Common across the theories is that risk perceptions motivate people to engage in self-protective behaviors. These theories, however, fail to recognize that people rarely estimate their risks accurately (Zeckhauser & Viscusi, 2000). People's risk perceptions are subjective and often deviate from objective risk estimates of one's susceptibility to and severity of a health risk (Gerend et al., 2004). However, subjective risk perceptions influence behavior more than objective risks estimates (Brewer & Hallman, 2006).

Biases in risk perceptions.

[2] Response efficacy is the effectiveness of the recommended action to reduce or eliminate the threat or risk whereas self-efficacy refers to people's own abilities to engage in and execute the recommended behavior.

Early models of decision making[3] characterized humans as rational beings who objectively search and evaluate all available information, and, ultimately, reach accurate decisions with the greatest benefit (Simon, 1955). However, both normative (how people should make decisions) and descriptive (how people actually make decisions) models of decision making are at odds with each other. Tversky and Kahneman (1986) argued, "the deviations of actual behavior from the normative model are too widespread to be ignored, too systematic to be dismissed as random error, and too fundamental to be accommodated by relaxing the normative system" (p. S252).

Evidence suggests human judgments are biased (i.e., consistently deviate from what would be expected based on objective evaluation of information or favor one decision over others) (Keren & Trigen, 2004). To account for limitations in human processing capabilities, Simon (1955) argued that human rationality is bounded. Later, Tversky and Kahneman (1974) developed the heuristics and biases approach to describe judgments and choices that depart from rational models of decision making. Subsequently, decades of research suggest that people rely on heuristics (e.g., availability, representativeness, anchoring and adjustment) and exhibit biases (e.g., optimistic bias, confirmation bias) in their judgment and decision-making processes (Kahneman, 1991; Tversky & Kahneman, 1974).

Health is no exception to this rule. People overestimate low probability, high-salient risks (e.g., nuclear risks) and underestimate high probability, highly controllable

[3] Early models of decision making (e.g., Bayes' theorem) required knowledge of all available information and the ability to process this information using statistical principles (Kahneman, 1991; Keren & Trigen, 2004). These models define the standards for measuring bias. Bayesian inference, sampling statistics, and regression analysis are the standard models in studies of judgment, whereas expected utility theory is the standard in the domain of choice (Kahneman, 1991).

voluntary ones (e.g., physical inactivity). People also fail to account for combined risks of simultaneous events (e.g., smoking and alcohol consumption) or those based on multiple or extended exposures to a risk (e.g., cumulative risk of smoking) (Svenson, 1984). Not only do laypeople exhibit these inaccuracies but also experts (e.g., physicians) (McNeil, Pauker, Sox, & Tversky, 2000).

Although risk perceptions can be biased upward or downward, underestimation of risks is more common (Brewer & Hallman, 2006). Studies show that risk perceptions are underestimated for health risks such as breast cancer (Woloshin et al., 1999), HIV (Raghubir & Menon, 1998) and smoking-related problems (Strecher et al., 1995; Weinstein, Slovic, & Gibson, 2004; Windschitl, 2002).

These downward estimates of one's risk are further strengthened by people's overconfidence in their judgments and search for evidence that is consistent with these judgments (i.e., confirmation bias). For example, educating people about probability does not eliminate people's overconfidence in their answers (Fischhoff, Slovic, & Lichtenstein, 1977). In support of confirmation bias, when presented with risk/benefit information about children's vaccines, non-vaccinator parents sought information that supported their previously held beliefs about the vaccine and discounted information inconsistent with their position (Meszaros et al., 1996). Similarly, participants used irrelevant information to underestimate their risks of encountering sexually transmitted diseases because the information was consistent with a desired outcome (e.g., having sex with an attractive person or maintaining a relationship with a current partner) (Knäuper, Kornik, Atkinson, Guberman, & Aydin, 2005).

Underestimation of risk prevents people from attending to health messages about risks to which they are susceptible and influences how they process such messages (Radcliffe & Klein, 2002). Furthermore, people who underestimate their risks are satisfied with their current behaviors and do not plan positive behavioral changes (e.g., quitting smoking, engaging in protected sex, using contraceptives, getting a swine flu vaccine) (Burger & Burns, 1988; Dillard et al., 2006; Larwood, 1978; Sheer & Cline, 1994).[4]

Theoretical Explanations for Underestimation of Health Risks

The literature reviewed thus far demonstrates how ubiquitous underestimation of health risk perceptions is. Such bias is resistant to de-biasing attempts (Weinstein & Klein, 2002) and persistent despite availability of information (Weinstein, 1980) and cognitive resources (Lench & Ditto, 2008).

This indicates a persistent source of the bias. The experiments described in this dissertation were designed to test two theoretical propositions to explain why people underestimate personal health risks.

Theoretical proposition 1. Underestimation of health risks is driven by a positivity bias. When the self-schema is active, perceptions of susceptibility will be biased upward for positive (e.g., healthy gums) and downward for negative (e.g., stroke) health conditions. Perceptions of severity will be biased downward for negative health conditions.

[4] Optimism about personal risks is considered a hallmark of mental and psychological health, which, in turn, is important for physiological health (See Maruta, Colligan, Malinchoc, & Offord, 2002; Reed, Kemeny, Taylor, Wang, & Visscher, 1994; Scheier & Carver, 1992; Taylor & Brown, 1988, 1994; Taylor, Lerner, Sherman, Sage, & McDowell, 2003).

Theoretical proposition 2. Underestimation of negative health risks is automatic. Biased risk perceptions possess two automaticity features: lack of awareness and efficiency. Underestimation of personal risks occurs without (a) conscious awareness of underestimation of risk as a bias or of self-schema activation as the source of this bias and (b) much cognitive resources.

Rationale for the aforementioned propositions is derived from research documenting that (a) health risk information is processed as self-relevant, (b) health risks are inconsistent with the positivity bias and self-threatening, and (c) self-schema activation is automatic, occurring despite lack of awareness, intention, control and cognitive resources.

Next, key findings supporting these two propositions are presented.

Health Risk Information is Processed as Self-relevant.

The self[5] is a unique mental structure that influences information processing (i.e., facilitates encoding and retrieval of information), an effect that is attributed to both structure and organization of self-knowledge in people's minds (Showers & Zeigler-Hill, 2003). Self-relevant information is better remembered compared to other methods of coding information (e.g., semantic) (Rogers, Kuiper, & Kirker, 1977; Symons & Johnson, 1997). Furthermore, people are quick in identifying and retrieving self-relevant information and descriptive traits and are confident about their judgments (Kuiper & Rogers, 1979; Markus, 1977). They are resistant to information inconsistent with their

[5] The meaning of the self is different in many of the conceptualizations of self-related phenomena (e.g., self-awareness, self-efficacy, self-focus, self-talk, loss of self), making it difficult for sociologists and psychologists to agree on a unifying definition. Gordon Allport (1961) mentioned that it was easy to feel the self, but difficult to define. One definition uses reflexive thinking as an underlying ability in the majority of self-related concepts. Thus, the self can be defined as a mental capacity that allows a person to take oneself as the object of one's thoughts; that is to think about him/herself (Leary & Tangney, 2003).

self-description (Markus, 1977). Studies have provided evidence for self-reference effects even on a neurological level (Kelley et al., 2002; Kircher et al., 2002).

Oftentimes, health risk messages explicitly reference the self. "Get yourself tested" (KFF, 2009) is an example of such messages. When the self is not explicitly referenced, health messages are most likely processed as self relevant because they urge the recipient to assess her own risk factors regarding the risk in question and take further action based on these risk perceptions (Kircher et al., 2002). However, self-relevant risk messages are at odds with the positivity bias and are self-threatening.

Health Risk Information is Inconsistent with the Positivity Bias and Self-threatening.

Inconsistency with positivity bias. People develop positive views of themselves early in life. Research shows that children, as early as five months, prefer positive feedback (e.g., approving voices). Societies' endorsement of positive reinforcement perpetuates this preference. Repeated activation renders these positive views well practiced, chronically accessible, and applicable for processing new information (Fernald, 1993; Paulhaus, 1993; Swann, Hixon, Stein-Seroussi, & Gilbert, 1990).

To maintain positive self-views, people develop defense mechanisms that guard against self-relevant negative information (Baumeister, Dale, & Sommer, 1998). People favor (Zuckerman, 1979), seek (Sedikides, 1993) and uncritically accept (Kunda, 1990) positive feedback but neglect (Baumeister & Cairns, 1992; Sedikides & Green, 2000), discount with skepticism (Kunda, 1990), attempt to refute (Wyer & Frey, 1983) or selectively forget negative feedback (Mischel, Ebbesen, & Zeiss, 1976). Furthermore, people think causes of positive events lie within themselves (e.g., personal skills) whereas similar events in lives of others are due to external reasons (e.g., luck) (Bradley,

1978; Ross, Green, & House, 1977). They circumvent their own flaws by judging them as common and unimportant while exaggerating their strengths (Marks, 1984).[6]

In health, similar patterns are evident. People uncritically accept positive health information and devalue negative feedback especially when unexpected (Renner, 2004). People selectively focus on their risk-reducing behaviors (Gerrard, Gibbons, & Warner, 1991) and attribute good health outcomes to personal reasons and bad outcomes to external ones (Kahlor, Dunwoody, & Griffin, 2002). Furthermore, health risk messages evoke defense mechanisms where a message is judged as less valid and accurate as opposed to messages consistent with one's preference for being healthy (Ditto & Lopez, 1992; Jemmott, Ditto, & Croyle, 1986).

On an aggregate level, meta-analyses have found that tailoring based on personal risk results in less acceptance of the message (Albarracin et al., 2003; Noar, Benac, & Harris, 2007). When susceptibility and severity have positively affected behavior, the target audience has been high-risk groups who had already acknowledged their risks (Janz, 1984).

Self-threat. Health risks are threatening, not only in the physical sense, but also to one's positive self regard (McCoy, Gibbons, & Gerrard, 1999). Two pieces of evidence support this reasoning.

First, people tend to self affirm (i.e., restore a positive self regard by emphasizing one's successes) after being threatened. Vulnerability to health risks is considered threatening as evidenced by participants' tendency to self affirm afterward. For example,

[6] The positivity bias does not rule out that people acknowledge "negative pockets of incompetence" about themselves (Taylor & Brown, 1988, p. 203). See also Showers & Zeigler-Hill (2003) on the organization of self-knowledge for undeniable negative events such as divorce.

listing risky sexual behaviors that increased vulnerability to sexually transmitted diseases led participants to self affirm by providing favorable ratings on a personality measure and reporting careful contraceptive behaviors (McCoy et al., 1999).

Second, people are more accepting of threatening information if their self is affirmed (Correll, Spencer, & Zanna, 2004). For example, self-affirmed participants were more accepting of health risk messages about the link between caffeine (Reed & Aspinwall, 1998) and alcohol consumption (Klein & Harris, 2009) and breast cancer, and of graphic warning labels on cigarette packs (Harris, Mayle, Mabbot, & Napper, 2007). Furthermore, self-affirmed participants engage in positive behaviors (i.e., condom purchase) (Sherman, Nelson, & Steele, 2000). Similar patterns have been documented using implicit measures (van Koningsbruggen, Das, & Roskos-Ewoldsen, 2009).

In sum, personal health risk messages are self-relevant, yet are at odds with positivity bias and self-threatening. These effects are important given evidence of the automatic activation of self-schema.

Self-schema Activation is Automatic.[7]

Research on the self has shifted from a focus on consciousness to unconsciousness and automatic processing. Studies have shown that the self-schema is automatically and unconsciously activated upon encountering self-relevant information.

[7] Automatic processes have one or more of the following features: lack of awareness, intention, control, or cognitive resources. Automatic processes are ones of which a person is not consciously aware, do not intend (no intention exists or no causal link exists between act and intention), is unable to change, stop, or avoid and require only limited processing resources (Bargh, 1994; Moors, & Houwer, 2007). For early work on the primacy of unconsciousness, see Baumeister, Bratslavsky, Muraven, & Tice (1998); Libet, Gleason, Wright, & Pearl (1983); Wegner & Wheatley (1999). For reviews on historical roots and context of automaticity research, see Bargh & Ferguson (2000); Bruner (1957); Nisbett & Wilson (1977).

For example, people automatically allocate attention to their names (Moray, 1959) and self-descriptive traits (Bargh, 1982).

Furthermore, the self is automatically associated with positive. Participants are faster associating the self with positive as opposed to negative items (Greenwald & Franham, 2000). People have implicit preference for their name letters and birthday dates, which can even influence life decisions such as selection of career and romantic partners (Jones, Pelham, & Carvallo, 2004; Koole, Dijksterhuis, & van Knippenberg, 2001).

Further, participants with limited cognitive resources (e.g., those under memory or time constraints) preferred positive social feedback and interaction partners who evaluated them favorably (Swann et al., 1990) and indicated higher likelihood of experiencing positive events (Lench & Ditto, 2008). Support for the positivity bias has also been found on a neurological level (Watson, Dritschel, Obansawin, & Jentzsch, 2007).

Additional Theoretical Propositions

Self-report perceptions of risk are sensitive to health risk characteristics as well as individual difference variables (Harris, Griffin, & Murray, 2008; Helweg-Larson & Shepperd, 2001). This dissertation aimed to test the effects of health risk characteristics and individual differences on implicit measures of risk perceptions. Further, it aimed to test the differential effect, if any, of risk characteristics and individual differences on susceptibility and severity, the two dimensions of risk perceptions of interest in this dissertation.

Health risk characteristics influencing risk perceptions.

Risks vary on several dimensions, which, in turn, influence risk perceptions. Frequency of a health risk is correlated with increased perceived susceptibility (Weinstein, 1980) and decreased severity perceptions (Jemmott et al., 1986). Similarly, personal experience with a risk increases susceptibility perceptions (Velde, Hooykaas, & Pligt, 1992; Weinstein, 1980), a result that has been explained in terms of availability and representativeness heuristics. For example, family history of cancer and memories of loved ones influence women's personal assessments of breast and/or ovarian cancer risk (Kenen, Ardern-Jones, & Eeles, 2003). Similar results were documented regarding women's perceived susceptibility to breast cancer, heart disease, and osteoporosis (Gerend et al., 2004; Katapodi, Facione, Humphreys, & Dodd, 2005).

Stereotype salience and exempt beliefs, on the other hand, are related to decreased risk perceptions (Weinstein, 1980, 1987). When people have a stereotypical image of a person to whom negative events can happen, they tend to underestimate their own risks. When no stereotype exists, they are less optimistic. One explanation is that stereotypes or exemplars result in a contrast, rather than assimilation, effect; that is, people easily distance themselves as different from the exemplar being displayed in the message (Zillmann, 2002), which leads them to underestimate their risks. Exempt beliefs are defined as the belief that if a person has not yet suffered a health condition, s/he is unlikely to encounter the risk in the future. These beliefs have been shown to influence risk perceptions downward (Weinstein, 1987).

Perceived health risk controllability is correlated with decreased risk perceptions (Taylor et al., 1992; Velde et al., 1992). Weinstein (1984) defined controllable risks as "preventable by personal action" (p. 431). Control is related to stereotype salience such

that people typically have no stereotype in mind for uncontrollable health risks (i.e., it can happen to anyone). Control is also related to personal experience. Experiencing a negative event negates illusions of control, and, thus, makes a risk available in people's minds (Helweg-Larsen & Shepperd, 2001).

In a meta-analysis of the relationship between optimistic bias and controllability, Klein and Larsen (2002) found that controllability explained about 10% of the variance (r = .49). In three experiments, Lin, Lin, and Ragubir (2003) found that self-positivity bias was greater for events perceived to be controllable. According to the self-positivity account, people prefer controllable risks to uncontrollable ones. To the extent that they can attribute risks to controllable events, people can maintain and boost their self-esteem by thinking they have the abilities and skills to reduce or eliminate the risk (Harris et al., 2008). Given this body of research, this summary proposition is proposed:

Theoretical proposition 3. Frequency of and personal experience with a health condition will elicit overestimated risk perceptions whereas stereotype salience, exempt beliefs, and controllability will elicit underestimated risk perceptions.

Individual differences influencing risk perceptions.

Several individual differences are correlated with risk perceptions. Controllability is an individual difference variable that correlates with risk perceptions. Hoorens and Buunk (1993) found that people high on internal locus of control (as opposed to external locus of control), those who believe that their health status is dependent on their own behaviors (as opposed to being dependent on external factors such as others or luck) (Wallston & Wallston, 1981), were more optimistic about their chances of encountering various health problems (e.g., alcohol problems, heart attack, suicide, AIDS, cancer).

Similarly, self-esteem is correlated with low levels of risk perceptions (Helweg-Larsen & Shepperd, 2001). Self-esteem is "the evaluative dimension of self-knowledge (e.g., "am I good at this?")" (Baumeister, 1999, p. 2). People high on self-esteem are more resilient in the face of obstacles, negative feedback, and life events (Shrauger & Rosenberg, 1970; Shrauger & Sorman, 1977). In health contexts, Robberson and Rogers (1988) found that a positive appeal to self-esteem (e.g., positive effects of exercise on self confidence, self acceptance, and appearance) resulted in more intentions to engage in healthy behaviors compared to a negative appeal to self-esteem. High self-esteem people resist threatening information (Gerrard, Kurylo, & Reis, 1991), engage in defensive processing of risk information resulting in an underestimation of health risks (Smith, Gerrard, & Gibbons, 1997) and have low commitment to behavioral change (Gibbons, Eggleston, & Benthin, 1997).

Self-efficacy is considered key in behavior enactment (Viswanath, Wallington, & Blake, 2009). Defined as confidence in one's own abilities to engage in and execute recommended behaviors (Bandura, 1977, 1998), self-efficacy is integral to several behavioral change theories (e.g., HBM, TRA, TPB, EPPM). Further, efficacy beliefs moderate the relationship between risk perceptions and self-protective and information-seeking behaviors. When informed about risks, people who are high on self-efficacy are more likely to engage in risk-reduction and information-seeking behaviors (Rimal, 2001; Rimal & Real, 2003).

Reactance is positively correlated with self-esteem (Brockner & Elkind, 1985). Reactance is a motivational state that aims to restore or maintain one's freedom when one's autonomy is eliminated or reduced (as when choices are perceived to be restricted).

Reactance is dependent on the importance of the behavioral/ attitudinal/ emotional domain, the magnitude of the threat, and the degree of reduction in one's freedom. In other words, the more important the domain is for a person, the greater the threat, and the greater the reduction is in one's freedom, the greater the magnitude of reactance a person exhibits (Brehm, 1966).

Conceptualized as a mix of negative cognitions and anger (Dillard & Shen, 2005), reactance results in going against the recommended or suggested alternative such as healthy foods (Bushman, 1998; Fitzsimons & Lehmann, 2004) even when recommended by an expert source, increased confidence in one's decisions (Fitzsimons & Lehmann, 2004), and initiation of risky behaviors (e.g., smoking) (Miller, Burgoon, Grandpre, & Alvaro, 2006). Furthermore, highly reactant patients show detrimental patterns to therapy as evidenced by low levels of improvement (based on therapists' ratings), boundary augmentation, which refers to engaging in behaviors meant to distance a person away from his/her therapist, and early termination of therapy (Seibel & Dowd, 1999). Recent evidence suggests that reactance can be triggered without people's conscious awareness or intent (Chartrand, Dalton, & Fitzsimons, 2007).

Finally, low risk perceptions are correlated with happy mood (Larsen & Shepperd, 2001) and dispositional optimism, a general expectancy that positive outcomes will happen in the future (Radcliffe & Klein, 2002). On the other hand, negative-affect related variables (e.g., sad mood, state and trait anxiety) are correlated with high levels of risk perceptions. Negative affect makes negative thoughts about oneself, and to a lesser extent negative thoughts about others, salient or accessible and therefore is associated with high levels of risk perceptions (Larsen & Shepperd, 2001). On a neurological level,

optimism involves the activation of the rostral anterior cingulate cortex, a region that shows irregularities among depressed individuals (Sharot, Riccardi, Raio, & Phelps, 2007).

Finally, self-consciousness refers to people's tendency to focus their attention inward or outward (Fenigstein, Scheier, & Buss, 1975). Research has shown that individual differences on self-consciousness with its two subscales, private and public self consciousness, reflect a chronic activation of self-knowledge (Hull, Slone, Meteyer, & Matthews, 2002).[8] Wheeler, Morrison, DeMarree, and Petty (2008) further demarcated two subscales of private self-consciousness: internal self-awareness -- one's awareness of his/her own feelings, and thoughts; and self-reflection -- one's rumination about the self. The previous research on individual differences can be summarized theoretically as:

Theoretical proposition 4. Internal locus of control, self-efficacy, self-esteem, reactance, dispositional optimism, and self-reflection will elicit lower levels of risk perception whereas depression, external locus of control, and self-awareness will elicit higher levels of risk perception.

In sum, risk perceptions are necessary for behavioral change. Yet, people's estimates of personal health risks are inaccurate and are most often underestimated for negative health conditions. Underestimation of health risks creates a paradoxical situation for health communication messages, which typically highlight personal susceptibility to and severity of health risks as a persuasive strategy.

This dissertation takes a psychological approach to explain why people underestimate health risks despite health communication efforts to inform them. It posits

[8] See Wheeler, DeMarree, & Petty (2007) on the "active self account" that advocates the importance of studying the role of the self on behavioral priming.

that people are predisposed to automatically underestimate self-relevant risks. Using implicit measures of risk perceptions, three experiments were designed to examine two defining features of automatic processes: (a) lack of conscious awareness of underestimation of risks as a bias and of self-schema activation as a source of this bias, and (b) efficiency.

The effects of health risk characteristics and individual differences on implicit measures of risk perceptions are also examined. It is unclear whether their influence on implicit measures of risk perceptions, if any, will mirror the patterns documented with self-report data or not. Finally, susceptibility and severity are examined separately to uncover a differential effect, if any, of health risk characteristics and individual differences on these two dimensions of risk perceptions investigated in this dissertation.

The next chapter outlines the design, hypotheses, and procedures of three experiments, and three pilot studies.

CHAPTER 3

METHODS

This dissertation examined the extent to which underestimation of personal health risk perceptions is an automatic process by testing two defining features of automatic processes: (a) lack of conscious awareness of both underestimation of health risk perceptions as a bias and the source of this bias (i.e., self-schema activation) and (b) efficiency (i.e., occurrence of the bias under limited cognitive resources).

Three experiments examined the effects of self-schema activation on health risk perceptions. The main research question was: Controlling for perceived health risk characteristics and individual difference variables, what is the relationship between self-schema activation and perceptions of personal susceptibility to and severity of health conditions?

Experiment 1 tested the hypothesis that people are not consciously aware of their underestimation of health risks as a bias and of the self-schema activation as a source of this bias. Experiment 2 examined health risk perceptions under cognitive load to determine whether underestimation of risk is an efficient process (i.e., a process that occurs when people are mentally taxed). Within the context of a health message, Experiment 3 examined the effects of health risk controllability and psychological reactance on health risk perceptions.

Experiment 1: Unconscious awareness of underestimation of personal health risk perceptions

Experiment 1 examined lack of conscious awareness of underestimation of personal health risk perceptions as a bias and of self-schema activation as a source of this bias. The experiment was a 3 (prime: self-identity, third-person, neutral words) x 2 (priming method: supraliminal, subliminal priming)[9] x 2 (valence of health condition: positive, negative) mixed-factorial design. Both the prime and priming method were between-subjects factors and valence of the health risk was a within-subjects factor. A conscious condition was added to the design for comparison with the priming conditions, bringing the total number of conditions to seven. In the conscious condition, participants were not primed and, thus, the condition was not crossed with the prime and priming method factors.

Previous research has shown that health risk information is inconsistent with the positivity bias and self-threatening. Self-schema activation and the positivity bias have been shown to operate at an automatic or unconscious level. Thus, the hypothesis tested in Experiment 1 was that when the self-schema was activated, perceptions of personal susceptibility to health conditions would be biased upward for positive health conditions and downward for negative ones (See Appendix 1: Table 1 for study hypotheses and results).

H1: *Responses and reaction times to questions about personal susceptibility to health conditions will be biased upward for positive conditions and downward for negative ones (main effect for valence of health condition) and for participants primed*

[9] A full description of these techniques and how they were applied is presented in the "Manipulations and measures" section of this chapter.

with self-identity words (main effects for prime). Participants will be more likely to respond (a) "yes" in response to positive health conditions and "no" in response to negative health conditions, and (b) to respond faster. Responses and reaction times will be biased for participants in the self-identity words condition compared to participants primed with third-person and neutral words.

Experiment 2: Efficiency of underestimation of personal health risk perceptions

Experiment 2 examined the efficiency feature of underestimation of personal health risk perceptions by manipulating participants' cognitive resources. The experiment examined the effects of self-schema activation on perceptions of personal susceptibility to and severity of negative health conditions under high and low cognitive load conditions using a 3 (prime: self-identity, third-person, neutral words) x 2 (cognitive load: high vs. low) between-subjects factorial design.

Previous literature has documented people's self-positivity bias under limited cognitive resources (either by imposing a time constraint or cognitive load). Imposing a constraint on participants' cognitive capacity serves two purposes. First, if people have an automatic tendency to judge themselves as less at risk for negative health conditions and more prone to experience positive health outcomes, this tendency should not be disrupted under scarce cognitive resources (e.g., when people are forced to adopt a speed criterion or are engaged in another simultaneous task). As a matter of fact, this tendency should be stronger under limited processing capacities. Second, constraining participants' cognitive resources simulates real time situations in which people typically engage in other activities while they are exposed to media messages in general and health messages in particular (Perse, 1990).

Consistent with the rationale and hypothesis for Experiment 1, the hypothesis tested in Experiment 2 was that when the self-schema is activated, perceptions of personal susceptibility to and severity of negative health conditions will be biased downward. This effect was expected to be stronger in the high cognitive load condition (i.e., under limited cognitive resources).

H2a: *Participants in the self-identity words condition (main effects for self-schema activation) and under high cognitive load (main effects for cognitive load) will be more likely to respond (a) "no" as opposed to "yes" to questions about personal susceptibility to and severity of negative health conditions, and (b) to respond faster, compared to participants in the third-person, and neutral words conditions, and low cognitive load condition.*

H2b: *Constraining participants' cognitive resources will bias responses and reaction times to questions about personal susceptibility to and severity of health conditions when the self-schema is activated. There will be an interaction effect between self-schema activation and cognitive load such that participants primed with self-identity words will be more likely to: (a) respond "no" as opposed to "yes" to questions of personal susceptibility to and severity of negative health conditions and (b) to respond faster, when under high cognitive load compared to low cognitive load.*

Experiment 2 differed from Experiment 1 in three ways. First, Experiment 2 was limited to negative health conditions because these conditions have implications for information processing, precautionary behavior, and initiation of a medical course of action (Larsen & Shepperd, 2001). Second, perceptions of severity were assessed. Third, Experiment 2 (and 3) relied exclusively on subliminal priming, rather than both

supraliminal and subliminal priming, for two reasons: (1) both subliminal and supraliminal conditions should yield similar results in Experiment 1, and (2) subliminal priming is considered a more stringent procedure that is in line with automaticity research because it ensures that participants are not consciously aware of the prime and its effects on their risk perceptions (Bargh & Chartrand, 2000).

Experiment 3: Effects of health risk controllability and psychological reactance on health risk perceptions

Experiment 3 examined the effects of health risk controllability, a health risk characteristic, and psychological reactance, an individual difference variable, on judgments of susceptibility to and severity of a health risk. The experiment was a 3 (prime: self-identity, third-person, neutral words) x 2 (health risk controllability: high vs. low, manipulated variable) between-subjects design. Psychological reactance was a measured independent variable.

Previous literature suggests both psychological reactance and perceived health risk controllability bias risk perceptions downward. These effects should be stronger when the self-schema is activated.

H3a: *Participants in the self-identity words condition (main effects for self-schema activation), high on reactance (main effects for psychological reactance), and in the highly controllable health risk condition (main effects for perceived health risk controllability) will be more likely (a) to respond "no" as opposed to "yes" to questions of personal susceptibility to and severity of a negative health condition, and (b) to respond faster, compared to participants in the third-person, and neutral words conditions, participants low on reactance, and those in the low controllability condition.*

H3b: *Health conditions perceived to be highly controllable will bias responses and reaction times to questions about personal susceptibility to and severity of health conditions when the self-schema is activated. There will be a two-way interaction effect between self-schema activation and perceived health risk controllability such that participants primed with self-identity words will be more likely to: (a) respond "no" as opposed to "yes" in response to questions about personality susceptibility to and severity of a health condition, and (b) respond faster, when the health condition is perceived as highly controllable as opposed to less controllable.*

H3c: *Psychological reactance will bias responses and reaction times to questions about personal susceptibility to and severity of health conditions when self-schema is activated. There will be a two-way interaction effect between self-schema activation and reactance such that participants high on reactance will be more likely to: (a) respond "no" as opposed to "yes" in response to questions about personal susceptibility to and severity of a health condition, and (b) respond faster, when primed with self-identity words compared to third-person and neutral words.*

H3d: *There will be a three-way interaction effect between self-schema activation, psychological reactance, and perceived health risk controllability such that participants high on reactance will be more likely to: (a) respond "no" as opposed to "yes" in response to questions about personal susceptibility to and severity of a health condition, and (b) respond faster, when primed with self-identity words compared to third-person and neutral words. This effect will be stronger for highly controllable health conditions compared to those perceived as less controllable.*

Additional Hypotheses

Previous studies have shown that perceived health risk characteristics and individual differences bias self-report risk perceptions upward or downward. No data are available regarding their effects on implicit measures of risk perceptions. Further, little is known about the differential effect, if any, of health risk characteristics and individual differences on susceptibility and severity, the two dimensions of risk perceptions investigated in this dissertation.

Several health risk characteristics (i.e., prevalence, stereotype salience, perceived risk controllability, exempt beliefs, and personal experience) were controlled for in examining the effects of the independent variables on perceptions of personal susceptibility to and severity of health conditions. Individual difference variables that have been shown to influence risk perceptions in previous studies were also controlled for. These variables were: depression, dispositional optimism, health locus of control, psychological reactance, self-consciousness, self-efficacy, and self-esteem. The last two hypotheses predict the direction of influence of these variables:

H4: *Participants will overestimate their susceptibility to highly prevalent health conditions with which they have had personal experience. Participants will underestimate their susceptibility to controllable health conditions, and ones for which exempt beliefs and stereotypes of a typical person who suffers the health condition are salient.*

H5: *Participants high on self-efficacy, internal locus of control, self-esteem, psychological reactance, dispositional optimism, and self-reflection will be more likely to underestimate their susceptibility to and severity of negative health conditions and overestimate their susceptibility to positive ones. Participants low on self-efficacy and*

reactance, high on external locus of control, self-awareness, and depression will be more likely to overestimate their susceptibility to and severity of negative health conditions and underestimate their susceptibility to positive ones.

Participants

A convenience sample of undergraduate and graduate students was recruited from the subject pool in the UNC-CH School of Journalism and Mass Communication as well as campus wide. Students participated in the experiments in return for course credit or monetary incentives. The UNC-CH Institutional Review Board reviewed and approved the study design and procedures.[10]

A priori power analysis suggested that, for a 6-condition study, a total of 53 participants per cell would be sufficient to detect small effect size ($\omega^2 = .04$) with a power of .80, at the standard .05 significance level (G Power Software; Keppel & Wickens, 2004).[11]

Procedures

Upon registration, participants received an email with a link to an online survey, which included control variables (e.g., self-efficacy). Order of presentation of survey items was randomized. To proceed with the survey, participants had to read an online consent form and agree to participate in the study. Having participants fill out the survey before coming to the lab minimized potential effects on later tasks (i.e., priming and dependent variable measures).

[10] IRB# 11 – 0547.

[11] Studies measuring response time as a dependent variable typically run a minimum of 30 participants per condition (e.g., Payne, 2001) to a maximum of 55 (e.g., Comello & Slater, 2011).

Upon arrival to the lab, participants were randomly assigned to experimental conditions and given a participant ID. They were asked to sign a hard copy of the consent form, turn off cell phones, and put away their backpacks.

At the end of each experimental session, participants answered funneled debriefing questions to ensure they were unaware of the nature and purpose of the primes, the relationship between the priming procedure and the dependent task, and any effect the priming task might have had on their subsequent responses to questions of personal susceptibility to and severity of health conditions (Bargh & Chartrand, 2000). Participants who were recruited for monetary incentives were compensated (See Table 2 for a summary of study procedures).

Studies were administered in the Media Effects Lab in the School of Journalism and Mass Communication. The lab accommodates five participants at a time. *MediaLab* and *DirectRT* software are installed on each computer.

Manipulations and Measures

In all three experiments, priming was used to manipulate self-schema activation and response key and reaction time were measured as the main dependent variables.

Independent variables.

Self-schema activation. Self-schema was manipulated by two priming techniques. In the supraliminal priming condition (Experiment 1: condition supraliminal), participants participated in a supposed language ability task. They were presented with 25 five-word sets and asked to form grammatically correct four-word sentences. This task is known as the "scrambled sentence task" (Srull & Wyer, 1979). Each of the five-word sets contained a self (i.e., *I*, *me*), third-person (i.e., *he*/*she*) or neutral (i.e., *it*) prime. Order of

presentation of the scrambled sentences was randomized (See Appendix 2 for study manipulations and measures).

The supraliminal priming technique ensured that participants were unaware of the effects of the prime (i.e., scrambled sentence task) on later tasks (i.e., risk perceptions) even though they were consciously aware of the prime.

Subliminal priming, although a weaker prime compared to the supraliminal method, ensured the primes were inaccessible for conscious awareness and, thus, awareness of the prime did not contribute to the hypothesized effects. In the subliminal prime conditions (Experiment 1,subliminal condition; and Experiment 3), participants participated in a supposedly visual acuity task, in which they were asked to indicate whether items flashed on a computer screen appeared from the top, bottom, left, or right side of a central fixation point. The flashed words were the primes (i.e., self-identity, third-person, or neutral words).

Participants were instructed to focus their eyes on the center of the computer screen because it was the best way to identify the location of the flashed items. The items were white on a black background. Primes appeared in the participants' parafoveal visual region.[12] Each prime was both front and back masked (10 milliseconds).[13] The prime itself lasted for 50 milliseconds. Order of presentation of primes and their location were randomized (following Bargh & Chartrand, 2000). In Experiment 1, a total of 50 primes were presented to participants, divided on two trials. In Experiment 3, this number was

[12] Parafoveal or peripheral visual region is 2 to 6 degrees from a central fixation point.

[13] The mask is a stimulus that has the same features of the prime but does not interfere with the prime or initiate any spread activation in the memory. The mask erases the visual buffer of a person, which ensures that the actual duration of the prime in people's visual memory is equal to its actual duration on the computer screen. In this study, the front mask was "&&&&" and the back mask was "xxxx."

increased to 80 primes, 40 per trial. In the first trial, participants were asked to press pre-assigned *yes* and *no* buttons to indicate whether the stimulus appeared from the right or left of the computer screen. In the second trial, they had to identify whether the stimulus appeared from the top or bottom of the screen.

Finally, for Experiment 2, the primes, front and back masked, were presented in participants' parafoveal vision followed by the target word (i.e., health conditions) appearing in the middle of the computer screen. Similar to Experiments 1 and 3, items were white on a black background. Each prime was both front and back masked (10 milliseconds). The prime itself lasted 50 milliseconds. Order of presentation of primes and their location were randomized.

Valence of health condition. In Experiment 1, perceptions of susceptibility to both positive and negative health conditions were assessed. Based on pilot work (described below), a list of 50 negative health conditions had been compiled. Ten health conditions were reworded to convey a positive health condition (e.g., healthy gums, healthy bones).

Cognitive load. In Experiment 2, cognitive resources were manipulated by having participants in the high cognitive load condition hold an eight-digit number in their memory versus a two-digit number for participants in the low cognitive load condition. Numbers were generated using Random.org, an online number generator website. Participants were prompted to memorize a new number every 25 health conditions.

Participants responded to four blocks of 25 health conditions each.[14] Order of presentation of blocks was randomized.

Psychological reactance. In Experiment 3, Hong's 14-item reactance scale was used to test the effects of reactance, as a measured individual difference variable, on health risk perceptions (Hong & Faedda, 1996).

Health risk controllability. In Experiment 3, risk controllability was manipulated. Based on pilot work described below, two versions of an informational pamphlet about Balamuthia infection, a rare but deadly disease, were presented to participants. Results of the pilot work suggested the manipulation was successful.

Dependent variables.

Response key. Two buttons on a buttons box were pre-assigned a *yes* or *no* response. Participants hit either button in response to the susceptibility to and severity of health condition presented on the screen.

Reaction time. Reaction times, the time that elapsed from the presentation of a target word (Experiments 1 and 2) or a short sentence (Experiment 3) on the computer screen till the participant hit one of the two pre-assigned response keys, were recorded in milliseconds.

Behavioral measure. Experiment 3 used a behavioral measure where participants were given a choice to take a one-page pamphlet that included additional information about Balamuthia infection, the health condition they had read about in the study.

Control variables.

[14] For susceptibility items, participants in the high-load condition memorized the numbers 25691843 for Block 1 and 97128563 for Block 2 versus 23 and 68, respectively, for the low-load condition. Similarly, for severity, participants in the high-load condition memorized the numbers 65341782 for Block 1 and 27981564 for Block 2 versus 17 and 26, respectively, for the low-load condition.

Individual difference variables. The study used Beck's (1979) depression inventory to measure depression. The 21-item scale measured behavioral indications of depression such as social withdrawal and sleep disturbance.

To measure dispositional optimism, a ten-item Life Orientation Test (Scheier, Carver, & Bridges, 1994) was used. The scale included items such as "I'm always optimistic about my future."

Control as an individual difference variable was measured using the health locus of control scale (Wallston, Wallston, Kaplan, & Maides, 1976). The scale's 11 items tap internal (e.g., "If I take care of myself, I can avoid illness") and external (e.g., "Good health is largely a matter of good fortune") locus of control.

Self-consciousness was measured using Fenigstein, Scheier, and Buss's (1975) 23-item scale. Example items included: "I'm always trying to figure myself out."

Self-efficacy was measured using Sherer et al.'s (1982) scale. It includes 17 items such as "When I make plans, I am certain I can make them work."

Rosenberg's (1979) 10-item scale was used to measure self-esteem. It included items such as "I take a positive attitude toward myself."

Finally, to ensure the results obtained were not due to differences in participants' affective states, a modified version of the affect-arousal scale (Aarts & Dijksterhuis, 2003; Salovery & Birnbaum, 1989) was administered at the end of each experimental session. The scale is an eight-item semantic differential measure of affect (e.g., sad/ happy) and arousal (e.g., calm/ excited).

Health risk characteristics. Data collected in Pilot Study 1 (described below) were used to control for health risk characteristics. In Pilot Study 1, undergraduate

students rated 99 health conditions on various dimensions: familiarity, prevalence, stereotype salience, perceived risk controllability, exempt belief, and personal experience. These variables were used to create clusters of health conditions that were familiar to the student population. These clusters were used to calculate mean response key and reaction times in Experiments 1 and 2.

Baseline reaction time and response key accuracy. To familiarize participants with the study procedures and collect baseline reaction time and response key accuracy, a list of 20 words, 10 animals (e.g., monkey) and 10 objects (e.g., desk), were randomly presented to participants. Participants were asked to indicate as quickly as possible whether each word was an animal, in which case they would press *yes*, or not, in which case they would press *no*.

Data Analysis

Data was analyzed using the PASW 19.0 statistical package. Data were screened for missing values, univariate and multivariate outliers, and fit to the assumptions underlying the statistical methods used (Tabachnick & Fidell, 2007).

Repeated measures analysis of variance was used for hypotheses testing. Significant interactions were followed by tests for simple main effects. Logistic regression analysis was used for dichotomous dependent variables (e.g., behavioral measure in Experiment 3).

For reaction times, data points less than 300 milliseconds and more than 5,000 were deleted. Then, outliers beyond four standard deviations were excluded from the analysis to reduce their influence on the solution. Finally, a log transformation was

performed to normalize response time data, which tend to be positively skewed. Analyses were performed on transformed data (Bargh & Chartrand, 2000; Fazio, 1990).

Pilot Work

Three pilot studies were conducted. Pilot Study 1 identified health conditions familiar to the student population, which were used in Experiments 1 and 2, and provided normative data to control for health risk characteristics. Pilot Study 2 pretested the risk controllability manipulation used in Experiment 3. Pilot Study 3 tested the associative strength between the self and sickness and health traits to determine whether sickness/health were traits that people considered (non) self-descriptive (See Appendix 3 for a description of pilot work).[15]

Pilot Study 1: Selection of health conditions.

To select health conditions familiar to the student population, a list of health conditions was compiled from several studies (e.g., Christensen-Szalanski, Brck, & Christensen-Szalanski, 1983; Harris et al., 2008; Klar & Ayal, 2004) and online resources (e.g., Centers for Disease Control and Prevention). Health conditions chosen were neither general in nature (e.g., digestive problems) nor gender specific (e.g., breast cancer).

Three groups of undergraduate students ($N = 70$ in total participated for course credit) rated a list of 33 different health conditions (a total of 99 conditions across the three groups) on several characteristics that affect risk perceptions (e.g., Christensen-Szalanski et al., 1983; Harris et al., 2008; Weinstein, 1980).

(1) Familiarity: the extent to which participants had previously encountered the name of the health condition.

[15] IRB# 10 – 2019.

(2) Frequency or population prevalence: the percentage of people to whom this health condition could occur.

(3) Stereotype salience: the ease with which participants could identify a person who suffers from the health condition.

(4) Perceived risk controllability: the extent to which a person could control the health condition.

(5) Personal experience: the extent to which each participant has personally experienced the health condition in question or known a close other (family member or a friend) who has suffered this condition.

(6) Exempt belief: the belief that if a health condition had not yet happened to a participant, it is unlikely to happen to him/her.

(7) Personal susceptibility: the likelihood of developing or experiencing the health condition.

(8) Severity: the noxiousness, seriousness, or dangerousness of the health condition if a participant developed it.

Results. Based on the familiarity ratings, 50 health conditions were known to 90% or more of the participants and were, thus, used in Experiments 1 and 2.[16]

A correlation matrix for ratings of health conditions on dimensions of perceived prevalence, stereotype salience, risk controllability, exempt belief, and personal experience, susceptibility, and severity as well as third-person susceptibility and severity appear in Table 3.

[16] Pilot Study 2 replicated familiarity of health conditions ratings collected in Pilot Study 1. Health conditions unfamiliar to the rating groups in Pilot Study 1 were also unknown to participants in Pilot Study 2.

A hierarchical cluster analysis was run on the 50 familiar health conditions based on five standardized health characteristics variables: perceived prevalence, stereotype salience, controllability, exempt beliefs, and personal experience.[17] Using Ward's method, the analysis produced three clusters, between which the health risk characteristics variables were significantly different (See Table 4 for descriptive statistics for clusters and differences on health risk characteristics). The first cluster included 17 health conditions perceived to be of low prevalence, salience, controllability, exempt beliefs, and personal experience (e.g., leukemia, HPV). The second cluster included 26 health conditions (e.g., allergies, diabetes) perceived to be moderate on these risk characteristics variables. The third cluster included seven conditions (e.g., flu, stress) that were scored high on these variables (See Appendix 3 for a complete list of health conditions familiar to undergraduate population and the cluster to which each condition belongs).

In sum, 50 health conditions were selected for inclusion in Experiments 1 and 2. These 50 conditions fell into three homogeneous clusters based on participants' ratings of them based on several health risk characteristics.

Pilot Study 2: Pretesting health risk controllability manipulation.

To pretest the health risk controllability manipulation used in Experiment 3, three groups of undergraduate students rated one of three versions of an informational pamphlet about Balamuthia infection. Balamuthia infection was chosen because it is a

[17] Variables were standardized to eliminate the influence of prevalence, which was measured on a 0 – 100 scale, on cluster formation.

rare disease, which reduced participants' prior knowledge about and/or personal experience with the disease as potential confounding variables.[18]

Three versions of the pamphlet provided identical information regarding susceptibility to and severity of the disease and were similar in word count. Pamphlets differed in levels of control over routes of infection (i.e., the extent to which one can prevent exposure to the disease), rate of progress (i.e., control over successive development of the disease from one stage to another), and curability (i.e., ease of diagnosis, and availability and effectiveness of treatment).

Participants read the informational pamphlet and, then, rated Balamuthia infection on various dimensions of perceived risk controllability for both the self and a third person (i.e., participants' perceptions of control that an average third-person would have over the disease). Dimensions of perceived health risk controllability included: control over susceptibility, severity, progression rate, treatment effectiveness, and general control (See Appendix 3 for a complete description of Pilot Study 2).

Results. Seventy students participated in the study for course credit. Students were randomly and equally assigned to one of three conditions: high-risk controllability, low-risk controllability, and a neutral condition (n = 24, 23, and 23, respectively).

All ten items measuring perceived health risk controllability, for the self and average third person, were examined. There were no missing values on any of the items. A correlation matrix along with means and standard deviations appears in Table 5.

[18] Familiarity ratings collected in Pilot Studies 1 and 2 confirmed students were not familiar with Balamuthia infection. In Pilot Study 1, none of the participants had heard about the disease whereas only 2 participants (8.7%) had heard about it in Pilot Study 2 but had no personal experience with it.

Two factors with eigenvalues greater than 1 were extracted using an oblique Principal Components Analysis.[19] The two factors explained 84.95% of the variance. The results revealed that perceived risk controllability had two underlying factors that were moderately correlated. The first factor was related to perceived general control whereas the second factor was related to perceived susceptibility control. Based on the analysis, two averaged indices were created. Eight items reflected general control ($\alpha = .96$), and two items reflected susceptibility control ($r = .79$), with a correlation of .58 between the two factors, which is suitable for a multivariate analysis of variance (See Table 6 for factor loadings, factor correlation, and internal reliability statistics).

A one-way multivariate analysis of variance was performed on perceived health risk susceptibility and general control as dependent variables. Evaluation of the assumptions of normality of sampling distributions, linearity, and homogeneity of variance within each experimental condition were satisfactory.[20]

---

[19] Evaluation of normality of distribution, outliers, and factorability were satisfactory. All variables were within the acceptable ±2 for skewness and kurtosis. One case exceeded the critical value of $Z_{.005}$. Multivariate outliers were checked using Mahalanobis distance. Two cases were above the critical value of chi-square ($\chi^2_{(.001, 10)} = 29.58$) but did not have a large Cook's D value, which suggested they were not influential on the solution. Kaiser-Meyer-Olkin (KMO) measure of sampling adequacy revealed there was a degree of common variance appropriate for conducting a Principal Components Analysis (KMO = .91). Each item's Measure of Sampling Adequacy (MSA) was greater than .60, with the lowest being .85. Accordingly, all items were retained for the analysis. Bartlett's test of sphericity indicated the correlation matrix is factorable ($\chi^2 = 791.05, p < .001$). The initial solution indicated there were two factors with eigenvalues greater than 1. Similarly, Catell's criterion for the scree plot suggested that there might be two factors. With the extraction of two factors, there were only five (11.0%) non-redundant residuals with absolute values greater than .05.

[20] The two dependent variables were within the acceptable range of ±2 for skewness and kurtosis for all three experimental conditions. No univariate outliers were detected at $Z_{.005}$ level. No multivariate outliers exceeded the critical value of chi-square ($\chi^2_{(.001, 2)} = 13.81$). Levene's test showed variances were homogeneous for the two dependent variables: susceptibility control ($F(2,67) = 2.17, p = .122$) and general control ($F(2, 67) = 1.58, p = .213$). Box's M test revealed no problems regarding the homogeneity of the variance-covariance matrices ($\chi^2 = 10.62, p = .118$). Bartlett's test of sphericity was significant ($\chi^2 = 10.41$, $p = .005$) suggesting the two dependent variables were suitable for multivariate analysis.

The Manova was significant (Pillais = .66, $F(4, 134) = 16.48$, $p < .001$) suggesting there were differences among the three conditions on at least one linear combination. As a follow-up to the significant MANOVA, a Discriminant Function Analysis was performed to find the linear combination(s) that maximize the differences among the groups. For a significant MANOVA, the first function is always found to be significant ($\Lambda = .36$, $p < .001$). The second function in isolation was not significant ($\Lambda = .96$, $p = .133$).[21]

Function 1's eigenvalue of 1.67 had a canonical correlation of .79 and explained 98% of the variance. Function 2's eigenvalue of .03 had a canonical correlation of .18 and explained 2% of the variance. The first function was heavily dominated by perceived susceptibility control[22] whereas the second function was heavily dominated by perceived general control.[23]

Conducting a one way ANOVA demonstrated that discriminant scores from the first function were significant ($F(2, 67) = 56.12$, $p < .001$) whereas scores from the second function were not ($F(2, 67) = 1.15$, $p = .321$). To find which groups differed from each other, Tukey HSD was conducted. For the first function, the test revealed all three conditions were significantly different from each other at the .05 level.

This series of analyses suggest the manipulation of health risk controllability was successful (see Figure 1). The Manova and the follow-up tests suggested there was one

[21] In the univariate world, all two models testing for differences between levels of independent variable, risk controllability, on the two dependent variables were found to be significant (perceived susceptibility control: $F(2, 69) = 50.47$, $p < .001$, perceived general control: $F(2, 69) = 20.87$, $p < .001$).

[22] The correlation between the first weighted composite and susceptibility control was .94 with a standardized coefficient of .84.

[23] The correlation between the second weighted composite and perceived general control was .80 with a standardized coefficient of .99.

function that separated the three experimental conditions, which was heavily dominated by perceived susceptibility control. Perceived susceptibility control varied by the levels of risk controllability manipulation. Participants in the high-risk controllability group exhibited a high level of perceived susceptibility control ($M = 4.72$, $SD = 1.09$), followed by the neutral ($M = 2.45$, $SD = 1.18$), and low-risk controllability ($M = 1.86$, $SD = .77$) groups.

In sum, Pilot Study 2 pretested the risk controllability manipulation used in Experiment 3. The manipulation was successful: Participants in the high controllability condition perceived themselves as being more in control over the likelihood of contracting the disease compared to participants in the low controllability condition.

Pilot Study 3: Associative strength between the self and health and sickness traits.

The purpose of this pilot study was to examine whether sickness/health are traits that people consider (non)self-descriptive similar to negative/positive traits, which has been shown in previous studies (Greenwald & Franham, 2000). The study was a 2 (prime: self-identity words vs. neutral) x 2 (trait category: positive, negative, health, sickness) mixed-factorial design. The prime was the between-subject factor and the trait category was the within-subject factor.

In response to whether traits presented on the computer screen were self-descriptive or not, it was hypothesized that *responses and reaction times to questions will be biased downward for negative and sickness traits and upward for positive and health ones (main effect for trait category) and for participants primed with self-identity words (main effects for prime). Participants will be more likely to respond (a) "no" in response to negative and sickness traits, and "yes" in response to positive and health traits, and*

(b) to respond faster. Responses and reaction times will be biased for participants in the self-identity words condition compared to those in the neutral condition.

Procedures. Under the guise of a personality traits study, participants were asked to indicate as quickly as possible whether each trait that appeared on the computer screen was self-descriptive or not by pressing one of two pre-assigned buttons on a buttons box: *yes* or *no*. A total of 40 personality traits had been randomly selected from Anderson's (1968) 200 high meaningful personality traits using Random.org, a random number generator. Twenty traits were positive (e.g., "sincere") and 20 were negative (e.g., "phony"). Embedded within Anderson's personality traits was a list of 13 health- (e.g., "healthy," "wholesome") and 11 sickness-related words (e.g., "unhealthy," "sick"). Order of presentation of traits was randomized (See Appendix 3 for a complete list of positive, negative, health, and sickness traits).

Participants were subliminally primed with self-identity words (e.g., *I*, *me*) whereas the neutral group was primed with a letter string (e.g., &&&xxx). Primes were white on a black background. Primes were presented in participants' parafoveal vision for 50 milliseconds and were both front- and back-masked (10 milliseconds for each mask) followed by a personality trait appearing in the middle of the computer screen till a response was recorded. Order of presentation of primes and their location were randomized.

Results. A total of 150 students participated in the study for a $5 incentive. They were randomly assigned to one of two conditions: self vs. neutral primes. Ninety-seven

participants were females (64.7%) and 128 were right-handed (85.3%).[24] Mean age was 20.93 (*SD* = 1.54). There were no missing values on any variable.

Baseline measures. No differences were detected between self and neutral conditions on baseline (i.e., practice trial) response key accuracy and reaction time. Participants in both self (*M* = 19.62, *SD* = .58) and neutral (*M* = 19.64, *SD* = .62) conditions correctly identified the practice-trial items as animals/non-animal (*t*(148) = -.13, *p* = .893).[25] Raw baseline reaction time data fell between 331 and 5,294 milliseconds. Data points exceeding 5 seconds were excluded (*n* = 1). Then, data points that were 4 *SD* away from the mean were considered outliers and excluded (*n* = 35), a 1.2% total data points lost. Mean baseline reaction time after outlier deletion was not statistically different between the self (*M* = 648.63, *SD* = 1.12) and neutral (*M* = 650.57, *SD* = 1.11) conditions (*t*(148) = -.16, *p* = .873).[26]

Effects of prime and trait category. Pilot Study 3 tested the effects of the prime (self vs. neutral) and trait category (positive, negative, health, sickness) on two dependent variables: response key (*yes*/*no*) and reaction time. In response to whether traits presented on the computer screen were self-descriptive (*yes*) or not (*no*), it was predicted that participants would respond "*yes*" to positive and health traits and "*no*" to negative and

[24] Data on right vs. left-handedness were collected to control for their influence on reaction time, which has been shown in previous studies (Nosek, Greenwald, & Banaji, 2007).

[25] Data were checked for assumptions of normality and homogeneity of variances. Skewness and kurtosis were within ±2 for each condition with the exception of kurtosis in the neutral condition (3.81). Homogeneity of variances were assumed as evidenced by a non significant Levene's test (*F*(1, 148) = .01, *p* = .984).

[26] Data were checked for assumptions of normality and homogeneity of variances. Skewness and kurtosis were within ±2 for each condition. Homogeneity of variances were assumed as evidenced by a non significant Levene's test (*F*(1, 148) = .15, *p* = .697).

sickness traits. Reaction times for "*yes*" responses to positive and health traits would be faster than "*no*" responses. Reaction times for "*no*" responses to negative and sickness traits would be faster than "*yes*" responses. Finally, it was predicted that these patterns would be stronger for participants in the self-prime condition than participants in the neutral condition.

(1) Effects of prime and trait category on response key. Means for response keys *yes* and *no* were calculated for each trait category Participants were similar across the two experimental conditions in responding *yes* (i.e., self-descriptive trait) to positive and health traits and *no* (i.e., non self-descriptive trait) to negative and sickness traits. Participants identified positive (e.g., "sincere") and health (e.g., "healthy") traits as self-descriptive in the self (M = .89 and .70, SD = .13 and .18, respectively) and neutral (M = .91 and .71, SD = .11 and .20, respectively) conditions. On the contrary, participants identified negative (e.g., "gossipy") and sickness (e.g., "unhealthy") traits as non self-descriptive in the self (M = .91 and .93, SD = .10 and .11, respectively) and neutral (M = .89 and .95, SD = .15 and .09, respectively) conditions (See Table 7).

A repeated measures analysis of variance was performed with prime as a between-subjects factor, and trait category (positive, negative, health, sickness) as a within-subjects factor, to detect differences on mean count of *yes* responses to trait categories (i.e., *yes* signifies a self-descriptive trait). Data were screened for fit of underlying assumptions of normality of the sampling distribution, homogeneity of

variance-covariance matrices, linearity, and multicolinearity.[27] Using Wilks' criterion, the interaction term between prime and trait category was not significant ($F(1.77, 262.31) = .57, p = .545, \eta_p^2 = .004$). For the between-subjects factor, no statistically significant differences were found between conditions when response key was averaged over all trait categories ($F(1, 148) = .19, p = .660, \eta_p^2 = .001$). Mean response key count was .43 and .44 for the self and neutral conditions, respectively. When averaged over groups, however, response key was found by Hotellings' *T* to be significant by trait category ($F(1.77, 262.31) = 1252.85, p < .001, \eta_p^2 = .894$).[28]

To correct for post hoc inflation of familywise Type I error, individual alphas for each of the six contrasts were set up using the False Discovery Rate approach (FDR).[29] Post hoc results indicated participants identified positive traits as self-descriptive ($M = .90$) significantly more than they identified negative ($M = .09$), health ($M = .70$), and sickness ($M = .05$) traits as self-descriptive. Similarly, participants identified health traits as self-descriptive significantly more than they identified negative and sickness traits as self-descriptive. Finally, participants identified negative traits as self-descriptive significantly more than sickness traits. All comparisons were significant at $p < .001$.

---

[27] Distribution of mean response key was not within the acceptable range of ±2 for negative and sickness traits. Sixteen data points fell beyond $Z_{.005}$. Six cases were considered multivariate outliers, which exceeded the critical chi-square value of $\chi^2_{(.001, 4)} = 18.46$ but only one had a large Cook's D. Homogeneity of variances was assumed for all dependent variables as evidenced by non-significant Levene's tests. Homogeneity of the variance –covariance matrices was assumed given equal *n*. Linearity of the relationship between the dependent variables was checked by plotting all pairs of dependent variables. Correlations between variables were moderate, which is not a threat to multicollinearity.

[28] Mauchly's test indicated that the assumption of sphericity had been violated ($\chi^2 = 154.50, p < .001$). Therefore, degrees of freedom were corrected using Greenhouse-Geisser estimates of sphericity ($\varepsilon = .59$).

[29] *P* values were set up at .008, .016, .025, .033, .041, and .05 for consecutive contrasts ranked from the smallest to biggest *p* values.

(2) Effects of prime and trait category on reaction time combined for yes and no response key. Raw reaction time data for target trait categories fell between 318 and 24,256 milliseconds. Data points exceeding 5 seconds were deleted ($n = 6$). Then, data points beyond 4 *SD* were considered outliers and deleted ($n = 89$), a total of 0.92% data points lost (See Table 8 for descriptive statistics).

A repeated measures analysis of variance was performed with prime as between-subjects factor, and trait category as a within-subjects factor, to detect differences on reaction time combined for the *yes* and *no* response keys. Mean reaction time was calculated for each of the four trait categories (positive, negative, health, sickness). Data were screened for the fit of underlying assumptions regarding the normality of the sampling distribution, homogeneity of variance-covariance matrices, linearity, and multicolinearity.[30]

Using Wilks' criterion, the interaction term between prime and trait category was not significant ($F(2.71, 401.11) = 1.98, p = .122, \eta_p^2 = .013$). For the between-subjects factor, no statistically significant differences were found between conditions when reaction time was averaged over all trait categories ($F(1, 148) = .004, p = .948, \eta_p^2 < .001$). Mean reaction time was 922.57 and 924.69 for the self and neutral conditions, respectively. When averaged over groups, however, reaction time was found by

[30] Distribution of reaction time within each condition was within the acceptable range of ±2 for skewness and kurtosis. Three cases fell beyond $Z_{.005}$. One case exceeded the critical value of $\chi^2_{(.001, 4)} = 18.46$ but did not have a large Cook's D. Homogeneity of variances was assumed for all dependent variables except for mean response time to sickness ($F(1, 148) = 5.19, p = .024$). Box's M test revealed no problems regarding the homogeneity of the variance-covariance matrices ($\chi^2 = 14.223, p = .182$). Linearity of the relationship between the dependent variables was checked by plotting all pairs of dependent variables. Variables were highly correlated, an expectation in multivariate analysis, and posed no problems.

Hotellings' T to be significant by trait category ($F(2.71, 401.11) = 64.03, p < .001, \eta_p^2 = .302$)[31] (See Figure 2).

Post hoc analysis, adjusted for Type I error inflation using the FDR approach, indicated participants responded significantly faster to positive traits ($M = 868.96$) compared to negative ($M = 933.25$), health ($M = 988.55$), and sickness ($M = 903.64$) traits. Further, people responded faster to negative traits compared to health traits. Finally, participants responded faster to sickness traits compared to negative and health traits. All comparisons were significant at $p < .001$.

Although the interaction term between prime, the between-subjects factor, and all four trait categories, the within-subjects factor, was not significant, limiting the analysis to positive and sickness traits revealed a significant interaction ($F(1, 148) = 3.80, p = .053, \eta_p^2 = .025$) and a within-subjects, trait category (positive vs. sickness) effect ($F(1, 148) = 12.62, p < .001, \eta_p^2 = .079$) whereas the prime, the between-subjects factor, was not significant ($F(1, 148) = .08, p = .772, \eta_p^2 = .001$).

As a follow up to a significant interaction, main effects, with alpha set to .025, revealed no differences between the two experimental groups with trait category held constant: positive ($F(1, 148) = .89, p = .346$) and sickness ($F(1, 148) = .16, p = .687$) traits. Mean reaction time to positive traits was 857.03 and 883.07 in the self and neutral conditions, respectively. Mean reaction time to sickness traits was 910.33 and 898.87 in the self and neutral conditions, respectively. With experimental group held constant, main effects revealed no differences in reaction time to positive and sickness traits within the

[31] Mauchly's test indicated that the assumption of sphericity had been violated ($\chi^2 = 22.63, p < .001$). Therefore, degrees of freedom were corrected using Greenhouse-Geisser estimates of sphericity ($\varepsilon = .90$).

neutral condition ($F(1, 148) = 1.29, p = .259$). Within the self condition, however, there was a statistically significant difference ($F(1, 148) = 15.14, p < .001$). Participants responded faster to positive ($M = 857.03$, $SD = 1.21$) compared to sickness ($M = 910.33$, $SD = 1.24$) traits.

(3) Effects of prime and trait category on reaction time split by yes and no response key. A closer look at Table 8 revealed a pattern in which participants identified positive ($M = 877.00$) and health ($M = 990.83$) traits as self-descriptive faster than they identified these traits as non self-descriptive ($M = 1{,}099.00$ and $1{,}124.60$, respectively). On the contrary, participants identified negative ($M = 963.82$) and sickness ($M = 916.22$) traits as non self-descriptive faster than they identified them as self-descriptive ($M = 1{,}202.26$ and $1{,}273.50$, respectively) (See Figures 3a and 3b). Performing a repeated measures analysis of variance on reaction time to trait categories (positive, negative, health, sickness) split by response key (*yes/no*)[32] showed a non significant interaction between prime and trait category ($F(4.81, 105.89) = 1.01, p = .413, \eta_p^2 = .044$) and prime ($F(1, 22) = .53, p = .474, \eta_p^2 = .024$). Trait category, however, had a significant effect on reaction time ($F(4.81, 105.89) = 13.71, p < .001, \eta^2 = .384$).[33]

Post hoc comparisons, corrected for familywise Type I error inflation using the FDR approach, showed six significant differences. Participants identified positive traits as self-descriptive ($M = 918.33$) significantly faster than they identified negative ($M =$

[32] Reaction time split by *yes* and *no* response keys signifies reaction time for traits identified as self-descriptive (i.e., time to respond "*yes*") and those identified as non self-descriptive (i.e., time to respond "*no*").

[33] Mauchly's test indicated that the assumption of sphericity had been violated ($\chi^2 = 47.72, p = .009$). Therefore, degrees of freedom were corrected using Greenhouse-Geisser estimates of sphericity ($\varepsilon = .68$).

1,235.94) and sickness (M = 1,261.82) traits as self-descriptive and health as non self-descriptive (M = 1,172.19). Participants also identified negative (M = 995.40) and sickness (M = 963.82) traits as non self-descriptive faster than they identified sickness traits as self-descriptive (M = 1,261.82). Finally, participants identified sickness traits as non self-descriptive (M = 963.82) faster than they identified negative traits as self-descriptive (M = 1,235.94).[34]

It is noteworthy that differences between the two groups were not attributed to differential affect/arousal status. Measured at the end of the experimental session, the affect/arousal index (α = .89) was similar for the self (M = 1.27, SD = 1.81) and neutral (M = 1.33, SD = 1.74) conditions ($t(148) = -.21$, $p = .832$ *ns*).[35]

In sum, Pilot Study 3 provided partial support for the hypothesis. The main effects for trait category were supported whereas the main effects for the self primes were not.

Consistent across the two experimental conditions, trait category was a powerful predictor of response key and reaction time. Participants identified positive and health traits as self-descriptive. In contrast, they identified sickness and negative traits as non self-descriptive. In terms of reaction times, participants responded faster to positive traits followed by sickness, negative, and health traits. Participants found it easier to identify positive and health traits as self-descriptive (as evidenced by faster reaction time) but

[34] The analysis on reaction time split by response key (*yes*/*no*) was performed on only 24 cases (n = 12 per condition). Few participants identified negative and sickness traits as self-descriptive and positive and health traits as non self-descriptive, which reduced the sample size from 150 to 24 with listwise deletion. Reported means are for 24 cases.

[35] Distribution of the dependent variable was within the acceptable range of ±2 for skewness and kurtosis. Equal variances were assumed as evidenced by a non significant Levene's test ($F(1, 148) = .19$, $p = .662$).

more difficult to identify negative and sickness traits as self-descriptive (as evidenced by slower reaction time).

Although there were no differences between the experimental conditions in response key and reaction time, participants in the self-prime condition responded significantly slower to sickness as opposed to positive traits.

Conclusions from Pilot Studies

Three pilot studies were conducted. Pilot Study 1 identified 50 health conditions with which the student population was familiar. These health conditions were then grouped into three homogeneous clusters based on several health risk characteristics. In the analysis for Experiment 2 (described in Chapter 4), response key and reaction time data were averaged across the health conditions that belonged to each cluster.

Pilot Study 2 showed that the manipulation of health risk controllability was successful. The informational pamphlet used in the pilot was used in Experiment 3 to test the effects of health risk controllability along with psychological reactance on risk perceptions when the self-schema was active.

Finally, Pilot Study 3 tested the associative strength between the self and health and sickness traits. The study provided preliminary evidence that sickness and health traits mirror negative and positive traits in their implicit (dis)association with the self, which has been shown in previous studies (e.g., Greenwald & Franham, 2000).

The next chapter describes the results from the three main experiments designed to test the hypotheses. Experiment 1 examined the proposition that people are not consciously aware of their underestimation of health risks as a bias and of self-schema activation as a source of the bias. Experiment 2 examined the proposition that

underestimation of health risks is an efficient process, one that occurs when people are mentally taxed. Finally, Experiment 3 examined the effects of a manipulated health risk characteristic (i.e., risk controllability) and an individual difference variable (i.e., psychological reactance) on health risk perceptions.

CHAPTER 4

RESULTS

Experiment 1: Unconscious awareness of underestimation of personal health risk perceptions

Experiment 1 tested the hypothesis that people are not consciously aware of their underestimation of health risk perceptions as a bias or of self-schema activation as the source of this bias. The experiment was a 3 (prime: self-identity, third-person, neutral) x 2 (priming method: subliminal, supraliminal) x 2 (valence of health condition: positive, negative) mixed-factorial design. Both the prime and priming method were between-subjects factors whereas the valence of health condition was a within-subjects factor. The study included an additional conscious condition, which was not crossed with the prime and priming method.

A total of 325 participants were recruited for a $10 incentive. They were randomly assigned to one of seven experimental conditions. The majority of participants were females (n = 243, 74.8%) and right-handed (n = 300, 92.3%). Mean age was 21.20 (SD = 2.39). Data for Experiment 1 included measures of individual differences (e.g., self-efficacy) as well as response key and reaction time data for susceptibility to 40 negative (e.g., HIV) and 10 positive (e.g., fertility) health conditions, for a total of 50.

First, baseline measures are described. Second, descriptive statistics and reliability of individual difference variables are presented along with their effects on response key

as a dependent variable. Third, response key and reaction time data are analyzed to detect differences between the experimental conditions.

Baseline measures.

A series of univariate ANOVAs, with prime and priming method as fixed factors, showed no differences between the experimental groups on baseline response key accuracy and reaction time. For baseline response key accuracy, there were no differences between the experimental conditions in total correct response key as a dependent variable. The interaction term ($F(2, 273) = .22, p = .801$), prime ($F(2, 273) = .72, p = .485$), and priming method ($F(1, 273) = .98, p = .321$) were not significant. Participants in the self ($M = 19.46$, $SD = 1.18$), third-person ($M = 19.64$, $SD = .68$) and neutral ($M = 19.44$, $SD = 1.44$) conditions correctly identified practice-trial items as animal/non-animal. Averaged across priming method, means were 19.48 ($SD = 1.05$) and 19.54 ($SD = 1.23$) for the subliminal and supraliminal priming conditions, respectively.[36] Similarly, there were no differences between the unconscious and conscious conditions ($F(1, 323) = .74, p = .388$).[37] Participants in the unconscious ($M = 19.51$, $SD = 1.15$) and conscious ($M = 19.67$, $SD = .63$) conditions correctly identified practice-trial items as animal/non-animal.

Raw baseline reaction time data fell between 336 and 9,471 milliseconds. Data points exceeding 5 seconds were excluded ($n = 2$). Then, data points that were 4 *SD* away from the mean were considered outliers and excluded ($n = 65$), a 1.03% total data points

[36] Total correct response key was transformed using 1/(K-X) formula after which skewness and kurtosis were within the acceptable ±2 within conditions. Two cases exceeded the critical value of $Z_{.005}$. Equal variances were not assumed as evidenced by a significant Levene's test ($F(5, 273) = 2.92, p = .014$).

[37] Total correct response key was transformed using 1/(K-X) formula after which skewness and kurtosis were within the acceptable ±2 within conditions. Equal variances were assumed as evidenced by a non-significant Levene's test ($F(1, 323) = 2.93, p = .087$).

lost. A univariate ANOVA performed on log-transformed data showed the interaction term between prime and priming method ($F(2, 273) = .65$ $p = .523$), prime ($F(2, 273) = .21, p = .805$), and priming method ($F(1, 273) = .68, p = .408$) were not significant. Mean reaction time to practice trial items was 634.74 ($SD = 1.11$), 637.67 ($SD = 1.11$), and 631.10 ($SD = 1.12$) for participants in the self, third-person, and neutral conditions, respectively. Averaged over priming method, reaction time was 637.96 ($SD = 1.12$) for the subliminal condition and 630.95 ($SD = 1.10$) for the supraliminal condition. Similarly, there were no differences between the unconscious and conscious conditions ($F(1, 323) = .00, p = .986$).[38] Participants in the unconscious conditions responded in 634.45 ($SD = 1.11$) milliseconds whereas those in the conscious condition responded in 634.30 ($SD = 1.09$) milliseconds.

Individual differences.

Individual difference variables were screened for normality of distributions and outliers. There were no missing values (See Table 9 for descriptive statistics and correlation matrix).

Participants scored low on the depression inventory ($M = 1.36$, $SD = .29$, $\alpha = .85$).[39] Anchored between 0 and 3, participants responded to 21 behavioral indications of depression (e.g., sleep disturbances), with high scores reflecting high levels of depression. Consistently, participants were optimistic, as measured by the life orientation

[38] The distribution of log-transformed reaction time data was within the acceptable range of ±2 for skewness and kurtosis within each experimental condition. Equal variances were assumed as evidenced by a non-significant Levene's test with prime and priming method as fixed factors ($F(5, 273) = .97, p = .433$) and with unconscious vs. conscious factor ($F(1, 323) = 2.47, p = .116$).

[39] Reported reliability is for 20 items of the depression inventory. A weight loss item was excluded because it reduced the sample size from 325 to 127 (those who were trying to loose weight) with listwise deletion. With the weight loss item included, reliability was .82 ($n = 127$).

test (*M* = 2.54, *SD* = .65, α = .79). Anchored between 0 = *strongly disagree* and 4 = *strongly agree*, with high scores reflecting high levels of optimism, participants responded to six statements such as "I'm always optimistic about my future."

Participants showed slightly high levels of internal health locus of control (*M* = 3.85, *SD* = .49, α = .58). Anchored between 1 = *strongly disagree* and 6 = *strongly agree*, participants responded to 11 items tapping internal (e.g., "If I take care of myself, I can avoid illness") and external (e.g., "Good health is largely a matter of good fortune") locus of control. The scale was coded in the direction of internal locus of control: High scores reflected high levels of internal health locus of control and low scores reflected high levels of external health locus of control.

Two factors with eigenvalues greater than 1 were extracted using an oblique Principal Axis Factoring. The two factors explained 23.39% of the variance.[40] Consistent with the literature, results revealed health locus of control had two underlying factors. The first factor was related to internal locus of control whereas the second factor was related to external locus of control. Based on the analysis, five items reflected internal locus of control (α = .60), and five items reflected external locus of control (α = .56),

[40] Evaluation of normality of distribution, outliers, and factorability were satisfactory. All variables were within the acceptable ±2 for skewness and kurtosis. Thirteen cases exceeded the critical value of $Z_{.005}$. Multivariate outliers were checked using Mahalanobis distance. Four cases were above the critical value of chi-square ($\chi^2_{(.001, 11)} = 31.26$) but did not have a large Cook's D value, which suggested they were not influential on the solution. Kaiser-Meyer-Olkin (KMO) measure of sampling adequacy revealed there was a degree of common variance appropriate for conducting a factor analysis (KMO = .69). Each item's Measure of Sampling Adequacy (MSA) was greater than .60, with the lowest being .65. Accordingly, all items were retained for the analysis. Bartlett's test of sphericity indicated the correlation matrix is factorable ($\chi^2 = 377.83$, $p < .001$). The initial solution indicated there were two factors with eigenvalues greater than 1. Similarly, Catell's criterion for the scree plot suggested that there might be two factors. With the extraction of two factors, there were 13 (23.0%) non-redundant residuals with absolute values greater than .05.

with a correlation of .08 between the two factors (See Table 10 for factor loadings, factor correlation, and internal reliability statistics).

Participants were slightly reactant ($M = 3.07$, $SD = .46$, $\alpha = .78$). Anchored between 1 = *strongly disagree* and 5 = *strongly agree*, participants responded to 14 statements that measured psychological reactance (e.g., "Regulations trigger a sense of resistance in me"). Psychological reactance was positively correlated with self-consciousness and negatively with self-efficacy and self-esteem.

Participants' scores were roughly at the mid point of the self-consciousness scale ($M = 2.53$, $SD = .37$, $\alpha = .74$) Anchored between 0 = *extremely uncharacteristic of me* and 4 = *extremely characteristic of me*, with high scores reflecting high levels of self-consciousness, participants responded to 23 items (e.g., "I am constantly examining my motives").

Three factors with eigenvalues greater than 1 were extracted using a varimax Principal Axis Factoring. The three factors explained 30.70% of the variance.[41] Consistent with the literature, results revealed self-consciousness had three underlying factors. The first factor was related to social anxiety (e.g., "I feel anxious when I speak in front of people"). The second was related to public self (e.g., "I'm concerned about what

[41] Evaluation of normality of distribution, outliers, and factorability were satisfactory. All variables were within the acceptable ±2 for skewness and kurtosis. Fifty-five cases exceeded the critical value of $Z_{.005}$. Multivariate outliers were checked using Mahalanobis distance. Five cases were above the critical value of chi-square ($\chi^2_{(.001, 23)} = 49.72$) but did not have a large Cook's D value, which suggested they were not influential on the solution. Kaiser-Meyer-Olkin (KMO) measure of sampling adequacy revealed there was a degree of common variance appropriate for conducting a factor analysis (KMO = .78). Each item's Measure of Sampling Adequacy (MSA) was greater than .60, with the lowest being .63. Accordingly, all items were retained for the analysis. Bartlett's test of sphericity indicated the correlation matrix is factorable ($\chi^2 = 1706.92$, $p < .001$). The initial solution indicated there were six factors with eigenvalues greater than 1. Catell's criterion for the scree plot suggested that there might be three or four factors. Consistent with the literature, three factors were extracted, after which there were 63 (24.0%) non-redundant residuals with absolute values greater than .05.

other people think of me"). The third factor was related to private self (e.g., "I'm generally attentive to my inner feelings"). Private self was further broken down to two subscales: self-awareness and self-reflection, which explained 37.12% of the variance, with a correlation of $r = -.49$ between the two subscales. Self-awareness included four items such as "I'm alert to changes in my mood" ($M = 2.91$, $SD = .51$, $\alpha = .54$). Self-reflection consisted of two items such as "I'm always trying to figure myself out" ($M = 2.63$, $SD = .86$, $r = .49$) (See Table 11 for factor loadings, factor correlation, and internal reliability statistics).

Participants were highly efficacious ($M = 3.59$, $SD = .50$, $\alpha = .86$). Anchored between 1 = *strongly agree* and 5 = *strongly disagree*, with high scores reflecting high self-efficacy, participants responded to 17 items such as "When I make plans, I am certain I can make them work." Similarly, participants scored high on self-esteem ($M = 3.04$, $SD = .46$, $\alpha = .86$). Anchored between 1 = *strongly agree* and 4 = *strongly disagree*, with high scores reflecting high self-esteem, participants responded to ten items such as "On the whole, I am satisfied with myself."

Relationship between individual differences and response key. It was predicted in Hypothesis 5 that participants high on self-efficacy, internal locus of control, self-esteem, psychological reactance, dispositional optimism, and self-reflection would be more likely to underestimate their susceptibility to negative health conditions and overestimate their susceptibility to positive ones.

To test Hypothesis 5, mean response key *no* (i.e., one's perception of him/herself as not susceptible to a given health condition) for susceptibility to positive and negative

health conditions was computed. A canonical variate analysis[42] was conducted to examine the number, if any, of the underlying composites between two sets of variables (individual differences and mean response key). The individual differences set included depression inventory, life orientation, health locus of control, psychological reactance, self reflection, self awareness, self consciousness, self efficacy, and self esteem. The response key set included mean *no* for susceptibility to positive and negative health conditions.

The canonical variate analysis showed that the two sets of variables were related on at least one pair of variates (Pillais = .12, $F = 2.27$, $p = .002$). A dimension reduction analysis showed functions 1 through 2 were significant ($\Lambda = .88$, $F = 2.28$, $p = .002$), suggesting that the first pair of variates was related. The first canonical correlation was .30, with an eigenvalue of .10 explaining 77.66% of the variance. Functions 2 through 2 in isolation were not significant ($\Lambda = .97$, $F = 1.16$, $p = .320$).

With a cutoff correlation of .3, data on the first canonical variate showed that the individual difference variables that were correlated with the canonical variate were depression, life orientation, self consciousness, self efficacy, and self esteem. Among the response key variables, susceptibility to negative health conditions correlated with the first canonical variate. This pair of canonical variates indicated that those who scored low on depression inventory (-.75) and self consciousness (-.32), and were high on optimism (as measured by life orientation) (.71), self efficacy (.73), and self esteem (.83) were associated with underestimated perceptions of susceptibility to negative health conditions (.99) (Table 12).

[42] Canonical correlation is a multivariate test used to analyze the relationships between two sets of variables (i.e., an independent variables set and a dependent variables set) (Tabachnik & Fidell, 2007).

Effects of prime, priming method, and valence of health condition on perceptions of susceptibility to health conditions.

Experiment 1 tested the effects of prime and priming method as between-subjects factors, and valence of health condition as a within-subjects factor, on response key and reaction time to susceptibility to positive and negative health conditions. In response to whether one was susceptible (*yes*) or not (*no*) to positive (e.g., healthy weight) and negative (e.g., stroke) health conditions, it was predicted in Hypothesis 1 that participants would respond "*yes*" to positive health conditions and "*no*" to negative ones. Reaction times for "*yes*" responses to positive conditions would be faster than "*no*" responses. Reaction times for "*no*" responses to negative conditions would be faster than "*yes*" responses. Finally, it was predicted that these patterns would be stronger for participants in the self-prime condition than participants in the third-person and neutral conditions. No differences were predicted based on priming method (sub vs. supraliminal conditions) and conscious vs. unconscious conditions.

(1) Effects on response key. A repeated measures analysis of variance was performed with prime and priming method as a between-subjects factor, and valence of health condition (positive, negative) as a within-subjects factor, to detect differences on mean count of *no* response key to health conditions (i.e., "*no*" signifies one's perception of him/herself as not susceptible to a given health condition). Data were screened for fit

of underlying assumptions of normality of sampling distribution, homogeneity of variance-covariance matrices, linearity, and multicolinearity.[43]

Using Wilks' criterion, the interaction term between prime and priming method was not significant ($F(2, 273) = .14$, $p = .868$, $\eta_p^2 = .001$). For the between-subjects factors, no statistically significant differences were found between conditions when response key count was averaged over primes ($F(2, 273) = .83$, $p = .435$, $\eta_p^2 = .006$) or priming method ($F(1, 273) = .33$, $p = .564$, $\eta_p^2 = .001$). When averaged over valence of health condition, however, response key was found to be significant ($F(1, 273) = 3445.80$, $p < .001$, $\eta_p^2 = .927$). As predicted, post hoc results indicated participants identified themselves as not susceptible to negative health conditions ($M = .76$) significantly more as compared to positive ones ($M = .09$) ($p < .001$) (Table 13).

(2) Effects on reaction time combined for yes and no response key. Raw reaction time data for positive and negative health conditions fell between 237 and 71,543 milliseconds. Data points below 300 milliseconds ($n = 2$) and above 5,000 milliseconds ($n = 10$) were deleted. Then, data points beyond 4 *SD* were considered outliers and deleted ($n = 157$), a total of 1.04% data points lost (See Table 14 for descriptive statistics).

[43] Distribution of mean response key was not within the acceptable range of ±2 for skewness and kurtosis. Seventeen cases fell beyond $Z_{.005}$. Six cases were considered multivariate outliers, which exceeded the critical chi-square value of $\chi^2_{(.001, 2)} = 13.81$ but only one had a large Cook's D. Homogeneity of variances was assumed for dependent variables as evidenced by non-significant Levene's tests ($F(5, 273) = 1.04$, $p = .392$ and $F(5, 273) = 1.16$, $p = .329$ for positive and negative conditions, respectively). Homogeneity of the variance –covariance matrices was assumed given equal *n*. Linearity of the relationship between the dependent variables was checked by plotting the two dependent variables. Correlation between two variables was -.16 ($p < .01$), which is not a threat to multicollinearity.

A repeated measures analysis of variance was performed with prime and priming method as between-subjects factors, and valence of health condition as a within-subjects factor, to detect differences on reaction time combined for *yes* and *no* response key. Mean reaction time was calculated for positive and negative health conditions. Data were screened for fit of underlying assumptions regarding normality of sampling distribution, homogeneity of variance-covariance matrices, linearity, and multicolinearity.[44]

For the between-subjects factors, no statistically significant differences were found between conditions based on the prime ($F(2, 273) = 1.16, p = .314, \eta_p^2 = .008$), priming method ($F(1, 273) = 2.12, p = .146, \eta_p^2 = .008$), or their interaction ($F(2, 273) = 2.07, p = .128, \eta_p^2 = .015$). Mean reaction time was 968.27, 1006.93, and 974.98 for self, third-person, and neutral conditions, respectively. Averaged over priming method, mean reaction time to positive health conditions was 979.48 and 1,028.01 for subliminal and supraliminal priming conditions, respectively. For negative health conditions, mean reaction time was 954.99 and 970.50 for the subliminal and supraliminal priming conditions, respectively.

Using Wilks' criterion, there was a significant interaction between priming method and valence of health condition ($F(1, 273) = 4.88, p = .028, \eta_p^2 = .018$). As a within-subjects factor, valence of health condition was significant ($F(1, 273) = 34.56, p <$

[44] Distribution of reaction time within each condition was within the acceptable range of ±2 for skewness and kurtosis. Eight cases fell beyond $Z_{.005}$. No cases exceeded the critical value of $\chi^2_{(.001, 2)} = 13.81$. Homogeneity of variances was assumed for dependent variables as evidenced by non significant Levene's tests ($F(5, 273) = .53, p = .748$ and $F(5, 273) = .80, p = .547$ for reaction time to positive and negative conditions, respectively). Box's M test revealed no problems regarding the homogeneity of the variance-covariance matrices ($\chi^2 = 12.25, p = .677$). Linearity of the relationship between the dependent variables was checked by plotting the two dependent variables. Variables were highly correlated ($r = .80, p < .01$), an expectation in multivariate analysis, and posed no problems.

.001, $\eta_p^2 = .112$). As a follow up to a significant interaction, main effects, with alpha set to .025, revealed no differences between the two priming groups with valence of health condition held constant: positive ($F(1, 277) = 3.80, p = .052$) and negative ($F(1, 277) = .55, p = .458$) traits. Reaction time to positive health conditions was 979.48 and 1,028.01 within the subliminal and supraliminal conditions, respectively. Reaction time to negative health conditions was 954.99 and 970.50 within the subliminal and supraliminal conditions, respectively.

With priming method held constant, main effects revealed differences in reaction time to positive and negative health conditions within the subliminal ($F(1, 277) = 6.79, p = .010$) and supraliminal ($F(1, 277) = 32.28, p < .001$) conditions. Participants were slower in responding to positive health conditions compared to negative ones. This difference in reaction time was statistically significant at $p = .011$ within the subliminal condition and $p < .001$ within the supraliminal condition. In the subliminal priming condition, mean reaction time to positive health conditions was 979.48 and 954.99 to negative conditions. Similarly, in the supraliminal priming condition, mean reaction time to positive health conditions was 1,028.01 and 970.50 to negative ones (Figure 4).[45]

(3) Effects on reaction time split by yes and no response key. A repeated measures analysis was performed with prime and priming method as between-subjects factors, and valence of health condition as a within-subjects factor, to detect differences

[45] To control for familywise Type I error inflation, *p* values were set up at .0125 and .025 for two consecutive contrasts ranked from the smallest to biggest *p* values.

on reaction time split by *yes* and *no* response key.[46] Mean reaction time was calculated for both *yes* and *no* responses to positive and negative health conditions. Data were screened for fit of underlying assumptions regarding normality of sampling distribution, homogeneity of variance-covariance matrices, linearity, and multicolinearity.[47]

For the between-subjects factors, no statistically significant differences were found between conditions based on the prime ($F(2, 142) = 2.18, p = .116, \eta_p^2 = .030$), priming method ($F(1, 142) = 1.13, p = .289, \eta_p^2 = .008$), or their interaction ($F(2, 142) = .90, p = .406, \eta_p^2 = .013$). The within-subjects factor, however, was significant ($F(1.64, 233.66) = 14.97, p < .001, \eta_p^2 = .095$).[48] As predicted, pairwise comparisons, correcting for familywise Type I error, showed that participants perceived themselves as not susceptible to negative health conditions ($M = 931.10$) faster than being susceptible to negative ($M = 1{,}023.29$) and positive ($M = 997.70$) conditions. Participants also perceived themselves as not susceptible to negative conditions faster than they perceived themselves as not susceptible to positive ones ($M = 1{,}051.96$). All pairwise comparisons were significant at $p < .001$ (Figure 5).[49]

---

[46] Reaction time split by *yes* and *no* response keys signifies reaction time for health conditions one identifies as likely to experience or is susceptible to (i.e., time to respond "*yes*") and those one identifies as less likely to experience or is not susceptible to (i.e., time to respond "*no*").

[47] Distribution of reaction time within each condition was within the acceptable range of ±2 for skewness and kurtosis. Fifteen cases fell beyond $Z_{.005}$. One case exceeded the critical value of $\chi^2_{(.001, 4)} = 18.46$. Homogeneity of variances was assumed for all four dependent variables. Box's M test revealed no problems regarding the homogeneity of the variance-covariance matrices ($\chi^2 = 67.86, p = .104$). Linearity of the relationship between the dependent variables was checked by plotting all pairs of dependent variables. Variables were moderately correlated, an expectation in multivariate analysis, and posed no problems.

[48] Mauchly's test indicated that the assumption of sphericity had been violated ($\chi^2 = 176.48, p < .001$). Therefore, degrees of freedom were corrected using Greenhouse-Geisser estimates of sphericity ($\varepsilon = .54$).

[49] This analysis was based on 148 cases with listwise deletion.

Effects of unconsciousness vs. consciousness on perceptions of susceptibility to health conditions.

Performing an analysis on response key and reaction time data with consciousness (unconscious vs. conscious) as a between-subjects factor produced results similar to the ones reported above. For the purposes of this analysis, all six conditions involving a priming procedure were combined into a single unconscious condition and compared to a conscious condition, the seventh condition in this experiment.

For mean count of *no* responses to health conditions (i.e., "*no*" signifies one's perception of him/herself as not susceptible to a given health condition), valence of health condition (positive vs. negative health conditions) was a significant predictor of the dependent variable ($F(1, 323) = 1832.19, p < .001, \eta_p^2 = .850$) whereas membership in the unconscious vs. conscious condition did not result in significant differences between the groups ($F(1, 323) = 2.54, p = .112, \eta_p^2 = .008$) nor was the interaction term between valence of health condition and the between-subjects factor significant ($F(1, 323) = .01, p = .910, \eta_p^2 < .001$).[50]

The valence of the health condition (positive vs. negative health conditions) was a significant predictor of reaction time. Combined for *yes* and *no* responses, valence of health condition was significant ($F(1, 323) = 11.67, p < .001, \eta_p^2 = .035$) whereas membership in unconscious vs. conscious group did not have an effect ($F(1, 323) = 2.75, p = .098, \eta_p^2 = .008$) nor did the interaction term between the within- and between-

[50] Distribution of response key within each condition was not within the acceptable range of ±2 for skewness and kurtosis. Homogeneity of variances was assumed for dependent variables as evidenced by non-significant Levene's tests ($F(1, 323) = .01, p = .893$ and $F(1, 323) = .001, p = .980$ for positive and negative conditions, respectively). With unequal *n*, Box's M test revealed a problem regarding the homogeneity of the variance-covariance matrices ($\chi^2 = 8.36, p = .042$).

subjects factors ($F(1, 323) = .91, p = .342, \eta_p^2 = .003$).[51] Similarly, split by *yes* and *no* responses, valence of health condition was a significant predictor of reaction time ($F(1.71, 280.85) = 8.07, p = .001, \eta_p^2 = .047$). On the contrary, membership in unconscious vs. conscious group did not have a significant effect ($F(1, 164) = 3.02, p = .084, \eta_p^2 = .018$) nor did the interaction term between the within- and between-subjects factors ($F(1.71, 280.85) = .94, p = .380, \eta_p^2 = .006$).[52]

Finally, a semantic differential affect/arousal index ($\alpha = .84$) was similar across experimental groups. The interaction term ($F(2, 273) = .33, p = .715$), prime ($F(2, 273) = .84, p = .432$), and priming method ($F(1, 273) = .004, p = .947$) were not significant. Measured at the end of experimental sessions, means were around the mid point of the semantic differential index: for the self ($M = 1.51$, $SD = 1.40$), third-person ($M = 1.39$, $SD = 1.53$), and neutral ($M = 1.24$, $SD = 1.32$) primes, averaged over prime conditions, and for the subliminal ($M = 1.39$, $SD = 1.46$) and supraliminal ($M = 1.38$, $SD = 1.37$) conditions, averaged over priming method conditions. Similarly, there were no differences when the unconscious conditions was compared to the conscious one ($F(1, 323) = .005, p = .945$). Means were 1.38 ($SD = 1.42$) and 1.40 ($SD = 1.66$), respectively.

---

[51] Distribution of reaction time within each condition was within the acceptable range for skewness and kurtosis. Homogeneity of variances was assumed for dependent variables as evidenced by non-significant Levene's tests ($F(1, 323) = 3.31, p = .070$ and $F(1, 323) = 2.67, p = .103$ for positive and negative conditions, respectively). Box's M test revealed no problems regarding the homogeneity of the variance-covariance matrices ($\chi^2 = 2.28, p = .523$).

[52] Performed on 166 cases, distribution of reaction time within each condition was within the acceptable range for skewness and kurtosis. Homogeneity of variances was assumed for all dependent variables as evidenced by non-significant Levene's tests. Box's M test revealed no problems regarding the homogeneity of the variance-covariance matrices ($\chi^2 = 13.65, p = .256$). Mauchly's test indicated that the assumption of sphericity had been violated ($\chi^2 = 180.93, p < .001$). Therefore, degrees of freedom were corrected using Greenhouse-Geisser estimates of sphericity ($\varepsilon = .51$).

This indicated that any differences between the experimental groups should not be attributed to a differential affect/arousal status.[53]

In sum, Experiment 1 provided partial support for Hypothesis 1. The hypothesized main effect of valence of health condition was supported whereas the main effect of self primes in biasing perceptions of susceptibility to health conditions was not. Consistent with the literature, no differences were found based on priming method (sub or supraliminal) (Bargh & Chartrand, 2000). Finally, no differences were found between the unconscious and conscious conditions in response key and reaction time as dependent variables.

Replicating the results of Pilot Study 3, Experiment 1 showed that the valence of the health condition was a powerful predictor of response key and reaction time. Participants perceived themselves as not susceptible to negative (e.g., diabetes) as opposed to positive (e.g., healthy heart) health conditions. In terms of reaction times, participants responded faster to negative health conditions compared to positive ones. Further, not being susceptible to a negative health condition was the easiest of decisions participants had to make (as evidenced by faster reaction time). In contrast, identifying oneself as being susceptible to negative and positive conditions as well as not being susceptible to positive conditions was more difficult (as evidenced by slower reaction time).

Finally, Experiment 1 partially supported Hypothesis 5. As predicted, low levels of depression and self-consciousness, and high levels of optimism, self-efficacy, and self-

[53] The distribution of affect/arousal index for each condition was within the acceptable range of ±2 for skewness and kurtosis. Three cases exceeded the critical value of $Z_{.005}$. Equal variances were not assumed with prime and priming method as fixed factors (Levene's test: $F(2, 470) = 2.37$ $p = .038$) but were assumed with conscious vs. unconscious as a factor (Levene's test: $F(1, 323) = 2.18$ $p = .140$).

esteem were associated with underestimated perceptions of susceptibility to negative health conditions. Internal locus of control, psychological reactance, and self-reflection were not associated with underestimation of health risks as hypothesized.

Experiment 2: Efficiency of underestimation of personal health risk perceptions

Experiment 2 tested the hypothesis that underestimation of health risk perceptions is an efficient process, one that occurs when people are mentally taxed. The experiment was a 3 (prime: self-identity, third-person, neutral words) x 2 (cognitive load: high, low) between-subjects design.

A total of 476 participants were recruited. They were randomly assigned to one of six experimental conditions. The majority of participants were females (n = 366, 76.9%) and right-handed (n = 428, 89.9%). Mean age was 21.68 (SD = 2.33). Participants were recruited for course credit (n = 225, 47.3%) or a $10 monetary incentive ($n$ = 251, 52.7%).

Data for Experiment 2 included measures of individual differences (e.g., self-efficacy) as well as response key and reaction time data for susceptibility to and severity of 50 health conditions. First, baseline measures are described. Second, descriptive statistics and reliability of individual difference variables are presented along with their effects on response key. Third, response key and reaction time data are analyzed to detect differences between the experimental conditions based on prime and cognitive load, the two manipulated variables in the study.

Baseline measures.

A series of univariate ANOVAs, with cognitive load and prime as fixed factors, showed no differences between the experimental groups on baseline (i.e., practice trial)

response key accuracy. They did differ, however, on baseline reaction time. For baseline response key accuracy, there were no differences between the experimental conditions in total correct response key as a dependent variable. The interaction term ($F(2, 470) = .66$, $p = .514$), cognitive load ($F(1, 470) = .71$, $p = .399$), and prime ($F(2, 470) = .12$, $p = .884$) were not significant. Participants in the self ($M = 19.61$, $SD = 1.17$), third-person ($M = 19.72$, $SD = .89$) and neutral ($M = 19.60$, $SD = 1.39$) conditions correctly identified practice-trial items as animals/non-animal. Averaged across cognitive load, means were 19.59 ($SD = 1.34$) and 19.69 ($SD = .96$) for the high and low load conditions, respectively.[54]

Raw baseline reaction time data fell between 303 and 10,905 milliseconds. Data points exceeding 5 seconds were excluded ($n = 2$). Then, data points that were 4 *SD* away from the mean were considered outliers and excluded ($n = 87$), a .91% total data points lost. A univariate ANOVA performed on log-transformed reaction time data showed the interaction term between cognitive load and prime ($F(2, 470) = 1.16$, $p = .311$) and cognitive load ($F(1, 470) = .001$, $p = .974$) were not significant.[55] Mean reaction time for participants in the high load condition was 665.27 ($SD = 1.12$) whereas reaction time for those in the low load condition was 665.11 ($SD = 1.13$).

There were significant differences, however, in baseline reaction time based on prime ($F(2, 470) = 6.68$, $p = .001$, $\eta_p^2 = .028$). Post hoc analysis, corrected for familywise

[54] Total correct response key was transformed using 1/(K-X) formula after which skewness and kurtosis were within the acceptable ±2. Ten cases exceeded the critical value of $Z_{.005}$. Equal variances were assumed as evidenced by a non-significant Levene's test ($F(5, 470) = 2.02$, $p = .073$).

[55] The distribution of log-transformed reaction time data was within the acceptable range of ±2 for skewness and kurtosis within each experimental condition. Equal variances were assumed as evidenced by a non-significant Levene's test ($F(5, 470) = .77$, $p = .565$).

Type I error inflation using the FDR approach, showed two significant differences. Participants in the self condition ($M = 684.38$, $SD = 1.13$) had significantly slower reaction time compared to those in the third-person ($M = 655.39$, $SD = 1.12$, $p = .004$) and neutral ($M = 656.14$, $SD = 1.12$, $p = .005$) conditions.

Individual differences.

Individual difference variables were screened for normality of distributions and outliers. There were no missing values (See Table 15 for descriptive statistics and correlation matrix).

Participants scored low on the depression inventory ($M = 1.35$, $SD = .28$, $\alpha = .83$).[56] Consistently, participants were optimistic, as measured by the life orientation test ($M = 2.61$, $SD = .62$, $\alpha = .82$).

Participants showed slightly high levels of internal health locus of control ($M = 3.79$, $SD = .50$, $\alpha = .62$). Consistent with the literature, results revealed health locus of control had two underlying factors, which explained 25.02% of the variance.[57] The first factor was related to internal locus of control whereas the second factor was related to

[56] The reported reliability is for 20 items of the depression inventory. A weight loss item was excluded because it reduced the sample size from 476 to 159 (those who were trying to loose weight) with listwise deletion. When the weight loss item was included, reliability was .85 ($N = 159$).

[57] Evaluation of normality of distribution, outliers, and factorability were satisfactory. All variables were within the acceptable ±2 for skewness and kurtosis. Twenty-six cases exceeded the critical value of $Z_{.005}$. Multivariate outliers were checked using Mahalanobis distance. Five cases were above the critical value of chi-square ($\chi^2_{(.001, 11)} = 31.26$) but did not have a large Cook's D value, which suggested they were not influential on the solution. Kaiser-Meyer-Olkin (KMO) measure of sampling adequacy revealed there was a degree of common variance appropriate for conducting a factor analysis (KMO = .73). Each item's Measure of Sampling Adequacy (MSA) was greater than .60, with the lowest being .65. Accordingly, all items were retained for the analysis. Bartlett's test of sphericity indicated the correlation matrix is factorable ($\chi^2 = 611.81$, $p < .001$). The initial solution indicated there were two factors with eigenvalues greater than 1. Similarly, Catell's criterion for the scree plot suggested that there might be two factors. With the extraction of two factors, there were 13 (23.0%) non-redundant residuals with absolute values greater than .05.

external locus of control. Based on the analysis, five items reflected internal locus of control ($\alpha = .67$), and six items reflected external locus of control ($\alpha = .56$), with a correlation of .13 between the two factors (See Table 10 for factor loadings, factor correlation, and internal reliability statistics).

Participants were slightly reactant ($M = 3.00$, $SD = .45$, $\alpha = .78$). Psychological reactance was positively correlated with self-consciousness and negatively with self-efficacy and self-esteem.

Participants' scores were roughly at the mid point of the self-consciousness scale ($M = 2.49$, $SD = .39$, $\alpha = .78$). Results revealed self-consciousness had three underlying factors, which explained 31.59% of the variance.[58] The first factor was related to social anxiety (e.g., "I feel anxious when I speak in front of people"). The second was related to public self (e.g., "I'm concerned about what other people think of me"). The third factor was related to private self (e.g., "I'm generally attentive to my inner feelings"). Private self was further broken down to two subscales: self-reflection and self-awareness, which explained 34.44% of the variance, with a correlation of .42. Self-reflection included four items such as "I reflect about myself a lot" ($M = 2.72$, $SD = .66$, $\alpha = .69$). Self-awareness included four items such as "I'm alert to changes in my mood" ($M = 2.92$, $SD = .48$, $\alpha =$

[58] Evaluations of normality of distribution, outliers, and factorability were satisfactory. All individual items were within the acceptable ±2 for skewness and kurtosis. One hundred and sixty seven cases exceeded the critical value of $Z_{.005}$. Ten cases exceeded the critical value of $\chi^2_{(.001, 23)} = 49.72$ but did not have large Cook's D values. Kaiser-Meyer-Olkin (KMO) measure of sampling adequacy revealed there was a degree of common variance and hence the sample was appropriate for conducting a Principal Axis Factoring (KMO = .81). Additionally, each item's Measure of Sampling Adequacy (MSA) was greater than .60, with the lowest being .67. Accordingly, all items were retained for the analysis. Bartlett's test of sphericity indicated the correlation matrix is factorable ($\chi^2 = 2590.39$, $p < .001$). The initial solution indicated there were five factors with eigenvalues greater than 1. Catell's criterion for the scree plot suggested that there might be three factors. With the extraction of three factors, there were 59 (23.0%) non-redundant residuals with absolute values greater than 0.05.

.60) (See Table 16 for factor loadings, factor correlation, and internal reliability statistics).

Finally, participants scored high on self efficacy ($M = 3.66$, $SD = .46$, $\alpha = .84$) and self esteem ($M = 3.08$, $SD = .44$, $\alpha = .86$).

Relationship between individual differences and response key. In Hypothesis 4, it was predicted that participants would overestimate their susceptibility to highly prevalent health conditions with which they have had personal experience and underestimate their susceptibility to controllable health conditions, and ones for which exempt beliefs and stereotypes of a typical person who suffers the health condition were salient. In Hypothesis 5, it was predicted that participants high on self-efficacy, internal locus of control, self-esteem, psychological reactance, dispositional optimism, and self-reflection would be more likely to underestimate their susceptibility to and severity of negative health conditions and overestimate their susceptibility to positive ones.

To test Hypotheses 4 and 5, mean response key *no* for both susceptibility to (i.e., one's perception of him/herself as not susceptible to a given health condition) and severity of (i.e., one's perception of a given health condition as not severe if s/he experiences it) three clusters of health conditions was computed (based on Pilot Study 1). A canonical variate analysis was conducted to examine the number, if any, of the underlying composites between two sets of variables (individual differences and mean response key) and their nature. The individual differences set included depression inventory, life orientation, health locus of control, psychological reactance, self reflection, self awareness, self consciousness, self efficacy, and self esteem. The response

key set included mean response key *no* to susceptibility and severity of three health condition clusters.

The canonical variate analysis showed that the two sets of variables were related on at least one pair of variates (Pillais= .20, F= 1.79, $p < .001$). A dimension reduction analysis showed functions 1 through 6 were significant ($\Lambda = .81$, $F = 1.80$, $p < .001$), and functions 2 through 6 were significant ($\Lambda = .88$, $F = 1.43$, $p = .039$), suggesting that two pairs of variates were related. The first canonical correlation was .28, with an eigenvalue of .08 explaining 41.44% of the variance. The second canonical correlation was .23, with an eigenvalue of .05 explaining 26.67% of the variance.

With a cutoff correlation of .3, data on the first canonical variate showed the individual differences variables that were correlated with the canonical variate were depression, self esteem, life orientation, self-consciousness, self-efficacy, and health locus of control. Among the response key variables, susceptibility to Cluster 2 and 3 health conditions correlated with the first canonical variate. This pair of canonical variates indicated that those who were depressed (.84) and self conscious (.46), and low on self esteem (-.67), life orientation (-.60), self efficacy (-.46), and health locus of control (-.40) were associated with increased perceived susceptibility to Cluster 2 health conditions (e.g., diabetes, heart failure) (-.64) and Cluster 3 health conditions (e.g., flu, diarrhea) (-.34).

Data on the second canonical variate showed that those who were low on life orientation (-.38) and who were self conscious (.33) were associated with decreased perceived severity of Cluster 1 health conditions (e.g., brain tumors) (.31) and

susceptibility to Cluster 3 health conditions (e.g., flu) (.32) but increased severity of health conditions in Cluster 3 (-.78) (Table 17).

Effects of prime and cognitive load on susceptibility to and severity of health conditions.

Anchored between 1 = *extremely easy* and 9 = *extremely difficult*, a manipulation check confirmed participants in the high cognitive load perceived memorizing an 8-digit number as more difficult ($M = 4.06$, $SD = 1.88$) compared to participants in the low load condition ($M = 1.67$, $SD = .97$) who memorized a 2-digit number ($t(356.19) = 17.37$, $p < .001$).[59]

Participants in the low load condition recalled the four numbers with 100% ($n = 238$), 99.6% ($n = 237$), 98.7% ($n = 235$), and 97.1% ($n = 231$) success across the four blocks. Participants in the high load had a lower success rate at 81.1% ($n = 193$), 78.2% ($n = 186$), 74.4% ($n = 177$), and 77.7% ($n = 185$). Inclusion of response key and reaction time data in the analysis was contingent upon successful recall of the number to ensure the cognitive load manipulation was at work.

In response to whether one were susceptible (*yes*) or not (*no*) to negative health conditions and whether these conditions would be severe (*yes*) or not (*no*) if one experienced them, it was predicted in Hypothesis 2a that participants would respond "*no*" when primed with self-identity words and when under high cognitive load. Reaction times for "*no*" responses would be faster than "*yes*" responses. It was predicted in

[59] Distribution of the dependent variable was within the acceptable range of ± 2 for each condition except for the kurtosis in the low cognitive load condition. Equal variances were not assumed as evidenced by a significant Levene's test ($F(1, 474) = 127.08$, $p < .001$).

Hypothesis 2b that these patterns would be stronger for participants in the self-prime condition when under high (as opposed to low) cognitive load.

(1) Effects of prime and cognitive load on response key. Six univariate ANOVAs were performed with cognitive load and prime as fixed factors.[60] Dependent variables were mean response key *yes* for susceptibility to and severity of three health condition clusters[61] (Table 18).

Cluster 1 health conditions. Perceived susceptibility to Cluster 1 health conditions (e.g., brain tumors) was affected by the prime ($F(2, 470) = 4.75, p = .009, \eta_p^2 = .020$) but not by cognitive load ($F(1, 470) = .05, p = .824, \eta_p^2 < .001$) nor the interaction term between the prime and load ($F(2, 470) = .45, p = .632, \eta_p^2 = .002$).[62] As predicted, pairwise comparisons, adjusted for familywise Type I error using the FDR approach, showed that participants in the self condition significantly underestimated the likelihood of contracting Cluster 1 health conditions ($M = .14$) compared to the neutral condition ($M = .23$) ($p = .006$). The third-person condition ($M = .19$) was not statistically different from either the self ($p = .230$) or neutral ($p = .322$) conditions. Means were .18 for the high cognitive load condition and .19 for the low condition (Figure 6).

---

[60] A decision in favor of a series of univariate over multivariate analysis of variance was made because multivariate analysis of variance best suits dependent variables that are highly negatively correlated or moderately correlated |.6| in either direction. MANOVA is considered wasteful if the variables are not correlated or are highly positively correlated (Tabachnik & Fidell, 2007). The correlations between mean response key to susceptibility and severity of health condition clusters included a mix of uncorrelated, highly and moderately positively correlated variables.

[61] Mean response key *yes* for susceptibility to health condition clusters signifies one's perception of him/herself as susceptible to health conditions that belonged to a given cluster. Mean response key *yes* for severity of health condition clusters signifies one's perception of health conditions that belonged to a given cluster as severe if s/he experiences it.

[62] Distribution of response key was within the acceptable range for skewness and kurtosis within each load and prime condition. Twelve cases exceeded critical value of $Z_{.005}$. Homogeneity of variances was assumed given equal *n* although Levene's test was significant ($F(5, 470) = 4.69, p < .001$).

As for the severity of Cluster 1 health conditions, the analysis showed that perceived severity was affected by the interaction term between cognitive load and prime ($F(2, 470) = 4.77, p = .009, \eta_p^2 = .020$), and load ($F(1, 470) = 46.15, p < .001, \eta_p^2 = .089$), but not by the prime ($F(2, 470) = .86, p = .422, \eta_p^2 = .004$).[63]

As a follow up to a significant interaction, main effects, with alpha set to .025, showed that within the high cognitive load condition, prime conditions were not statistically different from each other ($F(2, 470) = 1.12, p = .327$). Means were .54, .60, and .55 for the self, third-person, and neutral conditions, respectively. Within the low cognitive load condition, primes were statistically different ($F(2, 470) = 4.51, p = .011$). Pairwise comparisons, adjusted for familywise Type I error using the FDR approach, showed participants in the third-person condition ($M = .66$) significantly underestimated the severity of the diseases compared to those in the neutral condition ($M = .79$) ($p = .003$). Means for the self condition ($M = .74$) was not statistically different from those in the third-person ($p = .058$) or neutral ($p = .821$) conditions.

Further, cognitive load conditions differed significantly from each other within the self ($F(1, 470) = 22.62, p < .001$) and neutral ($F(1, 470) = 31.08, p < .001$) conditions but not within the third-person ($F(1, 470) = 2.07, p = .150$) condition. As predicted, pairwise comparisons, adjusted for familywise Type I error, showed participants primed with self-identity words underestimated the severity of Cluster 1 health conditions when under high load ($M = .54$) compared to those under low load ($M = .74$) ($p < .001$). Similarly, participants primed with neutral words underestimated the severity of diseases

[63] Distribution of response key was within the acceptable range for skewness and kurtosis with each experimental condition. No case exceeded critical value of $Z_{.005}$. Homogeneity of variances was assumed given equal *n* although Levene's test was significant ($F(5, 470) = 13.53, p < .001$).

when under high (M = .55) as opposed to low (M = .79) cognitive load (p < .001). Finally, participants' perceived severity of diseases did not differ under high (M = .60) and low (M = .66) conditions when primed with third-person words (p = .150) (Figure 7).

Cluster 2 health conditions. For susceptibility and severity to Cluster 2 health conditions, and susceptibility to Cluster 3 health conditions, cognitive load had an effect whereas the prime and the interaction term between cognitive load and prime did not have an effect. Perceived susceptibility to Cluster 2 health conditions (e.g., asthma, allergies) were different based on cognitive load ($F(1, 470) = 5.09, p = .024, \eta_p^2 = .011$) but not by prime ($F(2, 470) = 2.53, p = .081, \eta_p^2 = .011$) or load and prime interaction ($F(2, 470) = 1.10, p = .333, \eta_p^2 = .005$).[64] As predicted, participants in the high cognitive load condition underestimated the likelihood of contracting Cluster 2 health conditions (M = .28) compared to those in the low load condition (M = .32) (p = .024) (Figure 8).

Similarly, cognitive load had a significant effect on perceived severity of Cluster 2 health conditions ($F(1, 470) = 51.57, p < .001, \eta_p^2 = .099$) whereas the prime ($F(2, 470) = 2.68, p = .069, \eta_p^2 = .011$) and the interaction term between load and prime ($F(2, 470) = 2.03, p = .132, \eta_p^2 = .009$) did not have an effect.[65] As predicted, participants in the high cognitive load condition underestimated the severity of Cluster 2 health conditions (M = .39) compared to those in the low load condition (M = .53) (p < .001) (Figure 9).

---

[64] Distribution of response key was within the acceptable range of ±2 for skewness and kurtosis within each condition. Six cases exceeded the critical value of $Z_{.005}$. Homogeneity of variances was assumed given equal n. Levene's test was not significant ($F(5, 470) = 1.89, p = .094$).

[65] Distribution of response key was within the acceptable range of ±2 within each condition. No case exceeded $Z_{.005}$. Homogeneity of variances was assumed given equal n although Levene's test was significant ($F(5, 470) = 4.01, p < .001$).

Cluster 3 health conditions. Finally, perceived susceptibility to Cluster 3 health conditions (e.g., flu) was significantly different across the cognitive load conditions ($F(1, 470) = 20.94, p < .001, \eta_p^2 = .043$) but not across prime conditions ($F(2, 470) = .91, p = .403, \eta_p^2 = .004$). The interaction term between cognitive load and prime was not significant ($F(2, 470) = .02, p = .974, \eta_p^2 < .001$).[66] As predicted, participants in the high load conditions significantly underestimated the likelihood of contracting Cluster 3 health conditions ($M = .53$) compared to those in the low load condition ($M = .65$) ($p < .001$) (Figure 10). Perceived severity, on the other hand, was similar across cognitive load and prime conditions. The interaction term ($F(2, 470) = .48, p = .617, \eta_p^2 = .002$), cognitive load ($F(1, 470) = 2.22, p = .137, \eta_p^2$ .005), and prime ($F(2, 470) = 1.04, p = .354, \eta_p^2 = .004$) did not affect perceived severity of Cluster 3 health conditions[67] (Figure 11).

If alpha were set at .008 (for six univariate analyses) to correct for familywise Type I error, cognitive load would be the sole factor causing participants to underestimate the severity of Cluster 1 and 2 health conditions and susceptibility to Cluster 3 health conditions.

(2) Effects of prime and cognitive load on reaction time combined for yes and no responses.

Susceptibility reaction time. A repeated measures analysis of variance was performed with prime and cognitive load as between-subjects factors, and reaction time

[66] Distribution of response key was within the acceptable range of ±2 within each condition. No case exceeded $Z_{.005}$. Homogeneity of variances was assumed given equal *n* although Levene's test was significant ($F(5, 470) = 3.75, p = .002$).

[67] Distribution of response key was within the acceptable range for skewness and kurtosis. Fourteen cases exceeded $Z_{.005}$. Homogeneity of variances was assumed given equal *n*. Levene's test was not significant ($F(5, 470) = 1.45, p = .203$).

for susceptibility to Cluster 1, 2, and 3 health conditions as a repeated measure, to detect differences on reaction time combined for the *yes* and *no* response key. Mean reaction time was calculated for each of three health condition clusters. Data were screened for fit of underlying assumptions regarding normality of sampling distribution, homogeneity of variance-covariance matrices, linearity, and multicolinearity.[68]

For the between-subjects factors, no statistically significant differences were found between conditions based on the prime ($F(2, 452) = 2.43, p = .088, \eta_p^2 = .011$), or the interaction term between prime and cognitive load ($F(2, 452) = .663, p = .516, \eta_p^2 = .003$). Mean reaction time was 1,069.05, 1,047.12, and 1,018.59 for participants in the self, third-person, and neutral conditions, respectively. There were differences, however, based on cognitive load ($F(1, 452) = 11.05, p < .001, \eta_p^2 = .024$). Pairwise comparisons show that reaction time in the high cognitive load condition ($M = 1{,}076.46$) was significantly slower than those in the low load condition ($M = 1{,}013.91$) ($p = .001$) (Figure 12).

Within-subjects effects showed a significant 3-way interaction between prime, cognitive load, and repeated measure (i.e., reaction time for susceptibility to Clusters 1, 2, and 3) ($F(2.84, 642.79) = 3.10, p = .028, \eta_p^2 = .014$).[69] As a follow up to a significant 3-way interaction, simple interaction effects showed reaction time was different for health

[68] Distribution of reaction time was within the acceptable range for skewness and kurtosis within each condition. Twelve cases fell beyond $Z_{.005}$. Three cases exceeded the critical value of $\chi^2_{(.001, 3)} = 16.26$. Homogeneity of variances was assumed for dependent variables as evidenced by non significant Levene's tests ($F(5, 452) = .75, p = .586$, $F(5, 452) = 2.156, p = .058$, and $F(5, 452) = .249, p = .940$ for Clusters 1, 2, and 3, respectively). Box's M test revealed a problem regarding the homogeneity of the variance-covariance matrices ($\chi^2 = 46.52, p = .033$). Linearity of the relationship between the dependent variables was checked by plotting all pairs of dependent variables. Variables were highly correlated and posed no problems.

[69] Mauchly's test indicated that the assumption of sphericity had been violated ($\chi^2 = 235.18, p < .001$). Therefore, degrees of freedom were corrected using Greenhouse-Geisser estimates of sphericity ($\varepsilon = .71$).

condition clusters only for participants primed with self-identity words when under high cognitive load ($F(2, 904) = 3.78, p = .023$). Mean reaction times were 1,117.89 for Cluster 1 health conditions, 1,097.99 for Cluster 2 health conditions, and 1,065.12 for Cluster 3 health conditions. Bonferroni pairwise comparisons did not show statistical differences in reaction time by health condition cluster. Running three paired-tests, however, revealed that reaction time to Cluster 1 health conditions was significantly slower compared to reaction time to Cluster 3 health conditions ($t(71) = 2.09, p = .040$). Reaction time to Cluster 1 was not different from reaction time to Cluster 2 ($t(71) = 1.52$, $p = .132$). Reaction time did not differ between Cluster 2 and Cluster 3 health conditions ($t(71) = 1.56, p = .121$).

All other simple interactions were not significant: Reaction time to health condition clusters did not differ for participants primed with third-person ($F(2, 904) = 1.89, p = .152$) or neutral ($F(2, 904) = .90, p = .910$) when under high load. Similarly, reaction time to health condition clusters did not differ for participants primed with self-identity ($F(2, 904) = 1.33, p = .265$), third-person ($F(2, 904) = 1.36, p = .257$), or neutral ($F(2, 904) = .32, p = .728$) words when under low cognitive load.

Severity reaction time. A repeated measures analysis of variance was performed with prime and cognitive load as between-subjects factors, and reaction time for severity of Cluster 1, 2, and 3 health conditions as a repeated measure, to detect differences on reaction time combined for the *yes* and *no* response key. Mean reaction time was calculated for each of the three health condition clusters. Data were screened for fit of

underlying assumptions regarding normality of sampling distribution, homogeneity of variance-covariance matrices, linearity, and multicolinearity.[70]

For the between-subjects factors, reaction time to health condition clusters differed based on the interaction term between prime and load ($F(2, 446) = 3.15, p = .044, \eta_p^2 = .014$), and the prime ($F(2, 446) = 3.10, p = .046, \eta_p^2 = .014$) but not by cognitive load ($F(1, 446) = 2.19, p = .139, \eta_p^2 = .005$). As a follow up to a significant interaction, simple effects set at .01 for five simple effects, showed that cognitive load conditions were statistically different within the neutral prime condition ($F(3, 444) = 3.67, p = .012$) but not within the third-person ($F(3, 444) = .45, p = .713$) or self ($F(3, 444) = 2.34, p = .073$) prime conditions. Mean reaction time averaged across all health condition clusters was 1,035.14 in the high load and 959.40 in the low load condition for participants primed with neutral words ($p = .004$). For those primed with third-person words, reaction time was 1,039.92 and 1,016.24 in the high and low conditions, respectively. For those primed with self-identity words, reaction time was 1,032.76 and 1,059.25 in the high and low conditions, respectively.

Effects of the primes were evident within the low cognitive load ($F(6, 888) = 2.94, p = .007$) but not the high load ($F(6, 888) = 1.89, p = .076$) conditions. Pairwise comparisons showed that participants in the self condition ($M = 1{,}059.25$) were significantly slower than those in the neutral condition ($M = 959.40$) ($p = .001$) but did

[70] Distribution of reaction time was within the acceptable range for skewness and kurtosis within each condition. Thirty cases fell beyond $Z_{.005}$. Four cases exceeded the critical value of $\chi^2_{(.001, 3)} = 16.26$ but did not have a large Cook's D. Homogeneity of variances was assumed for Cluster 1 ($F(5, 446) = .30, p = .909$) and Cluster 2 ($F(5, 446) = .97, p = .430$) health conditions but not for Cluster 3 ($F(5, 446) = 3.09, p = .009$). Box's M test revealed a problem regarding the homogeneity of the variance-covariance matrices ($\chi^2 = 74.26, p < .001$). Linearity of the relationship between the dependent variables was checked by plotting all pairs of dependent variables. Variables were highly correlated and posed no problems.

not differ from those primed with third-person words (M = 1,016.24) (p = .346). The latter group did not differ from the neutral condition either (p = .097) (Figure 13).

Within-subjects effects show a significant interaction between prime and repeated measure (i.e., reaction time for severity of Cluster 1, 2, and 3 health conditions) (F(3.095, 892) = 3.26, p = .020, η_p^2 = .014).[71] As a follow up to a significant interaction, simple interaction effects showed reaction time was different for health condition clusters within the self (F(2, 989) = 18.00, p < .001) and neutral (F(2, 898) = 3.57, p = .029) prime conditions but not within the third-person condition (F(2, 898) = 2.96, p = .052). Within the self-prime condition, pairwise comparisons showed that reaction time to Cluster 3 health conditions (M = 1,006.93) was significantly faster than Cluster 2 (M = 1,083.92) (p < .001) and Cluster 1 (M = 1,051.96) (p = .004). Reaction time to Cluster 1 was significantly faster than Cluster 2 (p = .002). Similarly, within the neutral-prime condition, reaction time to Cluster 1 health conditions (M = 986.27) was significantly faster than reaction time to Cluster 2 (M= 1,013.91) (p = .006). However, reaction time to Cluster 1 was not different from Cluster 3 (M = 990.83). Cluster 2 was also not significantly different from reaction time to Cluster 3.

Susceptibility – severity relationship.

A closer look at Table 18 showed an inverted relationship between perceived susceptibility to a disease and perceived severity. For Cluster 1 health conditions (e.g., brain tumors), the mean perceived susceptibility (i.e., response key *yes* signifies one's perception s/he was susceptible to health conditions that belonged to a given cluster) was .19 whereas perceived severity (i.e., response key *yes* signifies one's perception health

[71] Mauchly's test indicated that the assumption of sphericity had been violated (χ^2 = 153.87, p < .001). Therefore, degrees of freedom were corrected using Greenhouse-Geisser estimates of sphericity (ε = .77).

conditions that belonged to a given cluster would be severe if s/he experienced it) was .64. On the contrary, mean perceived susceptibility to Cluster 3 health conditions (e.g., flu) was .59 with .14 severity. Perceived susceptibility to and severity of Cluster 2 health conditions (e.g., diabetes) fell in between Clusters 1 and 2, with perceived susceptibility at .30 and severity at .46 (Figure 14).

Similarly, a closer look at Table 19 showed differences in reaction time of susceptibility to and severity of health condition clusters. For Clusters 1 and 2, participants were faster dismissing the possibility that they were at risk for these health conditions than acknowledging they were. For severity, participants were faster acknowledging that Cluster 1 and 2 health conditions were severe than dismissing that they were not. Reaction time to Cluster 3 showed a reverse pattern. Participants were faster acknowledging they were at risk for these health conditions than dismissing they were not. For severity, participants were faster dismissing Cluster 3 health conditions were not severe than acknowledging they were (Figure 15).

To further probe the relationship between susceptibility and severity to different health condition clusters, mean reaction time was computed for the susceptibility to and severity of each health conditions cluster split by *yes* and *no* response key. Three repeated measures analyses of variance were performed, one for each cluster with four dependent variables, as a repeated measure: mean reaction time for susceptibility to health conditions cluster for (1) *yes*, and (2) *no* response keys, and mean reaction time for

severity of health conditions cluster for (3) *yes*, and (4) *no* response keys.[72] Cognitive load and primes were fixed factors.

Cluster 1. Results for Cluster 1 showed the two fixed factors, load ($F(1, 249) = .61, p = .435, \eta_p^2 = .002$) and prime ($F(2, 249) = 2.38, p = .094, \eta_p^2 = .019$), and their interaction ($F(2, 249) = .76, p = .466, \eta_p^2 = .006$) did not have an effect.[73] The repeated measure, however, had an effect ($F(2.862, 712.717) = 44.45, p < .001, \eta_p^2 = .151$).[74] Pairwise comparisons, adjusted for familywise Type I error, showed participants were slower in acknowledging they were susceptible to Cluster 1 health conditions (M = 1,250.25) than dismissing they were not (M = 1,180.32) ($p = .024$). On the contrary, participants were significantly faster acknowledging Cluster 1 health conditions were severe (M = 1,028.01) than dismissing they were not severe (M = 1,191.24) ($p < .001$). Further, participants were significantly slower in acknowledging they were susceptible to Cluster 1 health conditions than acknowledging these health conditions were severe ($p < .001$). And, participants were significantly slower in dismissing they were not susceptible

[72] Reaction time for susceptibility split by *yes* and *no* response keys signifies reaction time for health conditions one identifies as likely to experience or is susceptible to (i.e., time to respond "*yes*") and those one identifies as less likely to experience or is not susceptible to (i.e., time to respond "*no*"). For severity, reaction time split by *yes* and *no* response keys signifies reaction time for health conditions one identifies as severe if s/he experiences it (i.e., time to respond "*yes*") and those one identifies as not severe (i.e., time to respond "*no*").

[73] Distribution of reaction time was within the acceptable range for skewness and kurtosis within each condition. Twenty-three cases fell beyond $Z_{.005}$. Two cases exceeded the critical value of $\chi^2_{(.001, 4)} = 18.46$ but did not have a large Cook's D. Homogeneity of variances was assumed for all dependent variables except severity for response key *yes* ($F(5, 249) = 2.55, p = .028$). Box's M test revealed a problem regarding the homogeneity of the variance-covariance matrices ($\chi^2 = 109.92, p < .001$). Linearity of the relationship between the dependent variables was checked by plotting all pairs of dependent variables. Variables were moderately correlated.

[74] Mauchly's test indicated that the assumption of sphericity had been violated ($\chi^2 = 18.41, p < .001$). Therefore, degrees of freedom were corrected using Greenhouse-Geisser estimates of sphericity ($\varepsilon = .95$).

to Cluster 1 health conditions than acknowledging the health conditions were severe ($p <$ .001) (Figure 16).

Cluster 2. Results for Cluster 2 showed that load ($F(1, 420) = 6.46, p = .011, \eta_p^2 = .015$) had an effect on reaction time whereas prime ($F(2, 420) = 2.34, p = .097, \eta_p^2 = .011$) and the interaction between load and prime ($F(2, 420) = 1.76, p = .172, \eta_p^2 = .008$) did not.[75] Pairwise comparisons showed that participants in the high load condition (M = 1,104.07) were significantly slower than those in the low load (M = 1,059.25) ($p = .011$).

The repeated measures results showed that reaction time to susceptibility and severity split by response key as a repeated measure, had an effect ($F(2.850, 1197.155) = 25.06, p < .001, \eta_p^2 = .056$). Further, there were two significant interactions between reaction time and cognitive load ($F(2.850, 1197.155) = 3.97, p = .009, \eta_p^2 = .009$) and between reaction time and prime ($F(5.701, 1197.155) = 2.33, p = .033, \eta_p^2 = .011$).[76] As a follow up to significant interactions, simple effects, with alpha set at .025 for cognitive load and .016 for primes, showed that reaction time differed within both high ($F(3, 1272) = 15.16, p < .001$) and low ($F(3, 1272) = 13.44, p < .001$) load conditions. Within the high cognitive load condition, pairwise comparisons, adjusted for familywise Type I error, showed three significant differences. Participants were significantly slower in acknowledging they were susceptible to Cluster 2 health conditions (M = 1,153.45) than

[75] Distribution of reaction time was within the acceptable range of ± 2 for skewness and kurtosis within each condition. Twenty-nine cases fell beyond $Z_{.005}$. Eight cases exceeded the critical value of $\chi^2_{(.001, 4)} = 18.46$ but did not have a large Cook's D. Homogeneity of variances was assumed for all dependent variables. Box's M test revealed a problem regarding the homogeneity of the variance-covariance matrices ($\chi^2 = 91.99, p < .001$). Linearity of the relationship between the dependent variables was checked by plotting all pairs of dependent variables. Variables were moderately correlated.

[76] Mauchly's test indicated that the assumption of sphericity had been violated ($\chi^2 = 31.64, p < .001$). Therefore, degrees of freedom were corrected using Greenhouse-Geisser estimates of sphericity ($\varepsilon = .95$).

acknowledging these health conditions were severe (M = 1,039.92) ($p < .001$). On the contrary, participants were slower in dismissing the health conditions were not severe (M = 1,116.86) than acknowledging they were ($p < .001$). Finally, participants were slower in dismissing they were not susceptible to Cluster 2 health conditions (M = 1,111.73) than acknowledging the health conditions were severe ($p = .002$).

Within the low cognitive load condition, there were four significant differences. Participants were significantly slower in acknowledging they were susceptible to Cluster 2 health conditions (M = 1,099.00) than dismissing they were not (M = 1,025.65) ($p < .001$) and that the health conditions were severe (M = 1,028.01) ($p < .001$). On the contrary, participants were significantly slower in dismissing the health conditions as not severe (M = 1,091.44) than acknowledging they were ($p < .001$) and that one was not susceptible to them ($p < .001$).

Within the prime conditions, reaction time was significantly different within the self ($F(3, 1269) = 8.15, p < .001$), third-person ($F(3, 1269) = 4.36, p = .005$), and neutral ($F(3, 1269) = 16.90, p < .001$) conditions. Within the self-prime condition, pairwise comparisons showed three significant differences. Participants were significantly slower in acknowledging they were susceptible to Cluster 2 health conditions (M = 1,153.45) than dismissing they were not (M = 1,073.98) ($p = .001$) and that the health conditions were severe (M = 1,073.98) ($p = .001$). On the contrary, dismissing the health conditions as not severe (M = 1,132.40) was significantly slower that acknowledging they were ($p = .023$).

Within the third-person prime condition, pairwise comparisons showed two significant differences. Participants were significantly slower in acknowledging they

were susceptible to Cluster 2 health conditions (M = 1,106.62) than acknowledging these health conditions were severe (M = 1,044.72) (p = .002). The latter was significantly faster than dismissing these health conditions as not severe (M = 1,096.47) (p = .020).

Finally, within the neutral-prime condition, pairwise comparisons showed three significant differences. Participants were significantly slower in acknowledging they were susceptible to Cluster 2 health conditions (M = 1,114.29) or dismissing they were not (M = 1,069.05) than acknowledging these health conditions were severe (M = 984.01) ($p < .001$, for each comparison). Further, dismissing the health conditions as not severe (M = 1,086.42) was significantly slower than acknowledging they were ($p < .001$) (Figure 17).

Cluster 3. Results for Cluster 3 showed that both the interaction term between load and prime ($F(2, 202) = 3.64$, $p = .028$, $\eta_p^2 = .035$) and load ($F(1, 202) = 5.87$, $p = .016$, $\eta_p^2 = .028$) had an effect on reaction time whereas prime ($F(2, 202) = .59$, $p = .554$, $\eta_p^2 = .006$) did not.[77] As a follow up to a significant interaction, simple effects, with alpha set at .01, showed that cognitive load had an effect within the neutral prime ($F(4, 199) = 3.43$, $p = .010$) but not with the self ($F(4, 199) = 2.30$, $p = .059$) or third-person ($F(4, 199) = .19$, $p = .938$) conditions. Primes did not have an effect with the low ($F(8, 398) = 1.32$, $p = .229$) or high ($F(8, 398) = .79$, $p = .603$) cognitive load conditions. Pairwise comparisons for reaction time within the neutral condition did not reveal any significant

[77] Distribution of reaction time was within the acceptable range for skewness and kurtosis within each condition. Thirty-two cases fell beyond $Z_{.005}$. One case exceeded the critical value of $\chi^2_{(.001, 4)} = 18.46$ but did not have a large Cook's D. Homogeneity of variances was assumed for all dependent variables. Box's M test revealed no problems regarding the homogeneity of the variance-covariance matrices ($\chi^2 = 62.90$, $p = .165$). Linearity of the relationship between the dependent variables was checked by plotting all pairs of dependent variables. Variables were moderately correlated.

differences. Performing paired *t* tests, however, showed that acknowledging one was susceptible to Cluster 3 health conditions (M = 1,020.93) was significantly faster than dismissing one was not (M = 1,091.44) (p = .025). Further, dismissing the health conditions as not severe (M = 1,016.24) was significantly faster than dismissing one was not susceptible to them (p = .012) (Figure 18).

It is noteworthy that a semantic differential affect/arousal averaged index (α = .85) was similar across experimental groups. The interaction term ($F(2, 470) = .59, p = .554$), cognitive load ($F(1, 470) = .27, p = .603$), and prime ($F(2, 470) = .01, p = .981$) were not significant. Measured at the end of experimental sessions, means were around the mid point of the semantic differential index: for the self (M = .79, SD = 1.65), third-person (M = .78, SD = 1.44), and neutral (M = .81, SD = 1.66) primes, averaged over prime conditions, and for the high (M = .76, SD = 1.52) and low (M = .83, SD = 1.64) load, averaged over cognitive load conditions. This indicated that any differences between the experimental groups were not attributed to a differential affect/arousal status.[78]

In sum, Experiment 2 provided partial support for Hypotheses 2a and 2b regarding the effects of self primes and cognitive load and their interaction on biasing risk perceptions. As predicted in Hypothesis 2a, main effects for self primes were evident for susceptibility to Cluster 1 health conditions. Main effects for cognitive load were evident for susceptibility to Clusters 2 and 3 and severity of Cluster 2 conditions. As

[78] The distribution of affect/arousal index was within the acceptable range of ±2 for skewness and kurtosis for each condition. Forty-six cases exceeded the critical value of $Z_{.005}$. Equal variances were not assumed as evidenced by a significant Levene's test ($F(2, 470) = 2.37$ p = .038).

predicted in Hypothesis 2b, self primes and high cognitive load interacted to bias severity of Cluster 1 health conditions downward.

In terms of response key, participants primed with self-identity words underestimated the likelihood of experiencing Cluster 1 health conditions (e.g., brain tumors), the only significant effect of self primes on biasing risk perceptions. Participants who memorized an 8-digit number (i.e., high cognitive load) underestimated the likelihood of experiencing Cluster 2 (e.g., allergies, diabetes) and 3 (e.g., flu) health conditions and the severity of Cluster 2 health conditions. Further, participants primed with self-identity words underestimated the severity of Cluster 1 health conditions but only when under high cognitive load, in support of the interaction hypothesis (H2b). These effects, however, were not evident for Clusters 2 and 3 health conditions.

In terms of susceptibility reaction times, participants in the self-identity words condition had slow reaction times to Cluster 1 health conditions compared to Cluster 3 when they were under high cognitive load. For severity reaction time, participants in the self prime condition responded faster to Cluster 3, followed by Cluster 1, and 2 health conditions. Reaction times were slower in the high cognitive load compared to the low load condition.

Experiment 2 fully supported Hypothesis 4. Participants underestimated their susceptibility to Cluster 1 (e.g., leukemia, HPV) and 2 (e.g., allergies, diabetes) health conditions, which were perceived to be of low and moderate prevalence, salience, controllability, exempt beliefs, and personal experience. In contrast, they overestimated their susceptibility to Cluster 3 health conditions (e.g., flu, stress) that were scored high on these health risk characteristics variables.

Although no predictions were made regarding perceptions of severity based on health risk characteristics, Experiment 2 revealed an inverted relationship between susceptibility to and severity of health condition clusters. In terms of response key, participants acknowledged the severity of Cluster 1 and 2 health conditions but tended to underestimate their likelihood of experiencing them. In contrast, participants acknowledged their likelihood of experiencing Cluster 3 health conditions but tended to underestimate the severity of these health conditions. These judgments were also reflected in reaction time data. Participants were faster acknowledging the severity of Cluster 1 and 2 health conditions as opposed to dismissing their severity. On the other hand, participants were faster dismissing their likelihood of experiencing Cluster 1 and 2 health conditions as opposed to acknowledging the likelihood of experiencing them. Participants exhibited a reversed pattern for Cluster 3 health conditions. Participants were faster acknowledging their likelihood of experiencing these health conditions and dismissing their severity compared to dismissing the likelihood of experiencing these health conditions and acknowledging their severity. These fast reaction times reflect well-rehearsed judgments that participants have regarding their susceptibility to and severity of different health condition clusters.

In partial support of Hypothesis 5 (and consistent with Experiment 1), depression and self-consciousness, and low levels of optimism, self-efficacy, self-esteem, and health locus of control were associated with increased perceptions of susceptibility to Cluster 2 and 3 health conditions and severity of Cluster 3 health conditions. Further, self-consciousness and low levels of optimism were associated with increased perceptions of severity of Cluster 3 health conditions but decreased perceptions of susceptibility to

Cluster 3 and severity of Cluster 1 health conditions. Psychological reactance and self-awareness were not associated with overestimation of health risks as hypothesized.

Experiment 3: Effects of health risk controllability and psychological reactance on health risk perceptions

Within the context of a health message, Experiment 3 tested the effects of health risk controllability and psychological reactance on perceived susceptibility to and severity of a disease when primed with self-identity, third-person, or neutral words. The study also included a behavioral measure of information seeking. The experiment was a 3 (prime: self-identity, third-person, neutral words) x 2 (risk controllability: high, low) between-subjects design with psychological reactance as a measured variable.

A total of 90 participants were recruited for a $10 incentive. They were randomly assigned to one of six experimental conditions. The majority of participants were females (n = 53, 58.9%) and right-handed (n = 81, 90.0%). Mean age was 21.15 (SD = 2.92).

Data for Experiment 3 included measures of individual differences (e.g., self-efficacy) as well as response key and reaction time data for susceptibility to and severity of Balamuthia infection. First, baseline measures are described. Second, descriptive statistics and reliability of individual difference variables are presented. Third, response key and reaction time data are analyzed to detect differences between the experimental conditions based on experimental treatments.

Baseline measures.

A series of univariate ANOVAs, with prime and risk controllability as fixed factors, showed no differences between the experimental groups on baseline (i.e., practice trial) response key accuracy and reaction time. For baseline response key accuracy, there were no differences between the experimental conditions in total correct response key as

a dependent variable. The interaction term ($F(2, 84) = .24, p = .783$), risk controllability ($F(1, 84) = .14, p = .709$), and prime ($F(2, 84) = .66, p = .517$) were not significant. Participants in self ($M = 19.60, SD = .62$), third-person ($M = 19.70, SD = .59$) and neutral ($M = 19.76, SD = .43$) conditions correctly identified practice-trial items as animals/non-animal. Averaged across risk controllability, means were 19.66 ($SD = .60$) and 19.71 ($SD = .50$) for the controllable and uncontrollable conditions, respectively.[79]

Raw baseline reaction time data fell between 397 and 3,640 milliseconds. Data points that were 4 *SD* away from the mean were considered outliers and excluded ($n = 19$), a 1.05% total data points lost. A univariate ANOVA performed on log-transformed reaction time data showed the interaction term between risk controllability and prime ($F(2, 84) = .52, p = .593$), risk controllability ($F(1, 84) = .264, p = .609$) and prime ($F(2, 84) = 1.73, p = .183$) were not significant.[80] Mean reaction time for participants in the controllable condition was 629.94 ($SD = 1.11$) whereas reaction time for those in the uncontrollable condition was 622.58 ($SD = 1.11$). Averaged over primes, means were 610.66 ($SD = 1.10$) for the self condition, 643.13 ($SD = 1.10$) for the third-person condition, and 625.17 ($SD = 1.12$) for the neutral condition.

Individual differences.

[79] The distribution of total correct response key for each condition was within the acceptable range of ±2 for skewness and kurtosis. Four cases exceeded the critical value of $Z_{.005}$ (2.58). Equal variances were assumed as evidenced by a non-significant Levene's test ($F(5, 84) = 1.69, p = .145$).

[80] The distribution of log-transformed reaction time data was within the acceptable range of ±2 for skewness and kurtosis within each experimental condition. Equal variances were assumed as evidenced by a non-significant Levene's test ($F(5, 84) = .80, p = .551$).

Individual difference variables were screened for normality of distributions and outliers. There were no missing values (See Table 20 for descriptive statistics and correlation matrix).

Participants scored low on the depression inventory ($M = 1.41$, $SD = .34$, $\alpha = .87$).[81] Consistently, participants were optimistic, as measured by the life orientation test ($M = 2.56$, $SD = .77$, $\alpha = .85$).

Participants showed slightly high levels of internal health locus of control ($M = 3.88$, $SD = .61$, $\alpha = .73$). Consistent with the literature, results revealed health locus of control had two underlying factors, which explained 33.17% of the variance.[82] The first factor was related to internal locus of control whereas the second factor was related to external locus of control. Based on the analysis, four items reflected internal locus of control ($\alpha = .69$), and six items reflected external locus of control ($\alpha = .67$), with a correlation of $r = -.25$ between the two factors (See Table 20 for factor loadings, factor correlation, and internal reliability statistics).

[81] The reported reliability is for 20 items of the depression inventory. A weight loss item was excluded because it reduced the sample size from 90 to 29 (those who were trying to loose weight) with listwise deletion. When the weight loss item was included, reliability was .67 ($N = 29$).

[82] Evaluation of normality of distribution, outliers, and factorability were satisfactory. All variables were within the acceptable ±2 for skewness and kurtosis. Eight cases exceeded the critical value of $Z_{.005}$. Multivariate outliers were checked using Mahalanobis distance. No case was above the critical value of chi-square ($\chi^2_{(.001, 11)} = 31.26$). Kaiser-Meyer-Olkin (KMO) measure of sampling adequacy revealed there was a degree of common variance appropriate for conducting a factor analysis (KMO = .72). Each item's Measure of Sampling Adequacy (MSA) was greater than .60, with the lowest being .67. Accordingly, all items were retained for the analysis. Bartlett's test of sphericity indicated the correlation matrix is factorable ($\chi^2 = 203.63$, $p < .001$). The initial solution indicated there were two factors with eigenvalues greater than 1. Similarly, Catell's criterion for the scree plot suggested that there might be two factors. With the extraction of two factors, there were 22 (40.0%) non-redundant residuals with absolute values greater than .05.

Participants were slightly reactant ($M = 3.00$, $SD = .54$, $\alpha = .83$). Psychological reactance was positively correlated with self-consciousness and negatively with self-efficacy and self-esteem.

Participants' scores were roughly at the mid point of the self-consciousness scale ($M = 2.59$, $SD = .37$, $\alpha = .74$). Results revealed self-consciousness had three underlying factors, which explained 31.53% of the variance.[83] The first factor was related to social anxiety (e.g., "I feel anxious when I speak in front of people"). The second was related to public self (e.g., "I'm concerned about what other people think of me"). The third factor was related to private self (e.g., "I'm generally attentive to my inner feelings"). Private self was further broken down to two subscales: self-reflection and self-awareness, which explained 37.59% of the variance, with a correlation of .33. Self-reflection included four items such as "I reflect about myself a lot" ($M = 2.85$, $SD = .62$, $\alpha = .70$). Self-awareness included two items such as "I'm alert to changes in my mood" ($M = 3.10$, $SD = .57$, $r = .31$) (See Table 21 for factor loadings, factor correlation, and internal reliability statistics).

Finally, participants scored high on self efficacy ($M = 3.60$, $SD = .52$, $\alpha = .87$) and self esteem ($M = 3.10$, $SD = .50$, $\alpha = .89$).

[83] Evaluations of normality of distribution, outliers, and factorability were satisfactory. All individual items were within the acceptable range for skewness and kurtosis. Thirty-three cases exceeded the critical value of $Z_{.005}$. No case exceeded the critical value of $\chi^2_{(.001, 23)} = 49.72$. Kaiser-Meyer-Olkin (KMO) measure of sampling adequacy revealed there was a degree of common variance and hence the sample was appropriate for conducting a Principal Axis Factoring (KMO = .64). Some items had a Measure of Sampling Adequacy (MSA) less than .60, with the lowest being .32. All items were retained for the analysis. Bartlett's test of sphericity indicated the correlation matrix is factorable ($\chi^2 = 598.47$, $p < .001$). Initial solution indicated there were four factors with eigenvalues greater than 1. Catell's criterion for the scree plot suggested that there might be three factors. With the extraction of three factors, there were 107 (42.0%) non-redundant residuals with absolute values greater than .05.

Effects of prime, risk controllability, and psychological reactance on susceptibility to and severity of a health risk and information seeking.

Experiment 3 tested the effects of risk controllability (controllable vs. uncontrollable), primes (self, third-person, neutral), and psychological reactance on perceived susceptibility to and severity of a health condition, and an information-seeking behavioral measure.

In response to whether one was susceptible (*yes*) or not (*no*) to Balamuthia infection and whether the disease would be severe (*yes*) or not (*no*) if one experiences it, it was predicted in Hypothesis 3a that participants in the self-identity words condition, high on reactance, and in the highly controllable health risk condition would respond "*no*." Reaction times for "*no*" responses would be faster than "*yes*" responses. This pattern was predicted to be stronger for participants primed with self-identity words in the high controllability condition (H3b), for highly reactant participants when primed with self-identity words (H3c), and for highly reactant participants when primed with self-identity words and exposed to the high controllability (H3d).

(1) Effects on response key. A series of sequential logistic regressions[84] were performed to test the effects of individual difference variables and experimental treatment on response key for susceptibility to and severity of the health condition as well as a behavioral measure in which participants requested additional information about the disease or not.

A sequential logistic regression was performed to assess the prediction of perceived susceptibility to Balamuthia infection (0 = *no*, 1 = *yes*), first on the basis of

[84] Logistic regression predicts a binary outcome (e.g., yes/no) from a set of discrete, continuous, or a mix of discrete and continuous predictors (Tabachnik & Fidell, 2007).

individual difference variables and then after addition of primes and risk controllability.[85] There was a good model fit on the basis of the individual differences alone, $\chi^2 = 72.93$, $p = .671$, using a deviance criterion. After addition of the experimental treatment, $\chi^2 = 63.77$, $p = .840$, Nagelkerke $R^2 = .33$. Comparison of log-likelihood ratios for models with and without experimental treatment showed statistically significant improvement with the addition of experimental treatment ($\chi^2 = 9.15$, $p < .05$). Classification rates on the basis of individual differences alone were 98.9% for *no* and 1.1% for *yes,* with an overall classification rate of 81.1%. The addition of experimental treatment improved overall classification rate to 85.6%, which was reflected in a 10.0% success rate in predicting *yes* response to susceptibility of the disease. Looking at the contribution of individual predictors revealed that self-reflection and risk controllability statistically enhanced the prediction of susceptibility response key. Consistent with Hypothesis 3a, seventy five percent of participants who perceived Balamuthia infection as a disease they might contract were in the uncontrollable risk condition (Table 22).

A second sequential logistic regression was performed to assess the prediction of perceived severity to Balamuthia infection (0 = *no*, 1 = *yes*), first on the basis of individual difference variables and then after addition of primes and risk controllability.[86] There was a good model fit on the basis of the individual differences alone, $\chi^2 = 90.14$, $p = .184$, using a deviance criterion. After addition of the experimental treatment, $\chi^2 = 82.23$, $p = .293$, Nagelkerke $R^2 = .40$. Comparison of log-likelihood ratios for models

[85] Evaluation of adequacy of expected frequencies for categorical predictors revealed one cell with less than 5 observations. No serious violation of linearity in the logit was observed.

[86] Evaluation of adequacy of expected frequencies for categorical predictors revealed no need to restrict model goodness-of-fit tests. No serious violation of linearity in the logit was observed.

with and without experimental treatment showed statistically significant improvement with the addition of experimental treatment ($\chi^2 = 7.91, p < .05$). Classification rates on the basis of individual differences alone were 20.0% for *no* and 80.0% for *yes,* with an overall classification rate of 76.7%. The addition of experimental treatment improved overall classification rate to 81.1%, which was reflected in a 28.9% success rate in predicting *no* response to severity of the disease. Looking at the contribution of individual predictors revealed that depression, self-efficacy, self-awareness, and risk controllability statistically enhanced the prediction of severity response key. Consistent with Hypothesis 3a, sixty percent of participants who perceived Balamuthia infection to be severe were in the uncontrollable risk condition.

Finally, a third sequential logistic regression was performed to assess the prediction of behavioral measure in which participants requested additional information about Balamuthia infection (0 = *no*, 1 = *yes*), first on the basis of individual difference variables and then after addition of primes and risk controllability.[87] There was a good model fit on the basis of the individual differences alone, $\chi^2 = 91.00, p = .168$, using a deviance criterion. After addition of the experimental treatment, $\chi^2 = 83.41, p = .262$, Nagelkerke $R^2 = .34$. Comparison of log-likelihood ratios for models with and without experimental treatment showed no statistically significant improvement with the addition of experimental treatment ($\chi^2 = 7.58, p > .05$). Classification rates on the basis of individual differences alone were 84.4% for *no* and 15.6% for *yes,* with an overall classification rate of 77.8%. The addition of experimental treatment reduced overall

[87] Evaluation of adequacy of expected frequencies for categorical predictors revealed no need to restrict model goodness-of-fit tests. No serious violation of linearity in the logit was observed.

classification rate to 74.4%, but improved the rate in predicting the request of additional information about the disease to 21.1%. Looking at the contribution of individual predictors revealed that self-reflection, self-awareness, gender, and risk controllability statistically enhanced the prediction of severity response key. Sixty five percent of participants who requested additional information about Balamuthia infection were in the uncontrollable risk condition.

(2) Effects on reaction time combined for yes and no responses. A repeated measures analysis of variance was performed with prime and risk controllability, as fixed factors, and psychological reactance, as a continuous independent variable, and reaction time for susceptibility and severity of Balmauthia disease, as a repeated measure, to detect differences on reaction time combined for the *yes* and *no* response keys. Data were screened for fit of underlying assumptions regarding normality of sampling distribution, homogeneity of variance-covariance matrices, linearity, and multicolinearity.[88]

For the between-subjects factors, reaction time to susceptibility and severity of the disease did not differ based on the interaction term between prime and risk controllability ($F(2, 83) = .30, p = .737, \eta_p^2 = .007$), the prime ($F(2, 83) = .16, p = .846, \eta_p^2 = .004$), or risk controllability ($F(1, 83) = 1.372, p = .245, \eta_p^2 = .016$). Mean reaction times were 2,275.09 for the self prime, 2,344.22 for the third-person prime, and 2,296.14 for the

[88] Distribution of reaction time was within the acceptable range of ±2 for skewness and kurtosis within each condition. Two cases fell beyond $Z_{.005}$. Two cases exceeded the critical value of $\chi^2_{(.001, 2)} = 13.81$ but did not have a large Cook's D. Homogeneity of variances was assumed for the two dependent variables ($F(5, 84) = 1.42, p = .223$ for susceptibility and $F(5, 84) = 1.36, p = .246$ for severity). Box's M test revealed no problem regarding the homogeneity of the variance-covariance matrices ($\chi^2 = 16.78, p = .398$). Linearity of the relationship between the dependent variables was checked by plotting the two dependent variables. Variables were moderately correlated and posed no problems.

neutral prime. Averaged over controllability conditions, reaction times were 2,393.31 for the controllable condition and 2,218.19 for the uncontrollable condition.

Within-subjects results showed a significant interaction between psychological reactance and repeated measure (i.e., susceptibility and severity reaction time) ($F(1, 83) = 4.92, p = .029, \eta_p^2 = .056$). As a follow up to a significant interaction, a regression analysis showed that psychological reactance was a significant predictor of susceptibility reaction time ($F(1, 88) = 5.48, p = .021, R^2 = .059$). One unit increase in reactance was associated with slower reaction time ($\beta = -.242, p = .021$). This effect was not evident for severity reaction time ($F(1, 88) = .004, p = .951, R^2 = .00, \beta = .007, p = .951$).[89]

The semantic differential affect/arousal index ($\alpha = .83$) was similar across experimental groups. The interaction term ($F(2, 84) = .16, p = .849$), risk controllability ($F(1, 84) = .14, p = .706$), and prime ($F(2, 84) = .05, p = .945$) were not significant. Measured at the end of experimental sessions, means were around the mid point of the semantic differential index: for the self ($M = 1.18, SD = 1.36$), third-person ($M = 1.07, SD = 1.54$), and neutral ($M = 1.07, SD = 1.40$) primes, averaged over prime conditions, and for the controllable ($M = 1.17, SD = 1.53$) and uncontrollable ($M = 1.05, SD = 1.32$) conditions, averaged over risk controllability conditions. This indicated that any differences between the experimental groups were not attributed to a differential affect/arousal status.[90]

---

[89] Testing the effects of prime, risk controllability, and reactance on reaction time split by yes and no was not feasible because of lack of adequate number of cases for the analysis with listwise deletion.

[90] The distribution of affect/arousal index was within the acceptable range of ±2 for skewness and kurtosis for each condition. Nine cases exceeded the critical value of $Z_{.005}$. Equal variances were assumed as evidenced by a non-significant Levene's test ($F(5, 84) = .23$ $p = .945$).

In sum, Experiment 3 tested the effects of primes in the context of a health message along with the effects of risk controllability and psychological reactance on perceptions of susceptibility to and severity of Balamuthia infection. The experiment did not find support for the hypothesized main effects of primes (H3a) and the interaction between primes and risk controllability (H3b), primes and reactance (H3c), and primes, risk controllability, and reactance (H3d). Consistent with Hypothesis 3a, results showed that risk controllability was a powerful predictor of response key. Participants who read about Blamuthia as an uncontrollable disease perceived themselves to be susceptible to the disease; perceived the disease to be severe; and requested additional information about the disease at the end of the experimental session. Inconsistent with H3a, psychological reactance was associated with slower reaction times to perceived susceptibility.

Conclusion

Three experiments were conducted to test the automatic nature of health risk perceptions. Experiment 1 provided evidence of upward bias for positive health conditions and downward bias for negative ones. Experiment 2 provided evidence that underestimation of health risks is an efficient process that occurs when people are mentally taxed. Finally, in the context of a health message, Experiment 3 provided support for the effect of health risk controllability on risk perceptions. An uncontrollable disease elicited perceptions of susceptibility to and severity of the disease as well as information seeking.

In the next chapter, a summary of results is provided along with theoretical, practical, and methodological implications for the work presented here.

CHAPTER 5

CONCLUSIONS AND DISCUSSION

Although risk perceptions are necessary for positive behavioral change, they are inaccurate and often underestimated. This dissertation posited that the principles of automaticity apply to how people process personal health risk messages and may explain why people are biased to underestimate personal risks.

Two theoretical propositions were advanced:

(1) Underestimation of health risks is driven by a self-positivity bias. When the self-schema is active, perceptions of susceptibility will be biased upward for positive health conditions and downward for negative ones. Perceptions of severity will be biased downward for negative health conditions.

(2) Underestimation of personal susceptibility to and severity of negative health risks is automatic. Biased perceptions of risk possess two features of automatic processes: lack of conscious awareness and efficiency. Underestimation of personal risks occurs without (a) conscious awareness of underestimation of health risks as a bias and of self-schema activation as a source of the bias and (b) much cognitive resources.

Three pilot studies and three experiments tested these theoretical propositions and five derived hypotheses. Using implicit measures of risk perceptions, the studies provided conclusive evidence of (a) underestimation of susceptibility to negative health conditions and overestimation of susceptibility to positive ones and (b) efficiency of underestimation

of health risks. Results regarding self-schema activation as a source of underestimation of risks were not as conclusive.

Pilot Study 3 and Experiment 1 provided support for the first theoretical proposition. Consistent with previous literature, Pilot Study 3 provided evidence of implicit (dis)association between the self and negative/positive traits. Participants identified negative traits (e.g., gossipy) as non self-descriptive and positive traits (e.g., sincere) as self-descriptive. Sickness/health traits mirrored negative/positive traits in their implicit (dis)association with the self. Participants identified sickness traits (e.g., unhealthy) as non self-descriptive and health traits (e.g., healthy) as self-descriptive. In reaction time terminology, faster reaction times were recorded when participants identified negative and sickness traits as non self-descriptive and positive and health traits as self-descriptive.

Experiment 1 replicated the results of Pilot Study 3 with negative (e.g., stroke) and positive (e.g., healthy heart) health conditions. Participants underestimated the likelihood of experiencing negative health conditions and overestimated the likelihood of experiencing positive ones. Fast reaction times were recorded when participants dismissed the likelihood of experiencing negative health conditions and acknowledged the likelihood of experiencing positive ones.

Using implicit measures of risk perceptions, both Pilot Study 3 and Experiment 1 supported self-report studies of risk perceptions that documented people's tendency to underestimate their risks. Negative and sickness traits were deemed non self-descriptive whereas positive and health traits were deemed self-descriptive. Similarly, negative health conditions were deemed not likely to happen to oneself whereas positive

conditions were deemed likely to happen to oneself. Thus, it appears that making such judgments frequently qualifies them to migrate from consciousness to unconsciousness. Fast reaction time is an indication that these judgments are less thoughtful and well-rehearsed automatic processes.

Experiment 2 provided conclusive support that underestimation of health risks is an efficient process. Efficiency refers to processes that occur when people lack sufficient cognitive resources, time, or motivation to engage in elaborate processing. As supported in Experiment 2, when people lack cognitive resources, underestimation of health risks is more likely.

Evidence of self-schema activation as a source of underestimation of risks was sporadic, however. In Pilot Study 3, participants primed with self-identity words had slower reaction times in response to sickness traits compared to positive ones. Experiment 2 provided evidence of underestimation of perceptions of susceptibility to and severity of Cluster 1 health conditions (e.g., brain tumors) but only for perceptions of severity when under high load.

Such evidence, although limited to a specific cluster of health conditions, provides some insights to the effects of self-schema activation on biasing risk perceptions. One explanation could be that the self-schema is highly accessible (Bargh, 1989). Thus, self-schema could have been active in other prime conditions (i.e., third-person, neutral), which minimized the predicted differences for the self primes.

Self-report data from Pilot Studies 1 and 2 and implicit measures of risk perceptions from Experiment 2 revealed an inverted relationship between susceptibility and severity, the two dimensions that are typically added or multiplied to form an index

of health risk perceptions. Participants do not underestimate or overestimate both susceptibility and severity. If the severity of a health condition was undisputable (e.g., leukemia), participants downplayed their likelihood of experiencing the condition. In contrast, if susceptibility to a health condition was undisputable (e.g., stress), participants downplayed the severity of the condition. Such a relationship allows people to maintain risk perceptions at a manageable level in light of undeniable evidence about their susceptibility to or severity of a given health condition. Slovic and his colleagues documented a negative correlation between risk and benefit in people's minds, which is similar to the susceptibility – severity inverted relationship found in this study (Slovic, Fischoff, & Lichtenstein, 1982; Slovic & Peters, 2006).

Such judgments, again, reflect well-rehearsed automatic processes. Fast reaction times were recorded when participants acknowledged the severity of Cluster 1 (e.g., brain tumors) and 2 (e.g., diabetes) health conditions and dismissed their likelihood of experiencing such conditions. In contrast, fast reaction times were recorded when participants acknowledged their likelihood of experiencing Cluster 3 health conditions (e.g., flu, sunburn) and dismissed these health conditions as non severe.

Evidence from three studies (Pilot Study 3, Experiment 1, and Experiment 2) suggests there are *defaults* in people's minds when it comes to health risk perceptions:

(1) We are likely to experience positive health conditions and unlikely to experience negative health conditions. Dismissing the likelihood of experiencing negative health conditions was easy (as evidenced by fast reaction time).

(2) The default is underestimation of perceptions of susceptibility to and severity of a health condition. Underestimation of risk can be seen as the default because it occurs under limited cognitive resources.

(3) We are likely to acknowledge the severity of health conditions but dismiss the likelihood of experiencing them. In contrast, we acknowledge the likelihood of experiencing highly prevalent health conditions but dismiss them as not severe.

Experiments 1, 2, and 3 provided evidence regarding the relationship between individual difference variables (e.g., self-efficacy) and risk perceptions. Such individual differences have been shown in self-report studies to influence risk perceptions upwards or downwards. It was unclear from the literature whether these individual differences would have the same effect on implicit measures of risk perceptions. Evidence from all three experiments suggests that individual difference variables did not affect reaction time data.

On another level, these individual differences have shown some association with response key data. Consistent with previous literature, high levels of self-efficacy, self-esteem, and dispositional optimism (as measured by the life orientation test), and low levels of depression were associated with underestimation of risk. Evidence regarding the association between health locus of control, psychological reactance, self-awareness and self-reflection (the two subscales of self-consciousness), on the one hand, and biased health risks, on the other hand, was sporadic, but consistent with previous literature when found.

Finally, Experiments 2 and 3 provided evidence regarding health risk characteristics and their influence on risk perceptions. Participants tended to

underestimate their susceptibility to health conditions that score low on prevalence, personal experience, controllability, stereotype salience, and exempt beliefs. In contrast, participants tended to overestimate their susceptibility to health conditions that scored high on these health risk characteristics. Coupled with reaction time data on susceptibility to and severity of clusters of health conditions, it is evident that the health risk's characteristics play an important role in the perception of health risks. It remains to be tested whether people make compensatory (e.g., take all risk characteristics into account when estimating their risk perceptions) or non-compensatory (e.g., base risk perceptions on one risk characteristic and discount all others) decisions about health conditions based on health risk characteristics.

Manipulating health risk controllability in Experiment 3 elicited increased perceived susceptibility to and severity of the health condition in question. Further, exposure to information about an uncontrollable health condition prompted participants to seek additional information regarding the disease, as evidenced by a behavioral measure.

In the next section, theoretical, practical, and methodological implications of this work are presented. Limitations of the work are also discussed.

Theoretical implications

This dissertation posited that self-schema activation is the source of underestimated health risk perceptions. A theoretical model is presented in Figure 19 to describe how people arrive at these biased health risk perceptions. First, the model details how self-schema can be automatically activated. Second, it summarizes pathways by

which the active self-schema can bias risk perceptions via an indirect and/or a direct route of information processing.

Automatic Self-schema Activation.

Self-schema can be activated by one of three automatic processes: pre-conscious, post-conscious and goal-dependent automaticity – depending on preconditions necessary for each process to occur (Bargh, 1989, 1994). In pre-conscious automaticity, the mere presence of a stimulus triggers an automatic process beyond one's conscious awareness, control, or intention (Bargh, 1989, 1997). Because "self-relevant information is among the most frequently experienced, it is likely that individuals possess chronically accessible constructs for such domains of social information" (Bargh, 1989, p.13). Several cues – contingent upon one's proximal environment and activities – can trigger a pre-conscious activation of self-schema such as exposure to one's reflection in a mirror, name, or self-portrait (Eichstaedt & Silvia, 2003).

Post-conscious automaticity is a non-conscious outcome for a recent conscious thought. A message "it could be you" will activate the self-schema, which continues to influence message processing. Here, a person is consciously aware of the second person pronoun, but is unaware of its effects on self-schema activation and processing of the health message.

Goal-dependent automaticity is an intended or unintended outcome of a conscious goal. When attending to a risk message, a viewer's conscious goal would be to process the information. An intended outcome of goal-dependent automaticity would be to self-protect by discounting and refuting message arguments and underestimating one's risks (Higgins, 1987). In an unintended outcome, self-schema activation influences conscious

message processing and results in discounting and underestimating the risk, an outcome consistent with the chronic positive self-schema (Bargh, 1989), yet inconsistent with the intended outcome of reaching accurate risk estimates.

In sum, self-schema can be activated by one of three automaticity processes. The more preconditions required for an automatic process to occur, the less prevalent self-schema activation is because all necessary preconditions must be present (and vice versa). Pre-conscious automaticity does not require any preconditions. Post-conscious automaticity requires "awareness of instigating stimulus" whereas goal-dependent automaticity requires "specific processing goal in place, intention that effect occur, allocation of focal attention to processes" (Bargh, 1989, p.10). Thus, automatic activation of self-schema will occur more frequently under pre-conscious followed by post-conscious and, lastly, goal-dependent automatic processing.

Effects of Automatic Self-schema Activation on Risk Perceptions.

The self-schema, once active, can influence health risk information processing and bias risk perceptions in two ways: indirectly via elaborate/controlled information processing or directly in a completely automatic fashion.

Although the elaboration likelihood (ELM) and heuristic systematic (HSM) models assume people seek accurate judgments, both models propose ways in which judgments can be biased. Motivation (e.g., defense motive in HSM) and ability (i.e., prior knowledge) can bias outcomes of a persuasion process if people have a preference for one outcome versus another or if prior knowledge is imbalanced to begin with. In this regard, people will select, attend to, perceive information and/or selectively retrieve

elements of self-knowledge that support their motives and preferences (Chen & Chaiken, 1999; Giner-Sorolla & Chaiken, 1997; Petty & Wegner, 1999).

Automatic self-schema activation can influence elaborate/controlled information processing indirectly. When a message is encoded in terms of an active self-schema, one integrates additional information and inferences from that schema into the objective message, transforming the message into a subjective stimulus, which results in an understanding, interpretation and recall of information that is consistent with an overly positive self view (Bargh, 1997; Bruner, 1957; Higgins, 1989). This influence occurs through several routes. Self-schema directs attention to certain aspects of the message; message elements are further automatically evaluated as positive or negative (Bargh, Chaiken, Govender, & Pratto, 1992; Ferguson, 2007); judgments and decisions are based on active schemas, both conscious and unconscious, which makes the influence of automatic self-schema activation on conscious decisions and judgments likely.

Automatic self-schema activation will solely drive judgments in a completely automatic fashion when people do not engage in intentional information gathering or lack the ability and/or motivation to engage in elaborate information processing (Bargh, 1989).

Such influence of automatic processes on judgments and decisions is unquestionable because " one implicitly trusts in the veracity of the interpretation made, because one is not aware of any processing effort being applied" (Bargh, 1989, p.19). Further, automatic processes continue to operate with limited cognitive resources, resembling real life when people are distracted or engaged in other activities during exposure to health risk messages (Perse, 1990).

In the event that people are motivated to reach accurate decisions, they must be aware of both the magnitude and direction of the positivity bias and be motivated to and have the ability (time and processing capacities) to overcome this bias. Such a confluence of conditions rarely exists (Wegner & Petty, 1995).

To illustrate, consider a health risk message that explicitly references the self: "When was the last time you REALLY checked yourself? Syphilis is up to 365% in LA County gay men since 2001. So, it's essential to get checked out every 6 months or every 3 months if you're HIV+. The good news is syphilis is curable – but left untreated, it can permanently damage your health. (Tagline): (really) CHECK YOURSELF. Get tested for syphilis every 6 months" (County of LA Department of Public Health).

Upon reading this message, self-schema is activated (i.e., post-conscious automaticity because one is consciously aware of the words: you, yourself). Once active, self-schema can influence risk perceptions in two ways:

(a) An indirect path where one interested in learning about syphilis will engage in elaborate information processing. The positivity bias, however, would direct attention to certain messages aspects. One might think the 365% is unrealistic or think the message is not credible because no source is cited. In considering disease prevalence and message source, one is unaware this allocation of attention is driven by self-schema and, thus, trusts that his conclusion to discount the message is based on rational reasons (not a positivity bias). Finally, in making a risk judgment, a necessary step to get checked for syphilis, input from all active mental representations used to encode the message is integrated. This includes consciously (e.g., syphilis, gay men) and unconsciously (e.g., self-schema) activated schemas.

(b) A direct path where one does not have the motivation and/or ability to engage in elaborate processing. The information will simply be perceived as inconsistent with one's positive self-schema and therefore risk perceptions will be underestimated.

Practical implications

An agenda for research and practice is presented next. The research agenda integrates literature on the self and automatic processes to better understand personal risk perceptions. The practice agenda centers around two outcomes: message content and implementation and evaluation strategies.

A Research Agenda.

The self and risk perceptions. Underestimation of risk has been documented for personal risks. Are risk perceptions underestimated if one experiences the risk via a virtual self? The virtual self is a representation of one's self in the virtual world via an avatar (Fox & Bailenson, 2009). For example, in addressing calorie intake as a risk factor for obesity, one could experience an increase in size/body fat via the virtual self. Two contradictory hypotheses are worth exploring: risk perceptions (1) will be underestimated (as in self-report studies of risk perceptions) or (2) will be acknowledged because one's experience of the risk is almost real.

Another research area is risk perceptions for the extended self. "Extended self" refers to people we consider part of ourselves (e.g., children, significant others) (Belk, 1988). These perceptions are important when making proxy decisions. For example, a parent assesses her child's susceptibility to a disease and decides whether or not to vaccinate the child. Are the parent's perceptions of the child's susceptibility (i.e., parent's extended self) subject to the same biases in estimating personal risks?

Health risk communication could benefit from neuroimaging technology. Previous studies have shown active areas of the brain when processing self-relevant information and patterns involved in processing self + positive versus self + negative information (Kelley et al., 2002; Kircher et al., 2002; Watson et al., 2007). Neuroimaging can be used to record brain responses to personal health risk messages and provide neurological evidence on how people process health risk information.

Automatic processing and risk perceptions. Although logically derived from a body of research on automaticity and dual process theories of persuasion, the model proposed here should be empirically tested especially with high-risk groups and patient populations. With these populations, it is unknown whether biased risk perceptions will shift to severity and/or treatment effectiveness and in what ways, if any.

Given that automatic and controlled processing are not mutually exclusive (Bargh, 1989), scholars should look into the combination of automatic versus controlled processing of health risk information. Procedures such as the Process Dissociation Paradigm allow scholars to estimate the automatic and controlled contributions to any mental process.

Susceptibility – severity relationship. Theories of behavioral change differ in the way they combine the two dimensions of risk perceptions: susceptibility and severity. PMT (Rogers, 1975) and EPPM (Witte, 1992; Witte & Allen, 2000) posit an additive relationship whereas TRA, TPB, and the IBM posit a multiplicative one (Ajzen, 1991; Ajzen & Fishbein, 1973; Montaño & Kasprzyk, 2008). In other theories, like HBM, the nature of the relationship between susceptibility and severity is unidentified (Becker,

1974). Only few studies suggested that susceptibility and severity are two independent constructs (Witte & Allen, 2000).

Given the conclusive evidence of the inverted susceptibility – severity relationship found in this study, it is no longer acceptable to combine the two dimensions in an additive or multiplicative index. Future research should define more clearly the relationship between susceptibility and severity. It is not clear if the two dimensions are equally important or if one dimension drives people's risk perceptions (e.g., susceptibility drives preventive behaviors such as vaccination whereas severity drives detection behaviors such as cancer screening) (Brewer et al., 2007). Identifying the nature of the susceptibility – severity relationship will result in straightforward recommendations for health communication practitioners.

A Practice Agenda.

Message content. Risk messages should boost positivity bias when beneficial and override it when detrimental to behavioral change. For example, messages promoting initiation or maintenance of a behavior could emphasize optimism about its effectiveness. Furthermore, boosting optimism can help people who have acknowledged their risks cope with health issues (Taylor et al., 1992).

When maladaptive for behavioral change, risk messages should have alternative foci than personal risk. This includes using heuristics such as anchoring to bring risk perceptions closer to objective risk estimates (Dillard, McCaul, Kelso, & Klein, 2006); relying on identifiable factors such as age to socially pressure those at risk to adhere to recommended behaviors (Brewer & Hallman, 2006); delivering risk information for a similar other to override optimism about personal risks (Rimal & Morrison, 2006); using

prototypical people to trigger an assimilation rather than a contrast effect where people distance themselves from stereotypical exemplars (Menon et al., 2008; Zillmann, 2002).

Implementation and evaluation strategies. For audiences who underestimate their risks, messages focusing on beliefs, attitudes and norms or those tailored on personal risks are irrelevant (Witte & Allen, 2000). Thus, audience segmentation by positivity bias could improve the effectiveness of health interventions.

It is important to probe for a comprehensive list of individual difference variables (e.g., self efficacy, locus of control) and health risk characteristics (e.g., perceived risk controllability) that bias risk perceptions in the formative research phase of health interventions. Probing for predictors/correlates of underestimated risk perceptions is important because people are unlikely to elaborate on factors influencing a bias they are unaware of. Further, these variables should be assessed in the summative/evaluation phase of health interventions to detect unintended effects (e.g., desensitizing or overwhelming not-at-risk groups or those pessimistic about their risk) (Cho & Salmon, 2007).

Methodological implications

Health communication research should use priming and reaction times to advance our understanding of risk perceptions and health-related attitudes and behaviors. These methods uncover implicit cognitions, those beyond one's awareness of mental processes in place and override people's unwillingness to report these judgments (Bargh & Chartrand, 2000; Nisbett & Wilson, 1977; Wegner & Wheatley, 1999).

Limitations

Theoretical limitations.

This dissertation took an individual-level psychological approach to risk perceptions. It did not address cultural, anthropological, or sociological perspectives to risk perceptions (Lavino & Neumann, 2010).

As theorized here, personal health risk perceptions are subject to an efficient and out-of-awareness influence of self-schema activation. This by no means indicates that underestimation of risk is exclusively automatic. "Automatic processes, of whatever variety, do not occur in a vacuum, but in parallel or in combination with other ongoing automatic and controlled cognitive work" (Bargh, 1989, p. 27). In fact, the proposed model includes direct (automatic) and indirect (controlled) influences of self-schema activation on risk perceptions, both of which can be simultaneously at work.

Although this dissertation focused on underestimated risk perceptions, automatic self-schema activation can bias risk perceptions upward. In such instances, people overestimate their risks or exhibit a pessimistic bias. Research should explore individual difference variables (e.g., locus of control) (Hoorens & Buunk, 1993) and health risk characteristics (e.g., dread) (Slovic, 1987) that can alter the effect (direction and/or magnitude) of self-schema activation on risk perceptions.

Methodological limitations.

As with all experimental studies, the experiments conducted here are low on external validity (i.e., the extent to which the results are generalizable beyond the study participants) (Keppel & Wickens, 2004).

Selection of health traits in Pilot Study 3 and positive health conditions in Experiment 1 could have been the cause for slow reaction time data for health traits and positive conditions. Some health traits chosen for inclusion in Pilot Study 3 were not

frequently associated with health nor commonly used terms (e.g., "hale"). In Experiment 1, positive health conditions were created by rewording negative conditions to reflect a positive valence (e.g., healthy heart, healthy weight). As a result, response key and reaction time data for positive health conditions might have been driven by the length of the words common across positive health conditions.

Response key and reaction time data have limitations. The design of the experiments allowed participants to choose response key *yes* or *no*. Giving participants the choice of response key resulted in fewer data points for one response key when the majority of participants chose the other response key. For example, few participants responded *yes* in response to whether sickness traits were self-descriptive. As a result, few cases were available for analysis with the listwise deletion setting in statistical packages. A better approach could be the Implicit Association Test format. Although participants would be forced to give a certain response *yes* or *no* in any given trial, this method would ensure a complete data set of response key and reaction time data on all stimulus words. Further, reaction time, as a dependent variable, is potentially influenced by a host of variables other than the independent variable (e.g., age) (Nosek, Greenwald, & Banaji, 2007).

Results should be interpreted with caution if the data did not meet the assumptions underlying the statistical tests used. Further, several scales (e.g., health locus of control) had low internal reliabilities, which may have affected the rigor of the statistical tests. Finally, additional controls (e.g., race, ethnicity, language fluency) could have been included.

Conclusion

This dissertation took a psychological approach to explain why people underestimate health risks despite health communication efforts to inform them. It posited that people are predisposed to automatically underestimate self-relevant risks. Rationale for the theoretical propositions advanced here was derived from research documenting that health risk information is (a) self-relevant, (b) inconsistent with the positivity bias and self-threatening, and that (c) self-schema activation is automatic.

Three experiments were conducted. Experiment 1 tested the hypothesis that people underestimate their likelihood of experiencing negative health conditions and overestimate their likelihood of experiencing positive ones. Experiment 2 examined whether underestimation of health risks is an efficient process, one that occurs when people are mentally taxed. Finally, Experiment 3 tested the effects of health risk controllability and psychological reactance on health risk perceptions. In all three experiments, participants were primed with self-identity, third-person, or neutral words. Several individual difference variables (e.g., self efficacy, dispositional optimism) and characteristics of the health risk (e.g., prevalence) that influence risk perceptions were controlled for.

The experiments provided evidence of automatic underestimation of negative health conditions and overestimation of positive ones. Further, underestimation of risk is the default process when one is mentally taxed. Self-schema activation biased health risk perceptions for Cluster 1 health conditions. Finally, the experiments showed an inverted relationship between susceptibility and severity dimensions of health risk perceptions.

Together, these experiments show people automatically underestimate their health risks, that is, without awareness, intention, or effort. The automatic activation of health

risk perceptions is likely undetected because one is unaware of the activation of automatic processes and their effects on risk perceptions or of the triggers of these processes to begin with. Attempts to override or correct these automatic tendencies require awareness of the bias and its magnitude and direction, motivation to correct the bias and availability of processing resources (time and mental capacity). The presence of all these preconditions is unlikely, making biased health risk perceptions inevitable.

It is noteworthy that automatic processes are retained because they are functional. Both human processing abilities and information available to decision makers are limited. When a schema is constantly associated with a triggering environmental cue (e.g., a learned script on how to behave when checking in a clinic); when an object is always evaluated as positive or negative (e.g., health evaluated as positive and sickness as negative) (Slovic, Finucane, Peters, & MacGregor, 2004); and when a specific construct is frequently associated with a certain goal (e.g., self-enhancement goals), well-learned decisions migrate from consciousness to unconsciousness, which frees up the mind from thinking about them (Bargh, 2008; Baumeister, Bratslavsky, Muraven, & Tice, 1998).

The problem for health risk communication is with biased risk perceptions "rather than with their unconsciousness or automaticity per se" (Higgins, 1989, p. 76). Thus, to improve the effectiveness of health interventions, we need to understand and predict situations where these biases are most likely applied (Peters et al., 2006). Accordingly, health practitioners should design messages such that people are unlikely to be subject to decision biases or are likely to benefit from the intuitive/automatic information processing (Milkman, Chugh, & Bazerman, 2009).

The theoretical propositions advanced here suggest health risk perceptions are subject to positivity bias under both elaborative and non-elaborative modes of information processing: Underestimation of risks is the *default* option. The proposed model and agenda for health risk communication provides a road map that should advance our understanding of risk perceptions theoretically, practically, and methodologically.

APPENDIX 1:

TABLES AND FIGURES

Table 1: Summary results

Hypothesis	Results
PS3	Partially supported. Main effect for trait category was supported whereas the effect of self primes was not. Ps identified positive and health traits as self-descriptive and negative and sickness traits as non self-descriptive. Ps found it easier to identify positive and health traits as self-descriptive (as evidenced by faster reaction time) but more difficult to identify negative and sickness traits as self-descriptive (as evidenced by slower reaction time).
H1	Partially supported. Main effect for valence of health conditions was supported whereas the effect of self primes was not. Ps identified themselves as likely to experience positive health conditions and unlikely to experience negative ones. Not being susceptible to a negative health condition was the easiest of decisions participants had to make (as evidenced by faster reaction time). Identifying oneself as being susceptible to negative and positive conditions as well as not being susceptible to positive conditions was more difficult (as evidenced by slower reaction time).
H2a	Partially supported. Main effects for self primes were evident for susceptibility to Cluster 1 health conditions. Ps primed with self-identity words underestimated their susceptibility to Cluster 1 conditions. Main effects for cognitive load were evident for susceptibility to Clusters 2 and 3 and severity of Cluster 2 conditions. Ps under high cognitive load underestimated their susceptibility to Cluster 2 and 3 conditions and the severity of Cluster 2 conditions.
H2b	Partially supported. Self primes and high cognitive load interacted to bias severity of Cluster 1 health conditions downward. Ps primed with self-identity words underestimated the severity of Cluster 1 conditions when under high cognitive load.
H3a	Partially supported. Main effect for health risk controllability was supported whereas main effects for self primes and psychological reactance were not. Uncontrollable health conditions elicited perceptions of susceptibility to and severity of the disease.
H3b H3c H3d	Not supported.
H4	Supported. Participants underestimated their susceptibility to health conditions that scored low on prevalence, personal experience, controllability, stereotype salience, and exempt beliefs.
H5	Partially supported. Participants low on depression, high on self-efficacy, self-esteem, and dispositional optimism underestimated their susceptibility to negative health conditions.

Table 2: Study procedures

Experiment	Pre-measures	In-lab session				
		Welcome	Self-schema activation	Other independent variable manipulation	Dependent measures	
Experiment 1	Depression Inventory Dispositional optimism Health locus of control Psychological Reactance Self-consciousness Self-efficacy Self-esteem	Initial greeting, consent, and instructions	Supraliminal priming Subliminal priming None		Susceptibility to positive and negative health conditions Response key Reaction time	Debriefing
Experiment 2			Subliminal priming	Cognitive load (high vs. low)	Susceptibility to and severity of negative health conditions Response key Reaction time	
Experiment 3	Independent variable (measured) Psychological Reactance		Subliminal priming	Health risk controllability (controllable vs. uncontrollable)	Susceptibility to and severity of health condition Response key Reaction time Perceived risk controllability Behavioral measure	

Table 3: Zero-order correlation matrix for health risk characteristics, personal and third-person susceptibility and severity (Pilot studies 1 and 2)

	1	2	3	4	5	6	7	8	9	10	11
1. Prevalence $_{PS1}$	1										
2. Stereotype salience $_{PS1}$	.71**	1									
3. Controllability $_{PS1}$	.41**	.54**	1								
4. Exempt beliefs $_{PS1}$	.81**	.70**	.51**	1							
5. Personal experience $_{PS1}$	.86**	.70**	.18	.69**	1						
6. Personal susceptibility $_{PS1}$	.84**	.65**	.24*	.82**	.83**	1					
7. Personal severity$_{PS1}$	-.41**	-.18	-.14	-.25*	-.36**	-.38**	1				
8. Personal susceptibility$_{PS2}$	.87**	.64**	.25*	.73**	.86**	.81**	-.36**	1			
9. Personal severity$_{PS2}$	-.57**	-.40**	-.14	-.47**	-.56**	-.55**	.78**	-.56**	1		
10. Third-person susceptibility $_{PS2}$	.85**	.72**	.47**	.68**	.73**	.66**	-.37**	.85**	-.52**	1	
11. Third-person severity $_{PS2}$	-.55**	-.37**	-.11	-.45**	-.55**	-.53**	.77**	-.55**	.96**	-.47**	1
M	25.37	3.99	3.18	3.32	1.58	2.85	4.52	3.08	5.07	3.46	5.10
SD	20.61	1.28	1.38	1.11	1.55	1.26	1.02	1.28	1.10	1.30	1.10
N	86	86	86	86	86	86	86	93	93	93	93

N varied according to familiarity frequencies.

* $p < .05$, two-tailed. ** $p < .01$, two-tailed.

PS1 represents data from pilot study 1 whereas PS2 is data from pilot study 2.

Prevalence rates were measured on a 0 – 100 scale. Susceptibility and exempt belief were on a 0 – 7 scale, where 0 = *impossible* and 7 = *very likely*. Stereotype salience, controllability, and severity were on a 1 – 7 scale, where 7 represented ease of imaging a person who could suffer from the disease, high perceived risk controllability, and extreme severity. Personal experience was on 0 – 6 scale, where 0 = *has not happened to anyone I know before* to 6 = *has happened to me more than once*.

Table 4: Cluster analysis: Descriptive statistics and differences on health risk characteristics (Pilot study 1)

Variable	Cluster 1 $n = 17$	Cluster 2 $n = 26$	Cluster 3 $n = 7$	Equality of means test[*]
Prevalence[**]	15.22 (5.01)[e]	32.51 (12.86)[f]	77.34 (10.09)[g]	B-$F_{(2, 21.767)} = 104.15$
Stereotype salience	3.21 (.53)[a]	4.87 (.81)[b]	5.81 (.62)[c]	$F_{(2, 47)} = 43.73$
Controllability	2.50 (1.32)[a]	3.75 (1.07)[b]	4.60 (1.02)[b]	$F_{(2, 47)} = 10.01$
Exempt beliefs	2.67 (.48)[a]	3.91 (.47)[b]	5.68 (.45)[c]	$F_{(2, 47)} = 103.05$
Personal experience[**]	.70 (.47)[e]	2.07 (1.03)[f]	5.12 (.64)[g]	B-$F_{(2, 31.417)} = 95.39$

$N = 50$ diseases.
Cells represent means (and standard deviations).
[*] $p < .001$.
[**] Equal variances were not assumed as evidenced by a significant Levene's test for prevalence ($F(2, 47) = 4.11$, $p = .023$) and personal experience ($F(2,47) = 3.65$, $p = .033$). Brown-Forsythe, a robust test of equality of means, was reported.
[a, b, c] Groups statistically different at .05 level based on Tukey's HSD post hoc analysis.
[e, f, g] Groups statistically different at .05 level based on Dunnett C post hoc analysis for heterogeneous variances.
Prevalence rates were measured on a 0 – 100 scale, where 100 reflect high prevalence rates. Susceptibility and exempt belief were on a 0 – 7 scale, where 0 = *impossible* and 7 = *very likely*. Stereotype salience, controllability, and severity were on a 1 – 7 scale, where 7 represented ease of imaging a person who could suffer from the disease, high perceived risk controllability, and extreme severity. Personal experience was on 0 – 6 scale, where 0 = *has not happened to anyone I know before* to 6 = *has happened to me more than once*.

Table 5: Zero-order correlations for perceived health risk controllability dimensions (Pilot study 2)

	1	2	3	4	5	6	7	8	9	10
1. Personal susceptibility control	1									
2. Personal severity control	.45	1								
3. Personal progression rate control	.52	.85	1							
4. Personal treatment effectiveness control	.49	.82	.82	1						
5. Personal general control	.63	.81	.82	.83	1					
6. Third-person susceptibility control	.79	.48	.54	.50	.65	1				
7. Third-person severity control	.46	.81	.79	.77	.75	.49	1			
8. Third-person progression rate control	.53	.83	.86	.85	.78	.52	.90	1		
9. Third-person treatment effectiveness control	.54	.79	.82	.79	.76	.50	.79	.84	1	
10. Third-person general control	.61	.71	.73	.75	.83	.59	.73	.80	.72	1
M	3.09	2.73	3.21	3.23	2.99	3.00	2.71	3.10	3.20	2.89
SD	1.71	1.80	2.00	1.97	1.86	1.68	1.56	1.80	1.71	1.74

N = 70.
All correlations are significant at .05 level, 2-tailed.
Perceived health risk controllability was measured on a 1 – 7 scale, where 7 = *very easy to control*.

Table 6: Factor loadings for perceived health risk controllability (Pilot study 2)

Item	General control	Susceptibility control
1. Personal susceptibility control	.00	***.94***
2. Personal severity control	***.97***	-.09
3. Personal progression rate control	***.92***	.01
4. Personal treatment effectiveness control	***.93***	-.02
5. Personal general control	***.73***	.28
6. Third-person susceptibility control	.02	***.92***
7. Third-person severity control	***.95***	-.07
8. Third-person progression rate control	***.96***	-.01
9. Third-person treatment effectiveness control	***.89***	.01
10. Third-person general control	***.68***	.28
Rotation sums of squared loadings	7.232	4.331
Reliability	$\alpha = .96$	$r = .79$
Factor correlation	.58	

N = 70.
Solution is based on a Principal Components Analysis oblique rotation.
Factor loadings > .30 are in boldfaced.

Table 7: Mean response key descriptive statistics (Pilot study 3)

	Trait category				*M* 95% CI [LL – UL]
	Positive	Negative	Health	Sickness	
Self condition (*n* = 75)					
Self-descriptive	.89 (.13)	.08 (.10)	.70 (.18)	.06 (.11)	.43 [.42 – .45]
Non self-descriptive	.10 (.13)	.91 (.10)	.29 (.18)	.93 (.11)	.56 [.54 – .57]
Neutral condition (*n* = 75)					
Self-descriptive	.91 (.11)	.10 (.15)	.71 (.20)	.04 (.09)	.44 [.42 – .45]
Non self-descriptive	.08 (.11)	.89 (.15)	.28 (.20)	.95 (.09)	.55 [.54 – .57]
Self-descriptive					
M	.90	.09	.70	.05	
95% CI [LL – UL]	[.88 – .92]	[.07 – .11]	[.67 – .74]	[.03 – .07]	
Non self-descriptive					
M	.09	.90	.29	.94	
95% CI [LL – UL]	[.07 – .11]	[.88 – .92]	[.26 – .32]	[.92 – .96]	

N = 150.
Cells represent means (and standard deviations).

Table 8: Reaction time descriptive statistics (Pilot study 3)

	Trait category				*M* 95% CI [LL – UL]
	Positive	Negative	Health	Sickness	
Self condition (*n* = 75)	857.03 (1.21)	936.26 (1.23)	991.51 (1.24)	910.33 (1.24)	922.57 [885.11 – 961.61]
Self-descriptive	842.36 (1.20) *n* = 75	1119.95 (1.33) *n* = 49	958.95 (1.24) *n* = 74	1390.27 (1.40) *n* = 27	1088.93 [993.11 – 1191.24]
Non self-descriptive	1036.09 (1.39) *n* = 50	926.40 (1.24) *n* = 75	1090.18 (1.34) *n* = 74	897.22 (1.23) *n* = 75	993.11 [933.25 – 1056.81]
Neutral condition (*n* = 75)	883.07 (1.21)	931.32 (1.18)	987.18 (1.20)	898.87 (1.17)	924.69 [887.15 – 963.82]
Self-descriptive	867.96 (1.20) *n* = 75	1149.74 (1.28) *n* = 49	953.45 (1.22) *n* = 73	1213.38 (1.27) *n* = 18	1061.69 [954.99 – 1177.60]
Non self-descriptive	1180.04 (1.33) *n* = 44	923.42 (1.19) *n* = 75	1142.08 (1.35) *n* = 72	889.40 (1.17) *n* = 75	1051.96 [981.74 – 1124.60]
Combined reaction time					
M	868.96	933.25	988.55	903.64	
95% CI [LL – UL]	[843.33 – 897.42]	[905.73 – 963.82]	[957.19 – 1023.29]	[877.00 – 933.25]	
Reaction time (*yes*)					
M	877.00	1202.26	990.83	1273.50	
95% CI [LL – UL]	[826.03 – 931.10]	[1086.42 – 1333.52]	[918.33 – 1071.51]	[1142.87 – 1415.79]	
Reaction time (*no*)					
M	1099.00	963.82	1124.60	916.22	
95% CI [LL – UL]	[1030.38 – 1169.49]	[922.57 – 1009.25]	[1059.25 – 1193.98]	[879.02 – 954.99]	

N = 150.

Cells represent mean reaction time in milliseconds (and standard deviations).

Analysis was performed on log-transformed data. Reported means and standard deviations have been back transformed to milliseconds using $10^{\wedge}$ formula.

Table 9: Zero-order correlation matrix for individual difference variables (Experiment 1)

	1	2	3	4	5	6	7	8	9	10
1. Depression inventory	1									
2. Life orientation test	-.44**	1								
3. Health locus of control	-.00	.09	1							
4. Psychological reactance	.22**	-.12*	.07	1						
5. Self reflection	.21**	-.14**	.02	.19**	1					
6. Self awareness	.00	.15**	.06	.11*	.30**	1				
7. Self consciousness	.32**	-.26**	-.06	.10	.53**	.35**	1			
8. Self efficacy	-.37**	.44**	.16**	-.05	-.05	.27**	-.25**	1		
9. Self esteem	-.47**	.64**	.09	-.04	-.24**	.18**	-.37**	.55**	1	
10. Gender	.03	-.05	.00	-.10	-.05	.04	.03	.00	-.05	1
M	1.36	2.54	3.85	3.07	2.63	2.91	2.53	3.59	3.04	.74
SD	.29	.65	.49	.46	.86	.51	.37	.50	.46	.43
Scale Statistics										
# of items	20	6	11	14	2	4	23	17	10	-
Reliability[a]	.85	.79	.58	.78	.49	.54	.74	.86	.86	-

N = 325.
* $p < .05$, two-tailed. ** $p < .01$, two-tailed.
[a] Cronbach's alpha is reported for all scales except for the two-item self-reflection scale for which a correlation is reported.
Gender: 0 = *male*, 1= *female*.

Table 10: Factor loadings for health locus of control scale (Experiments 1, 2, and 3)

	Experiment 1		Experiment 2		Experiment 3	
Item	Internal	External	Internal	External	Internal	External
1. If I take care of myself, I can avoid illness	**.44**	.13	**.58**	.14	-.44	.41
2. Whenever I get sick it is because something I've done or not done	**.55**	-.13	**.57**	-.13	**-.79**	-.06
3. Good health is largely a matter of good fortune	.11	**.49**	.03	**.48**	.09	**.56**
4. No matter what I do, if I am going to get sick I will get sick	.28	**.48**	.23	**.39**	-.11	**.49**
5. Most people do not realize the extent to which their illnesses are controlled by accidental happenings	-.05	**.38**	-.13	**.38**	-.04	**.49**
6. I can only do what my doctor tells me to do	-.12	**.33**	-.13	**.39**	.20	**.51**
7. There are so many strange diseases that you can never know how or when you might pick one up	-.05	.24	.06	**.37**	-.11	**.49**
8. When I feel ill, I know it is because I have not been getting the proper exercise or eating right	**.48**	.01	**.52**	-.05	**-.58**	-.07
9. People who never get sick are just plain lucky	.20	**.50**	.20	**.45**	-.10	**.44**
10. People's ill health results from their own carelessness	**.52**	-.18	**.52**	-.01	**-.55**	.02
11. I am directly responsible for my health	**.46**	.16	**.50**	.11	**-.48**	.15
Rotation sums of squared loadings	1.425	1.200	1.678	1.163	2.038	1.950
Reliability	.60	.56	.67	.56	.69	.67
Factor correlation	.08		.13		-.25	

N = 325, 476, and 90 for Experiments 1, 2, and 3, respectively.
Solution is based on a Principal Axis Factoring oblique rotation.
Factor loadings > .30 are in boldfaced.

Table 11: Factor loadings for self-consciousness scale (Experiment 1)

Item	Social Anxiety	Public Self	Private Self	Self awareness	Self reflection
1. I'm always trying to figure myself out	.15	.15	**.59**	-.07	**-.82**
2. I'm concerned about my style of doing things	.04	**.38**	.16		
3. Generally, I'm not very aware of myself	-.12	.17	**.39**	**.42**	-.05
4. It takes me time to overcome my shyness in new situations	**.73**	.00	.02		
5. I reflect about myself a lot	.13	.06	**.77**	.37	-.47
6. I'm concerned about the way I present myself	-.03	**.63**	.16		
7. I'm often the subject of my own fantasies	-.03	.13	.12		
8. I have trouble working when someone is watching me	**.35**	.09	.06		
9. I never scrutinize myself	.22	.20	.25		
10. I get embarrassed very easily	**.58**	.32	-.08		
11. I'm self-conscious about the way I look	.27	**.58**	.01		
12. I don't find it hard to talk to strangers	**.63**	-.11	-.06		
13. I'm generally attentive to my inner feelings	-.07	.01	**.46**	**.71**	.11
14. I usually worry about making a good impression	.10	**.52**	.09		
15. I'm constantly examining my motives	.03	.14	**.56**	.04	**-.61**
16. I feel anxious when I speak in front of a group	**.57**	.03	-.12		
17. One of the last things I do before I leave my house is look in the mirror	.01	**.55**	-.02		
18. I sometimes have the feeling that I'm off somewhere watching myself	.03	.04	.24		
19. I'm concerned about what other people think of me	.20	**.57**	.01		
20. I'm alert to changes in my mood	-.00	.00	**.38**	**.49**	.00
21. I'm usually aware of my appearance	-.09	**.50**	.12		
22. I'm aware of the way my mind works when I work through a problem	-.18	-.01	**.34**	**.30**	-.08
23. Large groups make me nervous	**.73**	.06	.09		
Rotation sums of squared loadings	2.567	2.338	2.157	1.608	1.712
Reliability[a]	.77	.73	.70	.54	.49
Factor correlation				-.49	

N = 325.
[a] Cronbach's alpha is reported for all scales except for the two-item self-reflection scale for which a correlation is reported.
Solution is based on a Principal Axis Factoring varimax rotation.
Solution for private self subscales (self-refection and self-awareness) is based on a Principal Axis Factoring oblique rotation.
Factor loadings > .30 are in boldfaced.

Table 12: Standardized canonical coefficients, canonical correlation, and proportions of variance between individual differences and mean response key *no* to susceptibility to positive and negative health conditions (Experiment 1)

	First canonical variate	
	Correlations	Coefficients
Individual differences set		
Depression inventory	**-.75**	-.40
Life orientation test	**.71**	.16
Health locus of control	.25	.15
Psychological reactance	-.13	.00
Self reflection	-.16	.05
Self awareness	.08	-.19
Self consciousness	**-.32**	.15
Self efficacy	**.73**	.33
Self esteem	**.83**	.43
Percent variance	53.29	
Mean response key		
Susceptibility to positive conditions	-.28	-.12
Susceptibility to negative conditions	**.99**	.97
Percent variance	2.62	
Canonical correlation	.30	

N = 325.

Table 13: Mean response key descriptive statistics (Experiment 1)

	Valence of health condition				*M* 95% CI [LL – UL]
	Positive		Negative		
	Subliminal	Supraliminal	Subliminal	Supraliminal	
Self condition (*n* = 93)					
Yes	.89 (.11)	.93 (.08)	.22 (.11)	.19 (.09)	.56 [.54 – .58]
No	.10 (.11)	.06 (.08)	.77 (.11)	.80 (.09)	.43 [.41 – .45]
Third-person condition (*n* = 93)					
Yes	.91 (.11)	.90 (.10)	.24 (.14)	.26 (.17)	.58 [.56 – .59]
No	.08 (.11)	.10 (.10)	.75 (.14)	.73 (.17)	.42 [.40 – .43]
Neutral condition (*n* = 93)					
Yes	.90 (.17)	.90 (.09)	.23 (.15)	.25 (.12)	.57 [.55 – .59]
No	.10 (.17)	.09 (.09)	.76 (.15)	.74 (.12)	.42 [.40 – .44]
Yes					
M	.90	.91	.23	.23	
95% CI [LL – UL]	[.88 – .92]	[.89 – .93]	[.21 – .26]	[.21 – .26]	
No					
M	.09	.08	.76	.76	
95% CI [LL – UL]	[.07 – .11]	[.06 – .10]	[.74 – .78]	[.73 – .78]	

N = 279.
Cells represent means (and standard deviations).

Table 14: Reaction time descriptive statistics (Experiment 1)

	Valence of health condition				*M* 95% CI [LL – UL]
	Positive		Negative		
	Subliminal	Supraliminal	Subliminal	Supraliminal	
Self condition (*n* = 93)	993.11	968.27	974.98	939.72	968.27 [933.25 – 1006.93]
Response key "*yes*"	941.88	957.19	993.11	1009.25	
Response key "*no*"	1006.93	1000.00	914.11	916.22	
Third-person condition (*n* = 93)	993.11	1076.46	968.27	993.11	1006.93 [970.50 – 1044.72]
Response key "*yes*"	1018.59	1101.53	1035.14	1064.14	
Response key "*no*"	1183.04	1056.81	977.23	981.74	
Neutral condition (*n* = 93)	954.99	1044.72	920.44	979.48	974.98 [937.56 – 1011.57]
Response key "*yes*"	831.76	1066.59	997.70	1037.52	
Response key "*no*"	986.27	1099.00	847.22	948.41	
Combined reaction time					
M	979.48	1028.01	954.99	970.50	
95% CI [LL – UL]	[948.41 – 1013.91]	[993.11 – 1064.14]	[926.82 – 988.55]	[941.88 – 1000]	
Reaction time (*yes*)					
M	957.19	1039.92	1009.25	1037.52	
95% CI [LL – UL]	[909.91 – 1006.93]	[990.83 – 1091.44]	[966.05 – 1051.96]	[995.40 – 1081.43]	
Reaction time (*no*)					
M	1054.38	1051.96	912.01	948.41	
95% CI [LL – UL]	[968.27 – 1150.80]	[966.05 – 1142.87]	[868.96 – 957.19]	[905.41 – 993.11]	

N = 279.

Cells represent mean reaction time in milliseconds.

Analysis was performed on log-transformed data. Reported means have been back transformed to milliseconds using $10^{\wedge}$ formula.

Table 15: Zero-order correlation matrix for individual difference variables (Experiment 2)

	1	2	3	4	5	6	7	8	9	10
1. Depression inventory	1									
2. Life orientation test	-.49**	1								
3. Health locus of control	-.17**	.23**	1							
4. Psychological reactance	.19**	-.18**	-.03	1						
5. Self reflection	.24**	-.13**	-.07*	.18**	1					
6. Self awareness	-.10*	.17**	.02	.02	.33**	1				
7. Self consciousness	.29**	-.27**	-.09*	.10*	.64**	.27**	1			
8. Self efficacy	-.41**	.50**	.16**	-.12**	-.11**	.28**	-.28**	1		
9. Self esteem	-.56**	.63**	.12**	-.11*	-.21**	.25**	-.35**	.59**	1	
10. Gender	.12**	-.01	-.10*	-.05	.06	.01	.22**	-.03	-.03	1
M	1.35	2.61	3.79	3.00	2.72	2.92	2.49	3.66	3.08	.76
SD	.28	.62	.50	.45	.66	.48	.39	.46	.44	.42
Scale Statistics										
# of items	20	6	11	14	4	4	23	17	10	-
α	.83	.82	.62	.78	.69	.60	.78	.84	.86	-

N = 476.
* $p < .05$, two-tailed. ** $p < .01$, two-tailed.
Gender: 0 = *male*, 1= *female*.

Table 16: Factor loadings for self-consciousness scale (Experiment 2)

Item	Social Anxiety	Public Self	Private Self	Self reflection	Self awareness
1. I'm always trying to figure myself out	.27	.10	**.61**	**.78**	-.08
2. I'm concerned about my style of doing things	.24	.19	.13		
3. Generally, I'm not very aware of myself	-.25	.17	**.35**	-.04	**.57**
4. It takes me time to overcome my shyness in new situations	**.77**	.04	.04		
5. I reflect about myself a lot	.05	.18	**.69**	**.58**	.23
6. I'm concerned about the way I present myself	.19	**.56**	.16		
7. I'm often the subject of my own fantasies	.01	.17	.19		
8. I have trouble working when someone is watching me	**.39**	.14	.04		
9. I never scrutinize myself	.16	.15	**.37**	**.40**	.03
10. I get embarrassed very easily	**.60**	.31	.06		
11. I'm self-conscious about the way I look	.29	**.49**	.09		
12. I don't find it hard to talk to strangers	**.59**	.02	-.14		
13. I'm generally attentive to my inner feelings	-.15	.13	**.55**	.16	**.57**
14. I usually worry about making a good impression	.15	**.60**	.09		
15. I'm constantly examining my motives	.15	.01	**.50**	**.57**	-.03
16. I feel anxious when I speak in front of a group	**.57**	.15	-.09		
17. One of the last things I do before I leave my house is look in the mirror	.02	**.54**	.01		
18. I sometimes have the feeling that I'm off somewhere watching myself	.19	.02	.23		
19. I'm concerned about what other people think of me	.23	**.62**	.05		
20. I'm alert to changes in my mood	-.11	.01	**.37**	.01	**.50**
21. I'm usually aware of my appearance	-.11	**.59**	.09		
22. I'm aware of the way my mind works when I work through a problem	-.15	-.06	**.33**	-.02	**.43**
23. Large groups make me nervous	**.66**	.01	.04		
Rotation sums of squared loadings	2.817	2.305	2.146	1.810	1.541
Reliability	.79	.74	.70	.69	.60
Factor correlation				.42	

N = 476.
Solution is based on a Principal Axis Factoring varimax rotation.
Solution for private self subscales (self refection and self awareness) is based on a Principal Axis Factoring oblique rotation.

Table 17: Standardized canonical coefficients, canonical correlation, and proportions of variance between individual differences and mean response key *no* to susceptibility and severity of health conditions (Experiment 2)

	First canonical variate		Second canonical variate	
	Correlations	Coefficients	Correlations	Coefficients
Individual differences set				
Depression inventory	**.84**	.67	.01	-.32
Life orientation test	**-.60**	-.09	**-.38**	-.61
Health locus of control	**-.40**	-.24	-.18	-.12
Psychological reactance	-.05	-.19	.24	.10
Self reflection	.13	-.38	.66	.77
Self awareness	-.07	.07	.15	-.12
Self consciousness	**.46**	.40	**.33**	-.11
Self efficacy	**-.46**	.04	.28	.75
Self esteem	**-.67**	-.22	-.20	-.22
Percent variance	1.93		.56	
Mean response key				
Susceptibility to Cluster 1 health conditions	-.28	1.63	-.05	.53
Severity of Cluster 1 health conditions	.20	.50	**.31**	-.05
Susceptibility to Cluster 2 health conditions	**-.64**	-2.15	-.06	-.44
Severity of Cluster 2 health conditions	-.04	-.53	.00	.55
Susceptibility to Cluster 3 health conditions	**-.34**	.09	**.32**	.42
Severity of Cluster 3 health conditions	-.04	.29	**-.78**	-1.12
Percent variance	11.04		13.77	
Canonical correlation	.28		.23	

N = 476.

Table 18: Mean response key *yes* descriptive statistics (Experiment 2)

	Prime			
	Self (n = 160)	Third-person (n = 158)	Neutral (n = 158)	*M* 95% [LL – UL]
Susceptibility				
High load (n = 238)				
Cluster 1	.14 (.21)	.20 (.25)	.21 (.27)	.18 [.15 – .22]
Cluster 2	.26 (.18)	.31 (.21)	.26 (.22)	.28 [.25 – .30]
Cluster 3	.53 (.31)	.55 (.29)	.52 (.32)	.53 [.50 – .57]
Low load (n = 238)				
Cluster 1	.15 (.24)	.18 (.23)	.24 (.29)	.19 [.16 – .22]
Cluster 2	.28 (.21)	.34 (.20)	.34 (.24)	.32 [.29 – .35]
Cluster 3	.65 (.24)	.68 (.25)	.63 (.23)	.65 [.62 – .69]
Severity				
High load (n = 238)				
Cluster 1	.54 (.35)	.60 (.31)	.55 (.29)	.56 [.52 – .59]
Cluster 2	.36 (.23)	.42 (.22)	.40 (.22)	.39 [.37 – .42]
Cluster 3	.14 (.20)	.11 (.14)	.14 (.20)	.13 [.11 – .15]
Low load (n = 238)				
Cluster 1	.74 (.22)	.66 (.26)	.79 (.17)	.73 [.69 – .76]
Cluster 2	.51 (.17)	.51 (.18)	.57 (.15)	.53 [.50 – .56]
Cluster 3	.14 (.19)	.15 (.17)	.18 (.20)	.16 [.13 – .18]

N = 476.
Cells represent means (and standard deviations).

Table 19: Reaction time descriptive statistics (Experiment 2)

	Prime				
	Self	Third-person	Neutral	*Yes* 95% [LL – UL]	*No* 95% [LL – UL]
Susceptibility					
High load					
Cluster 1	1116.86	1061.69	1061.69	1258.92 [1202.26 – 1318.25]	1191.24 [1137.62 – 1250.25]
Cluster 2	1099.00	1056.81	1069.05	1127.19 [1086.42 – 1169.49]	1164.12 [1114.29 – 1218.98]
Cluster 3	1064.14	1091.44	1061.69	1059.25 [1016.24 – 1104.07]	1180.32 [1124.60 – 1241.65]
Low load					
Cluster 1	1039.92	1042.31	979.48	1267.65 [1213.38 – 1324.34]	1122.01 [1073.98 – 1174.89]
Cluster 2	1030.38	1013.91	977.23	1101.53 [1064.14 – 1140.24]	1091.44 [1047.12 – 1140.24]
Cluster 3	1059.25	1020.93	966.05	1016.24 [977.23 – 1056.81]	1127.19 [1076.46 – 1183.04]
Severity					
High load					
Cluster 1	1056.81	1035.14	1028.01	1042.31 [995.40 – 1091.44]	1210.59 [1145.51 – 1279.38]
Cluster 2	1069.05	1061.69	1042.31	1069.05 [1025.65 – 1114.29]	1135.01 [1088.93 – 1183.04]
Cluster 3	977.23	1020.93	1035.14	1086.42 [1018.59 – 1158.77]	1042.31 [997.70 – 1091.44]
Low load					
Cluster 1	1047.12	1006.93	946.23	1020.93 [979.48 – 1064.14]	1169.49 [1114.29 – 1227.43]
Cluster 2	1096.47	1030.38	988.55	1042.31 [1004.61 – 1083.92]	1111.73 [1071.51 – 1153.45]
Cluster 3	1037.52	1011.57	948.41	1086.42 [1025.65 – 1148.15]	1013.91 [974.98 – 1054.38]

N = 476.
Cells represent means.

Table 20: Zero-order correlation matrix for individual difference variables (Experiment 3)

	1	2	3	4	5	6	7	8	9	10
1. Depression inventory	1									
2. Life orientation test	-.59**	1								
3. Health locus of control	.01	.07	1							
4. Psychological reactance	.39**	-.45**	.06	1						
5. Self reflection	.32**	-.23*	.12	.30**	1					
6. Self awareness	-.12	-.01	.04	.08	.23*	1				
7. Self consciousness	.47**	-.28**	.04	.31**	.61**	.30**	1			
8. Self efficacy	-.45**	.32**	.15	-.02	-.01	.13	-.24*	1		
9. Self esteem	-.66**	.66**	.08	-.38**	-.19	.12	-.33**	.64**	1	
10. Gender	.06	.16	-.11	-.21*	.01	-.04	.13	-.07	.08	1
M	1.41	2.56	3.88	3.00	2.85	3.10	2.59	3.60	3.10	
SD	.34	.77	.61	.54	.62	.57	.37	.52	.50	
Scale Statistics										
# of items	20	6	11	14	4	2	23	17	10	-
Reliability[a]	.87	.85	.73	.83	.70	.31	.74	.87	.89	-

$N = 90$.
* $p < .05$, two-tailed. ** $p < .01$, two-tailed.
[a] Cronbach's alpha is reported for all scales except for the two-item self-reflection scale for which a correlation is reported.
Gender: 0 = *male*, 1= *female*.

Table 21: Factor loadings for self-consciousness scale (Experiment 3)

Item	Social Anxiety	Public Self	Private Self	Self reflection	Self awareness
1. I'm always trying to figure myself out	.27	.24	**.56**	**.81**	-.09
2. I'm concerned about my style of doing things	.01	**.46**	.01		
3. Generally, I'm not very aware of myself	-.06	.03	**.51**	.21	**.41**
4. It takes me time to overcome my shyness in new situations	**.84**	.00	-.11		
5. I reflect about myself a lot	-.03	.06	**.61**	**.56**	.14
6. I'm concerned about the way I present myself	.20	**.46**	.20		
7. I'm often the subject of my own fantasies	-.07	.05	.10		
8. I have trouble working when someone is watching me	.28	.09	.05		
9. I never scrutinize myself	.15	.20	**.31**	**.49**	-.10
10. I get embarrassed very easily	**.48**	.29	.16		
11. I'm self-conscious about the way I look	.18	**.84**	.06		
12. I don't find it hard to talk to strangers	**.71**	.10	-.12		
13. I'm generally attentive to my inner feelings	-.11	.12	**.63**	.36	.40
14. I usually worry about making a good impression	.12	**.34**	.10		
15. I'm constantly examining my motives	.15	.04	**.65**	**.59**	.14
16. I feel anxious when I speak in front of a group	**.53**	.20	-.09		
17. One of the last things I do before I leave my house is look in the mirror	-.01	**.49**	.03		
18. I sometimes have the feeling that I'm off somewhere watching myself	.01	.12	.34	.18	.24
19. I'm concerned about what other people think of me	.21	**.64**	-.01		
20. I'm alert to changes in my mood	-.03	-.15	**.34**	-.19	**.76**
21. I'm usually aware of my appearance	-.09	**.32**	.19		
22. I'm aware of the way my mind works when I work through a problem	-.21	-.21	.24		
23. Large groups make me nervous	**.57**	-.00	.07		
Rotation sums of squared loadings	2.501	2.377	2.375	2.082	1.378
Reliability[a]	.77	.71	.71	.70	.31
Factor correlation				.33	

$N = 90$.

[a] Cronbach's alpha is reported for all scales except for the two-item self-reflection scale for which a correlation is reported.

Solution is based on a Principal Axis Factoring varimax rotation.

Solution for private self subscales (self refection and self awareness) is based on a Principal Axis Factoring oblique rotation.

Table 22: Logistic regression analysis of susceptibility, severity, and behavioral measure as a function of individual difference variables and experimental treatment (Experiment 3)

	Susceptibility		Severity		Behavioral measure	
	B	χ^2	*B*	χ^2	B	χ^2
1. Depression inventory	2.06	1.73	3.24	5.72*	.47	.13
2. Life orientation test	.22	.08	-.04	.006	.53	.80
3. Health locus of control	-.87	2.61	.02	.004	-.34	.46
4. Psychological reactance	.27	.10	-.61	1.03	-.91	1.89
5. Self reflection	1.54	5.36*	-.83	1.68	1.25*	4.79*
6. Self awareness	.42	.38	1.92	9.89**	1.90**	8.84**
7. Self consciousness	-.41	.10	-1.65	1.94	-.76	.44
8. Self efficacy	-.06	.005	1.55	3.91*	-.54	.46
9. Self esteem	1.36	1.11	-.68	.43	-.46	.19
10. Gender	-.93	1.37	.77	1.38	1.43*	4.83*
Model χ^2 (*df* = 10)		11.30		22.99		17.20
11. Self prime	.93	1.64	.43	3.04	.25	.84
12. Neutral prime	-.11		-.76		.68	
13. Risk controllability (uncontrollable)	1.90	7.47**	1.35*	5.36*	1.53*	6.56*
Model χ^2 (*df* =13)		20.46		30.90		24.78

N = 90.
* *p* < .05. ** *p* < .01. *** *p* < .001.
χ^2 to remove variable from the model.

Figure 1. Perceived health risk susceptibility control: Manipulation check (Pilot Study 2)

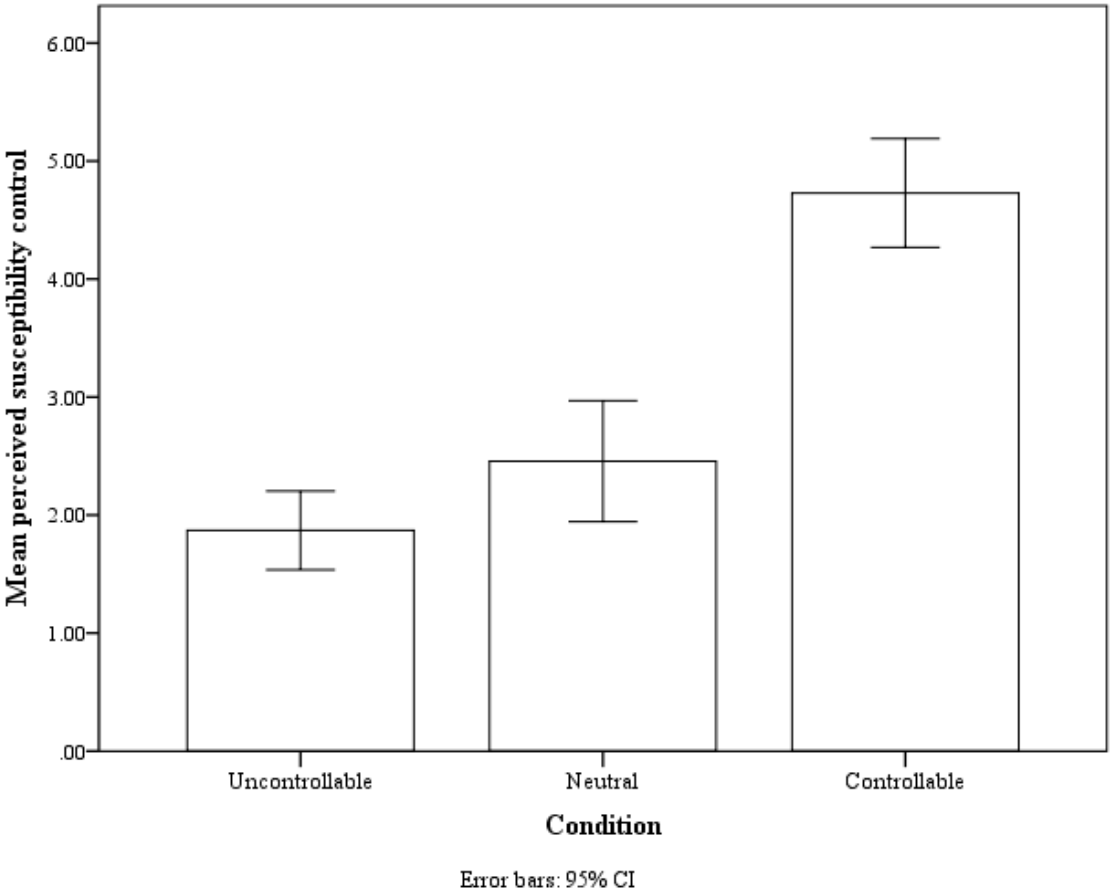


Figure 2. Mean reaction time by prime (self vs. neutral) and trait category (Pilot Study 3)

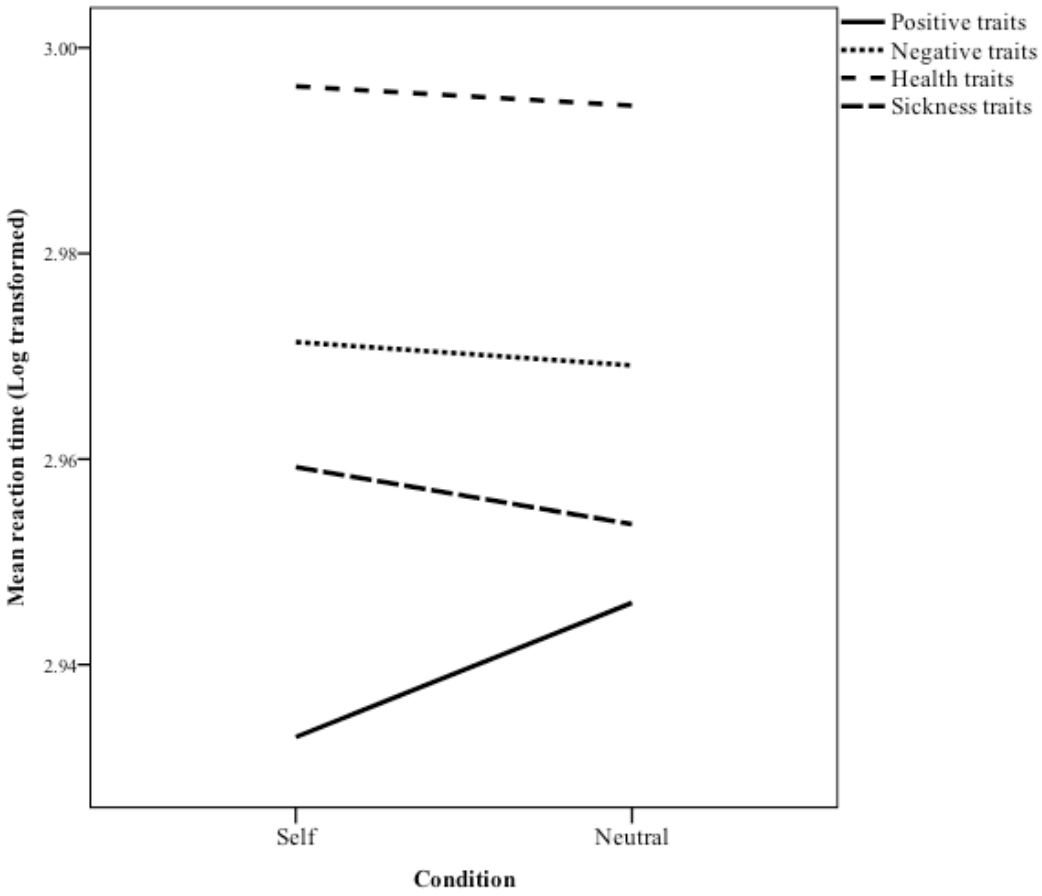


Figure 3a. Mean reaction time by trait category and response key split by *yes* and *no* (Pilot Study 3: Self condition)

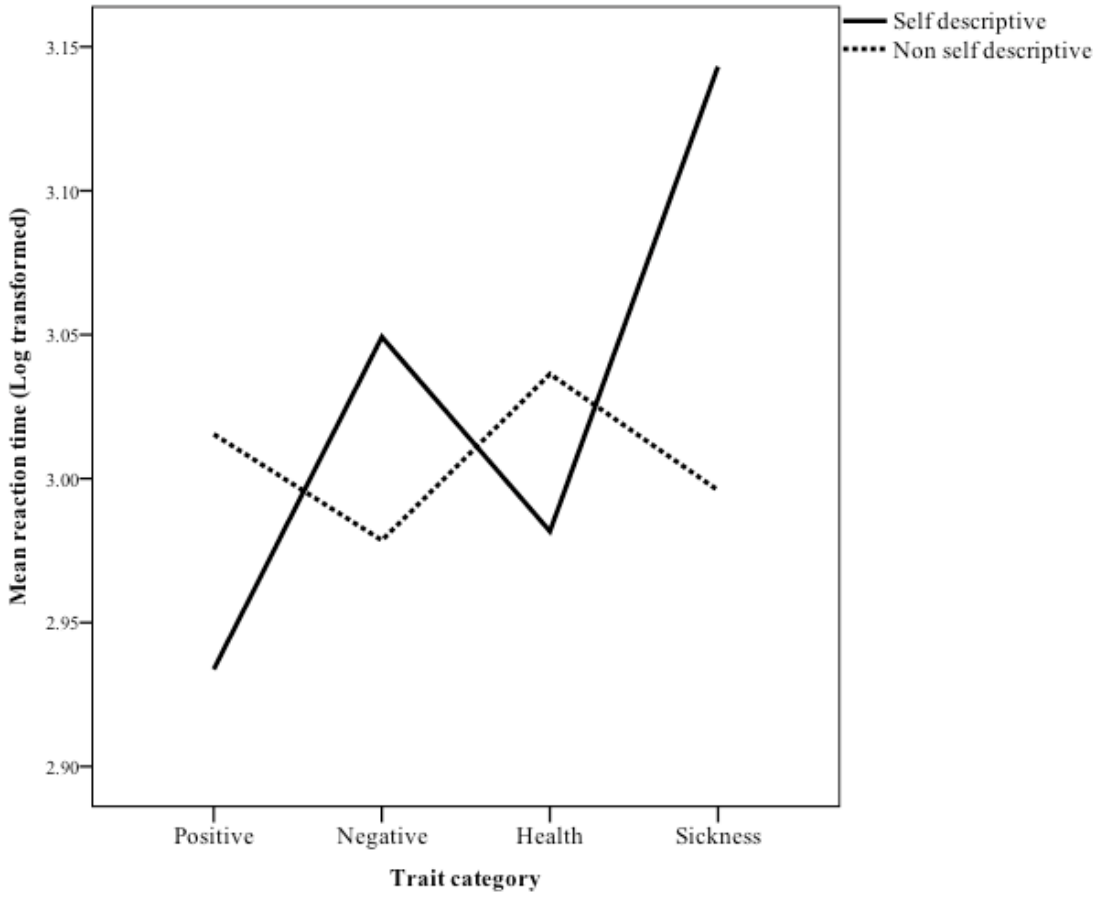


Figure 3b. Mean reaction time by trait category and response key split by *yes* and *no* (Pilot Study 3: Neutral condition)

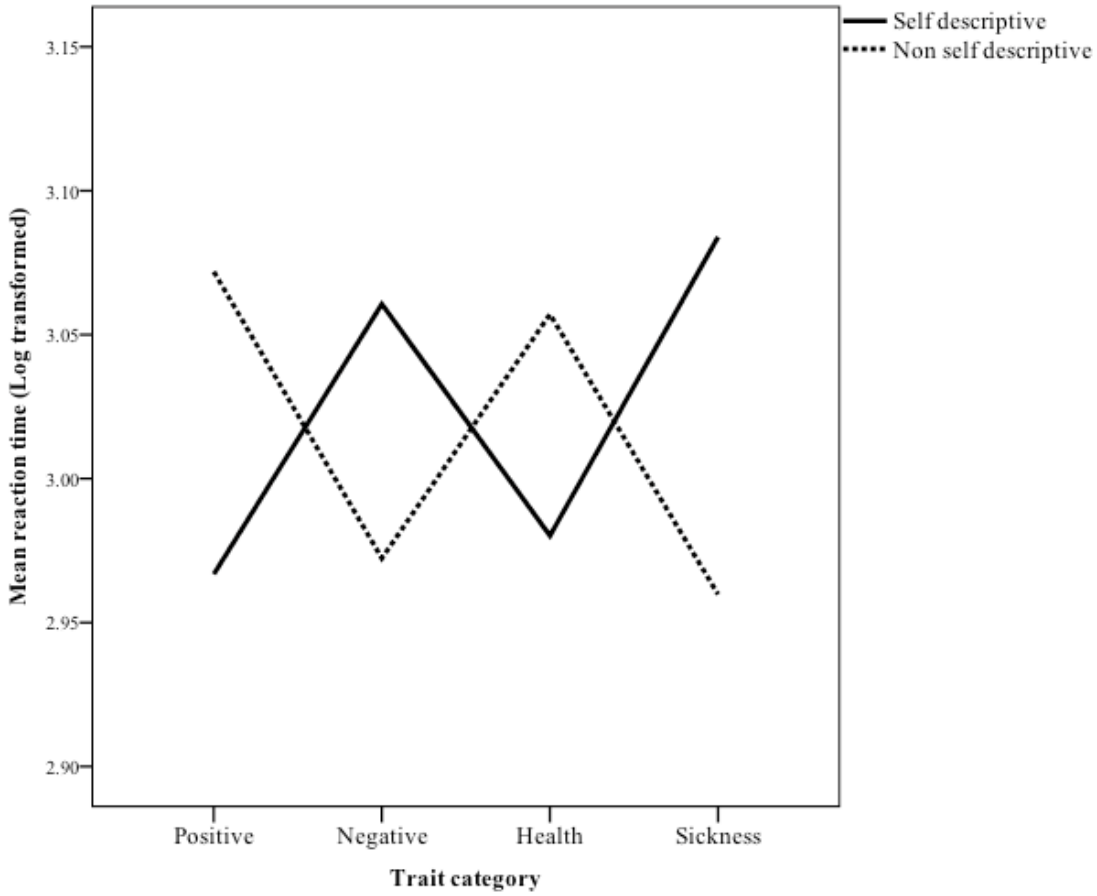


Figure 4. Mean reaction time to positive and negative health conditions by priming method (Experiment 1)

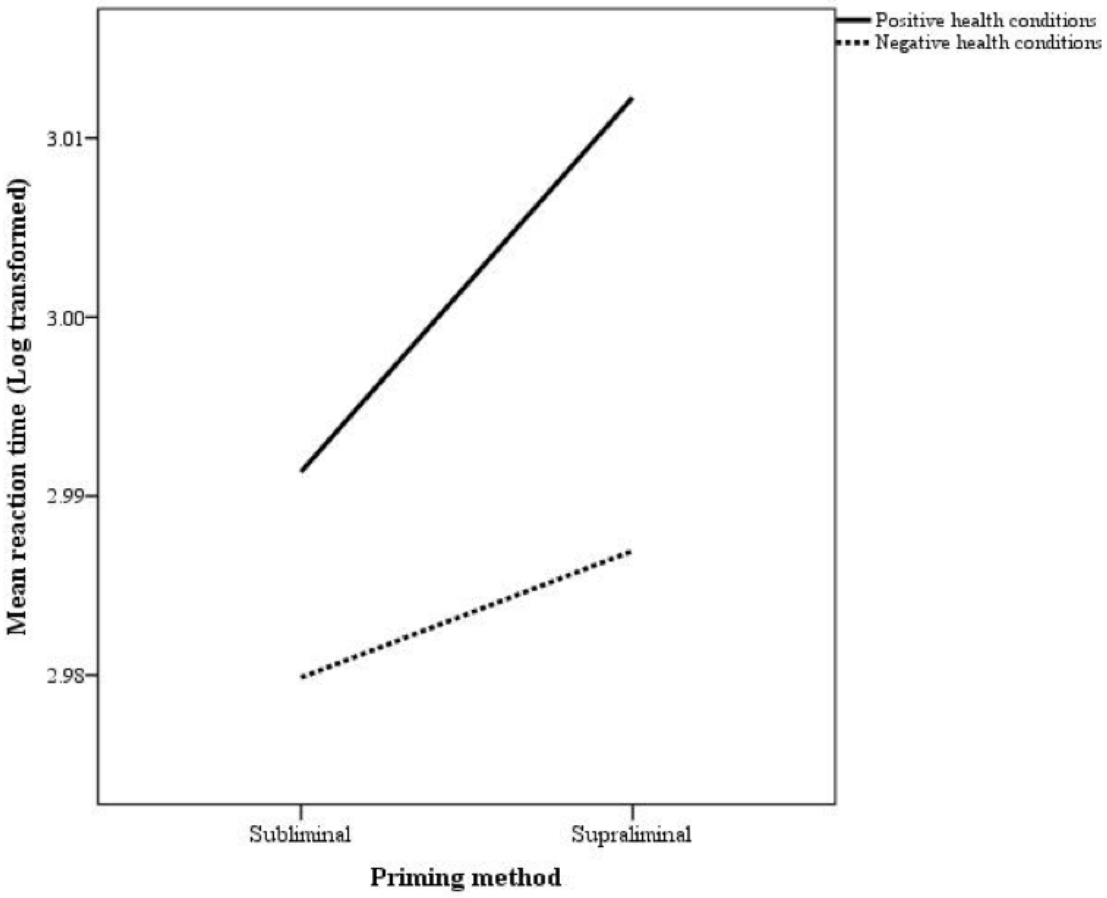


Figure 5. Mean reaction time for susceptibility to positive and negative health conditions split by *yes* and *no* responses (Experiment 1)

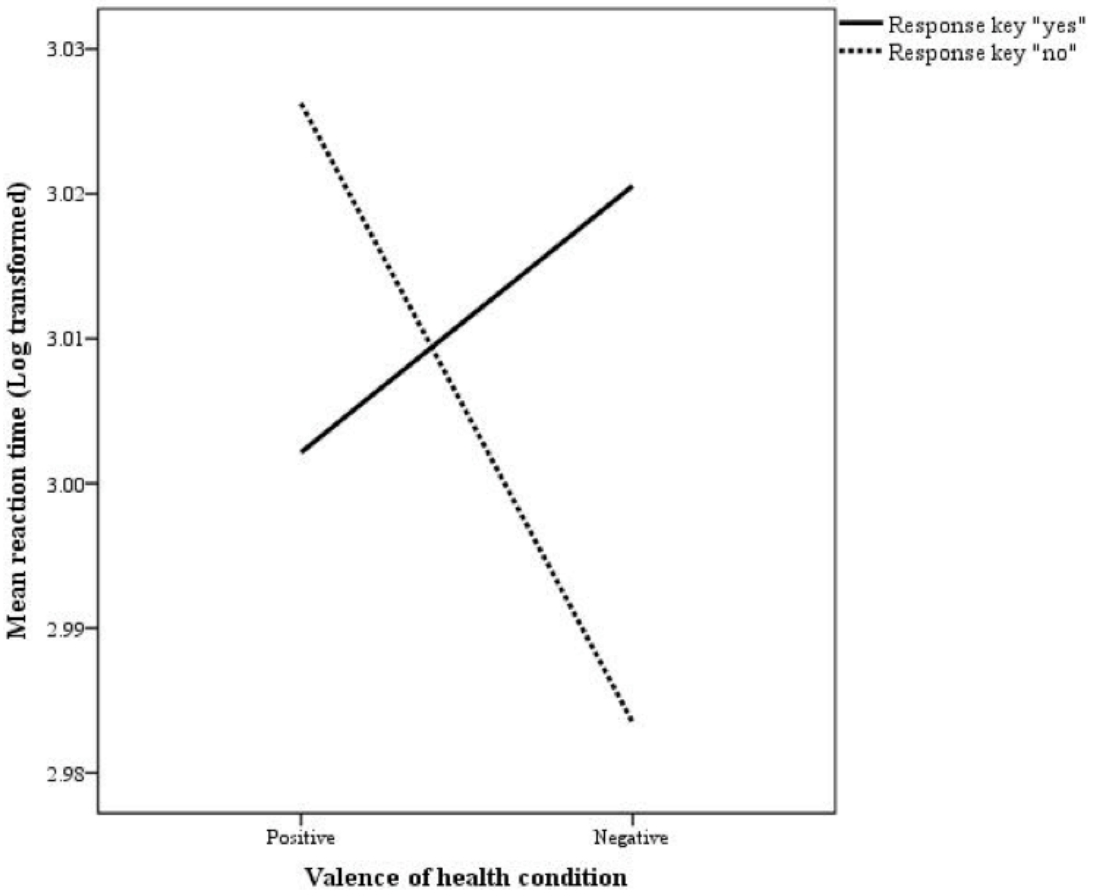


Figure 6. Mean perceived susceptibility (response key *yes*) to Cluster 1 health conditions (Experiment 2)

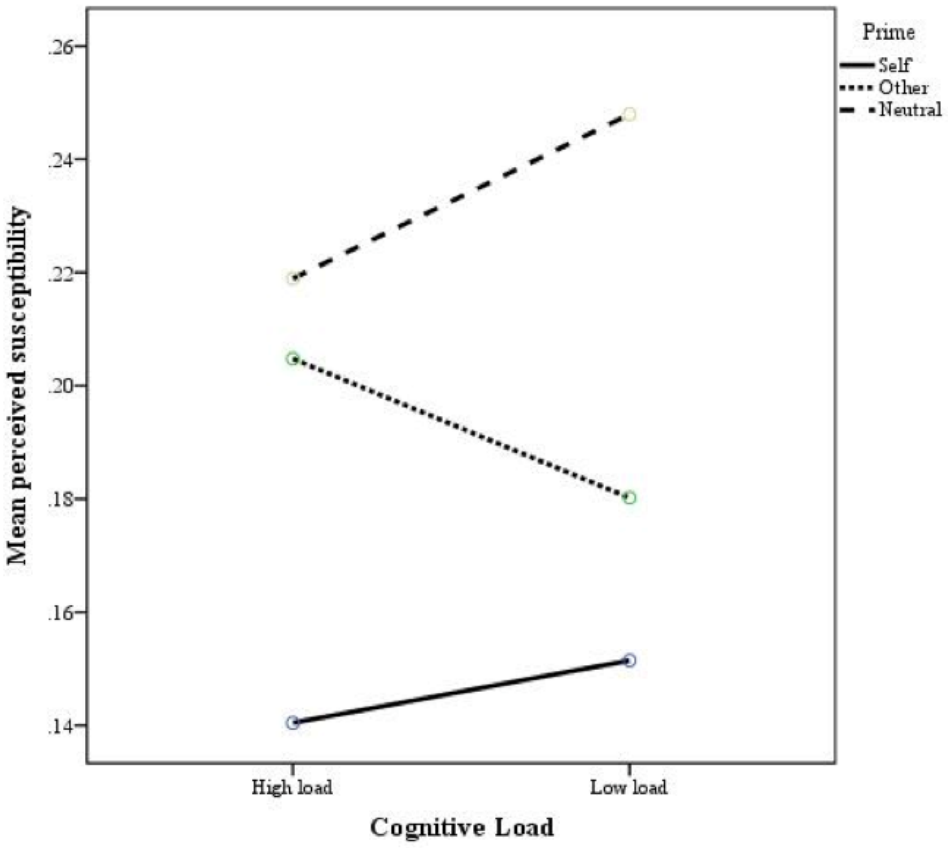


Figure 7. Mean perceived severity (response key *yes*) of Cluster 1 health conditions (Experiment 2)

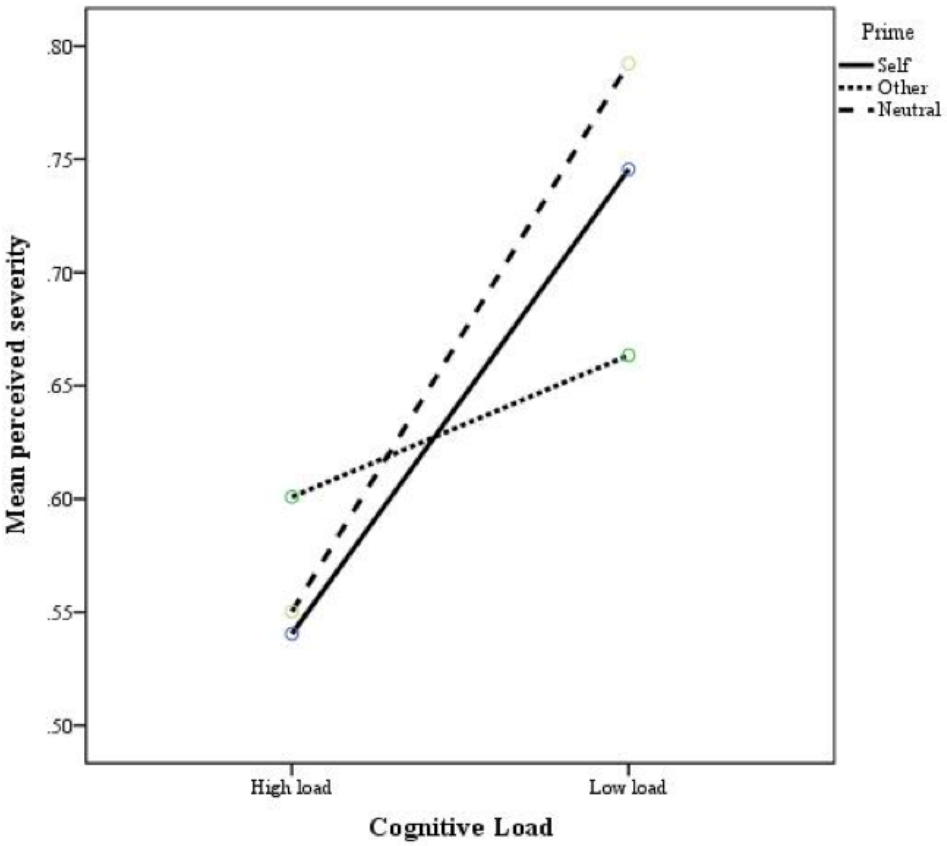


Figure 8. Mean perceived susceptibility (response key *yes*) to Cluster 2 health conditions (Experiment 2)

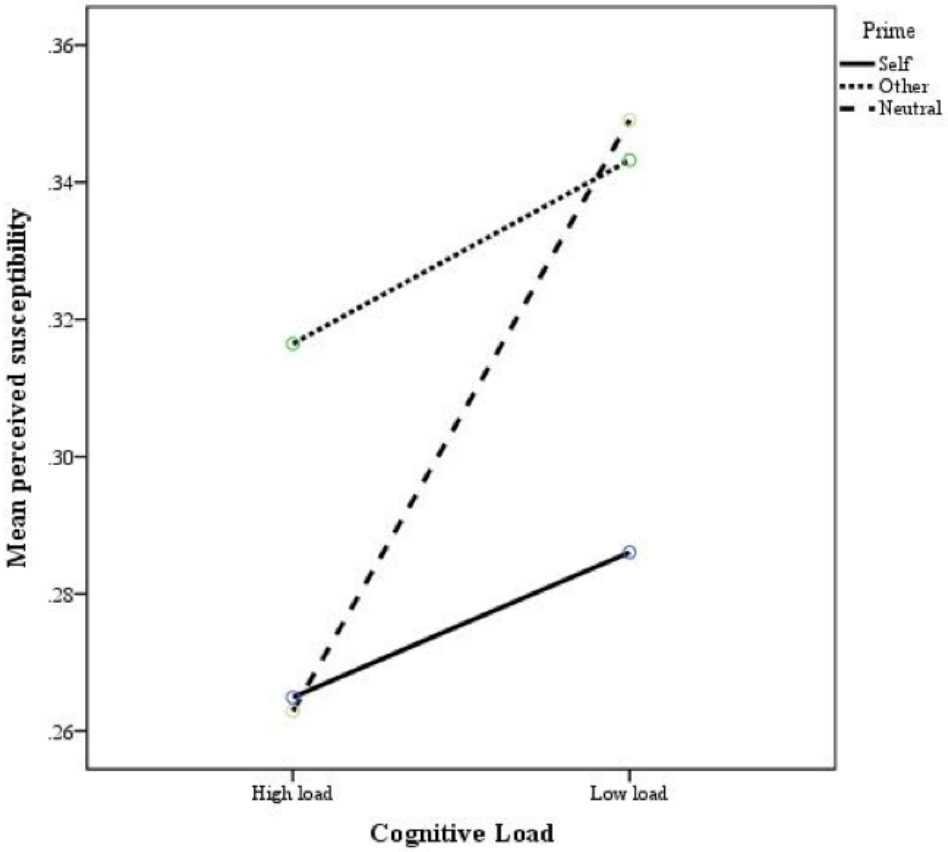


Figure 9. Mean perceived severity (response key *yes*) of Cluster 2 health conditions (Experiment 2)

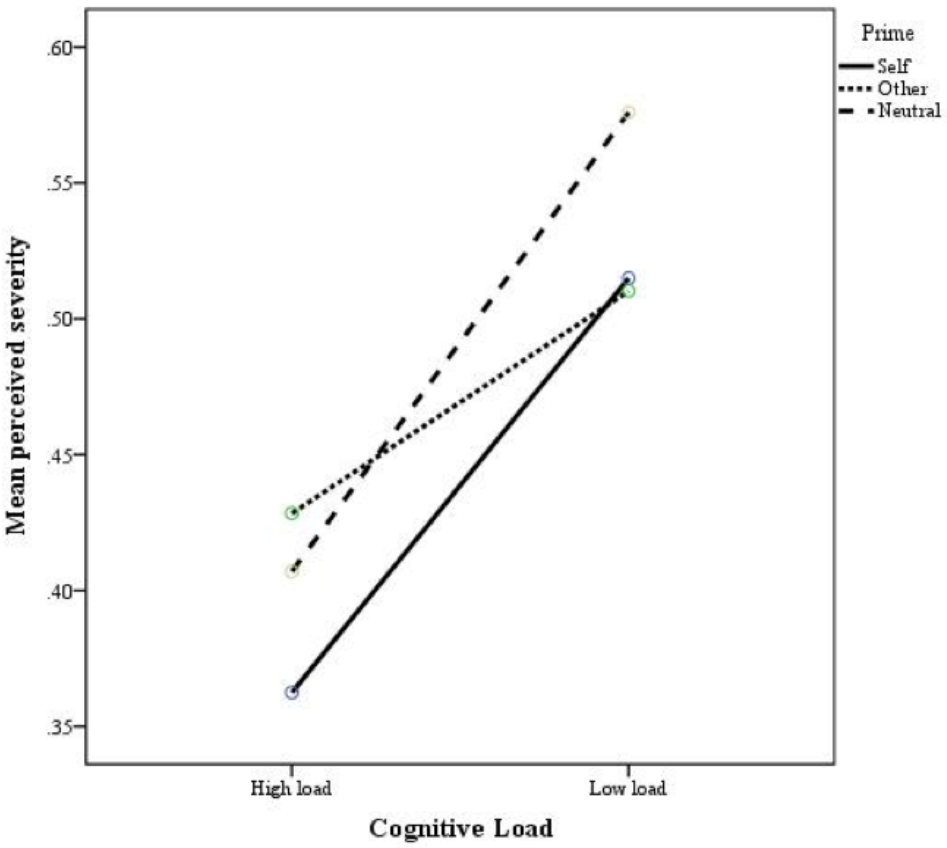


Figure 10. Mean perceived susceptibility (response key *yes*) to Cluster 3 health conditions (Experiment 2)

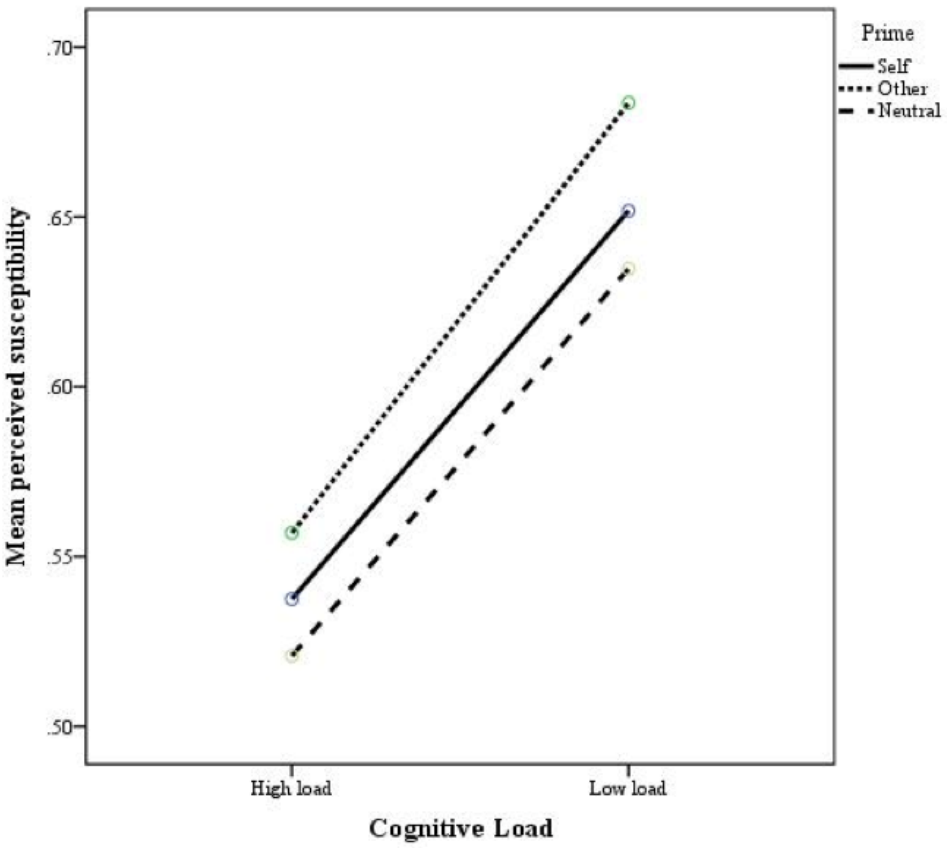


Figure 11. Mean perceived severity (response key *yes*) of Cluster 3 health conditions (Experiment 2)

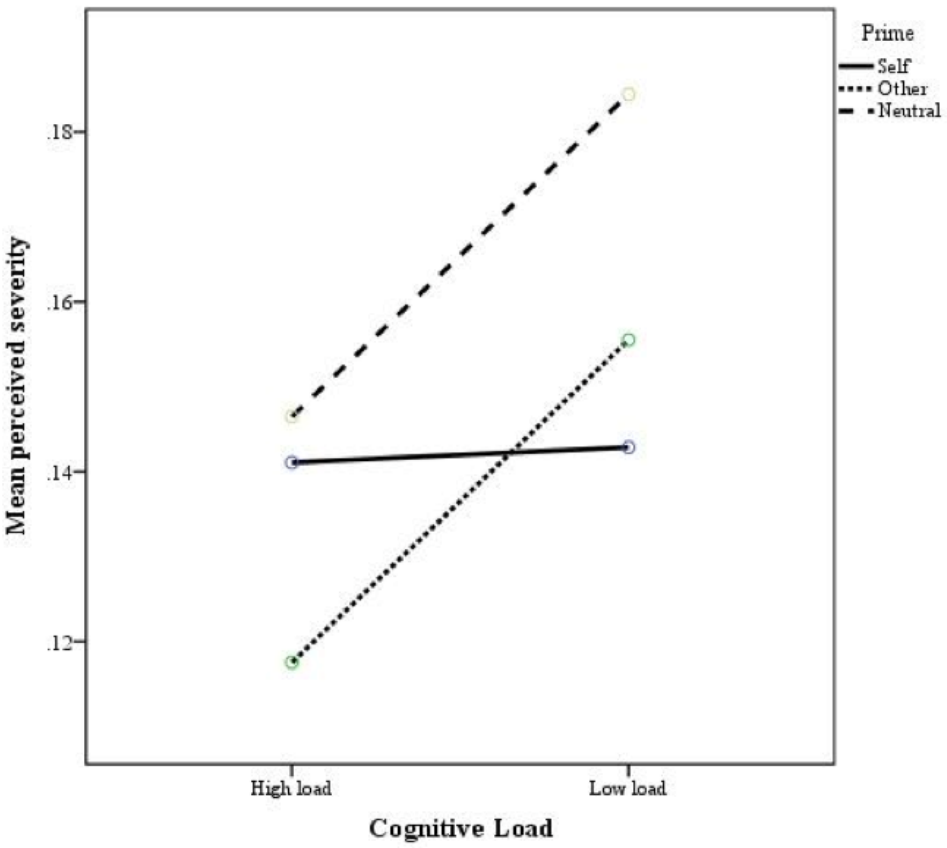


Figure 12. Mean reaction time for perceived susceptibility to health condition clusters (combined for *yes* and *no* responses) (Experiment 2)

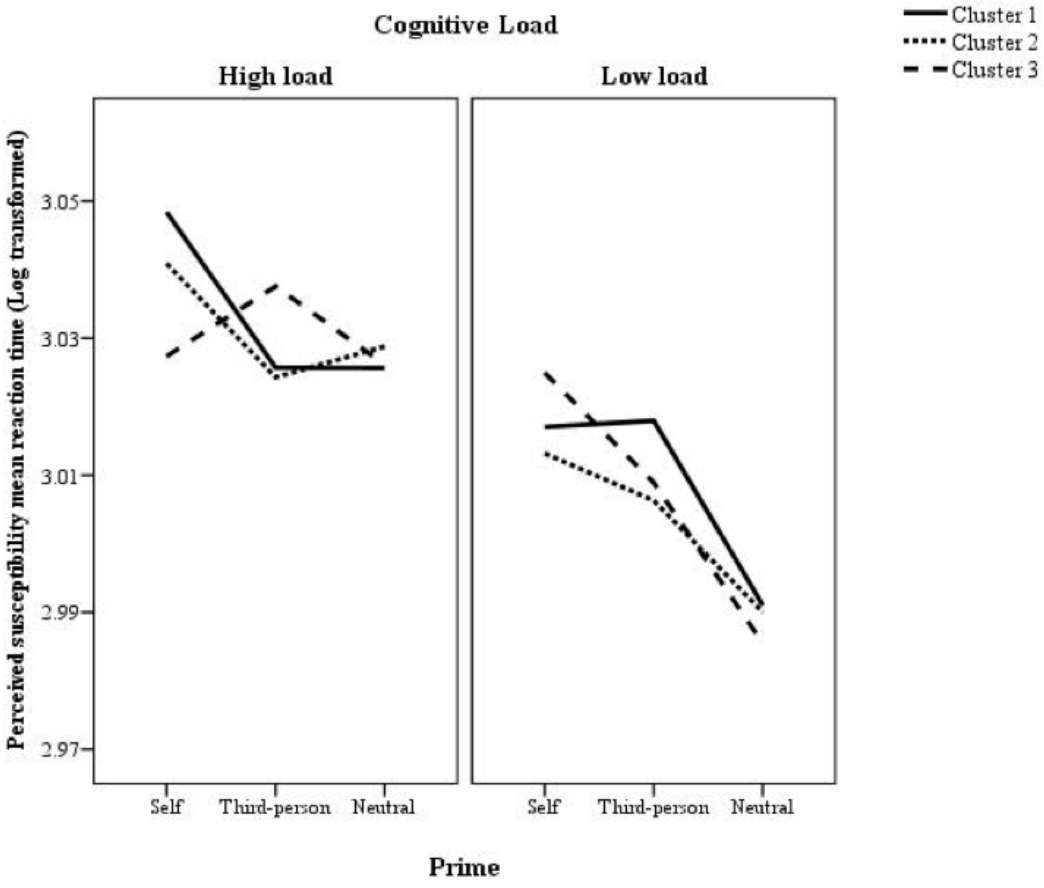


Figure 13. Mean reaction time for perceived severity of health condition clusters (combined for *yes* and *no* responses) (Experiment 2)

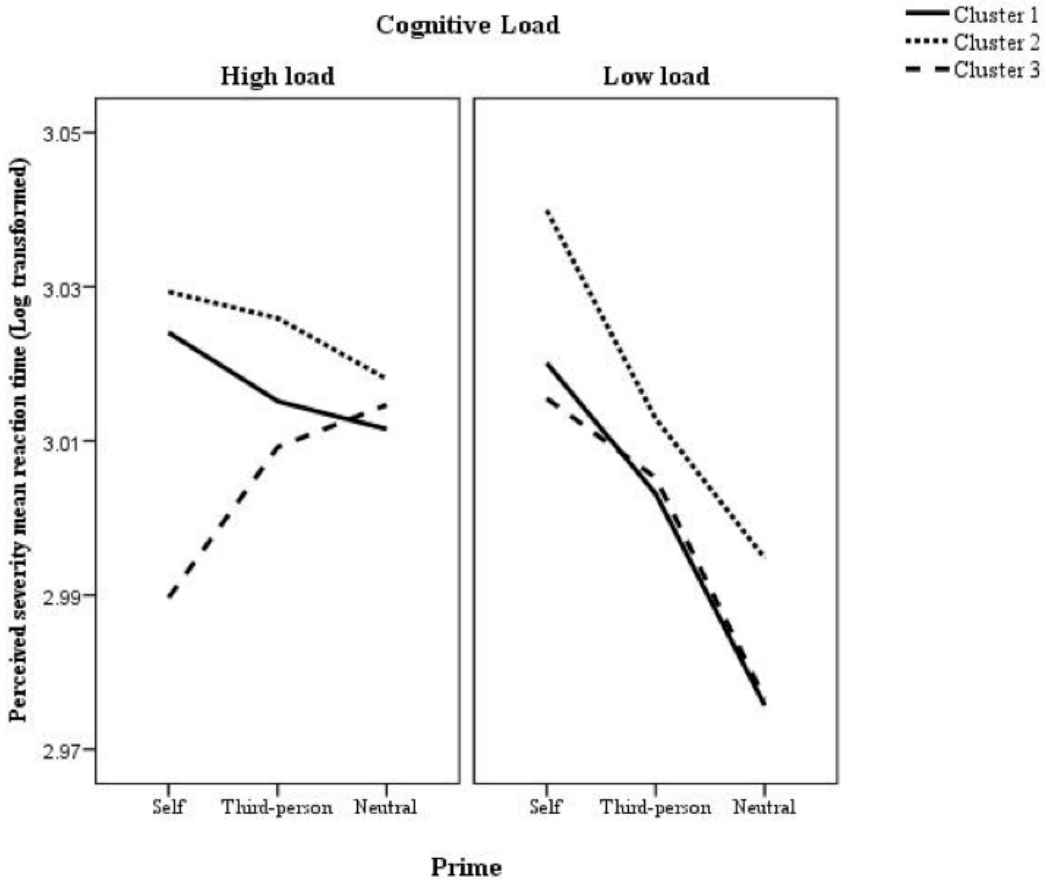


Figure 14. Response key *yes* for perceived susceptibility and severity by health condition clusters (Experiment 2)

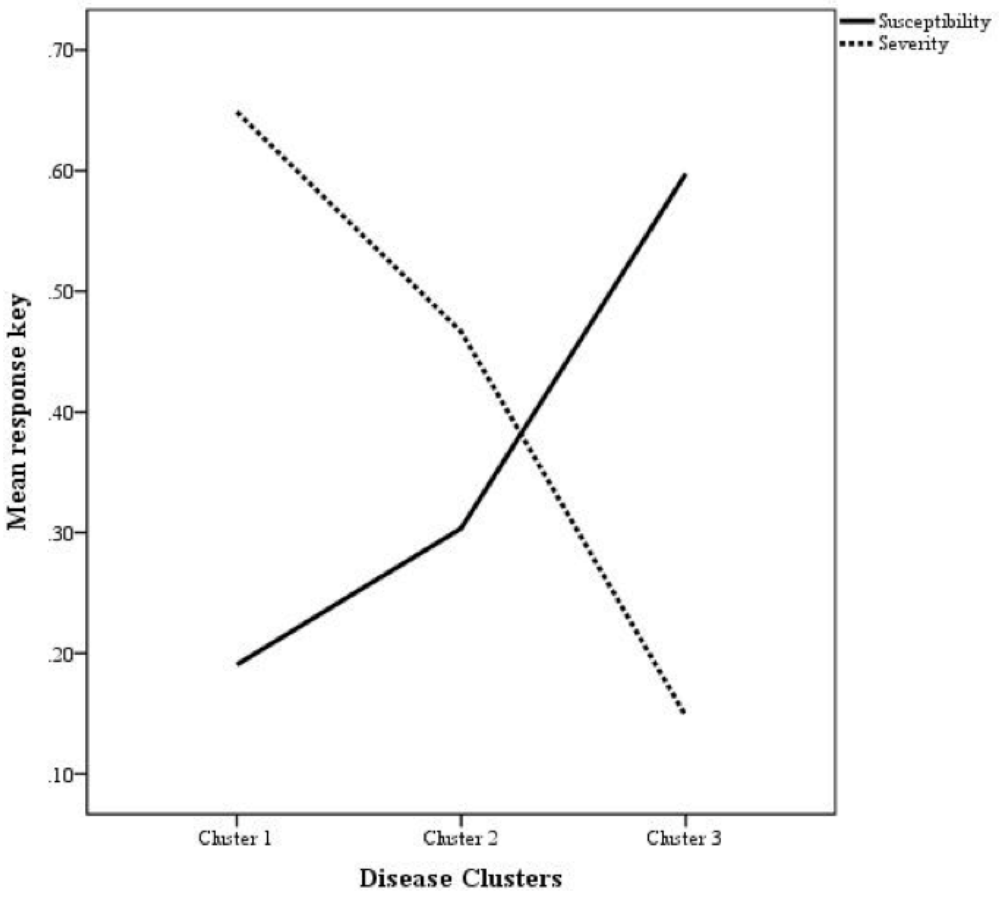


Figure 15. Mean reaction time for susceptibility to and severity of health condition clusters split by response key *yes* and *no* (Experiment 2)

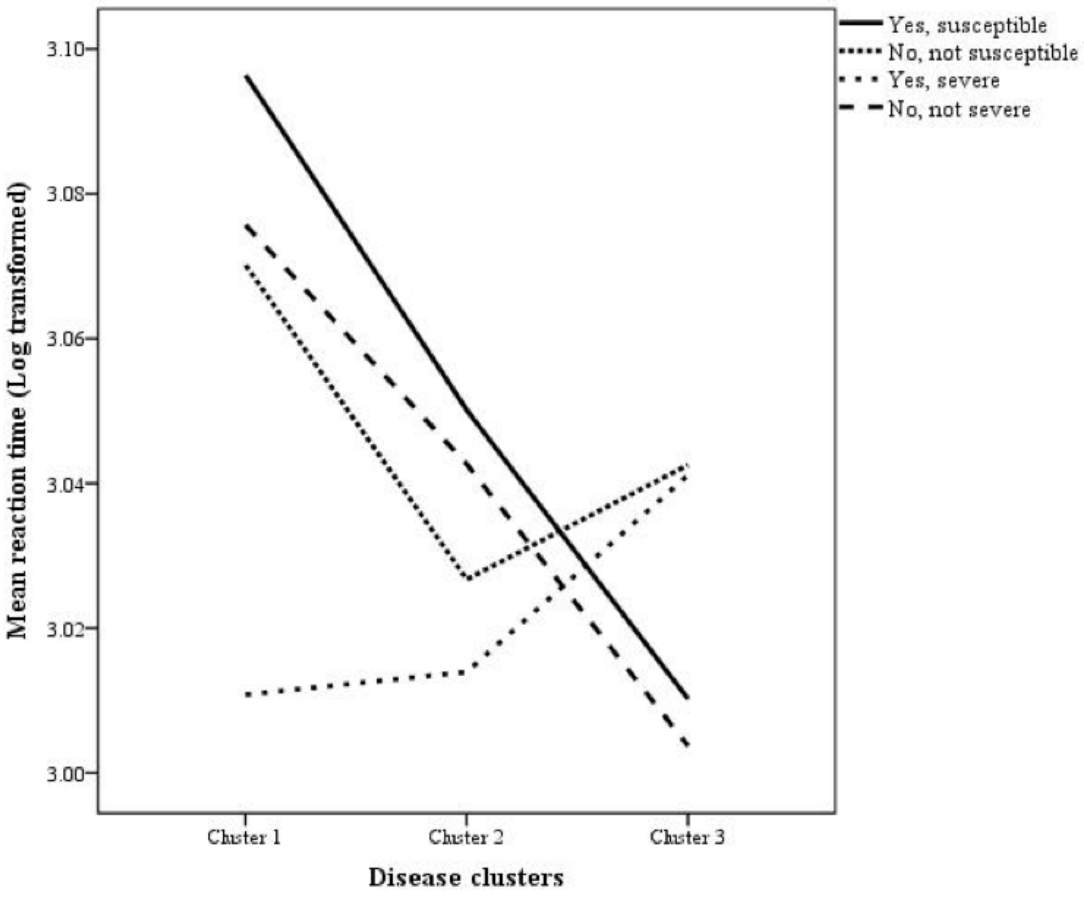


Figure 16. Mean reaction time for susceptibility to and severity of Cluster 1 health conditions split by *yes* and *no* responses (Experiment 2)

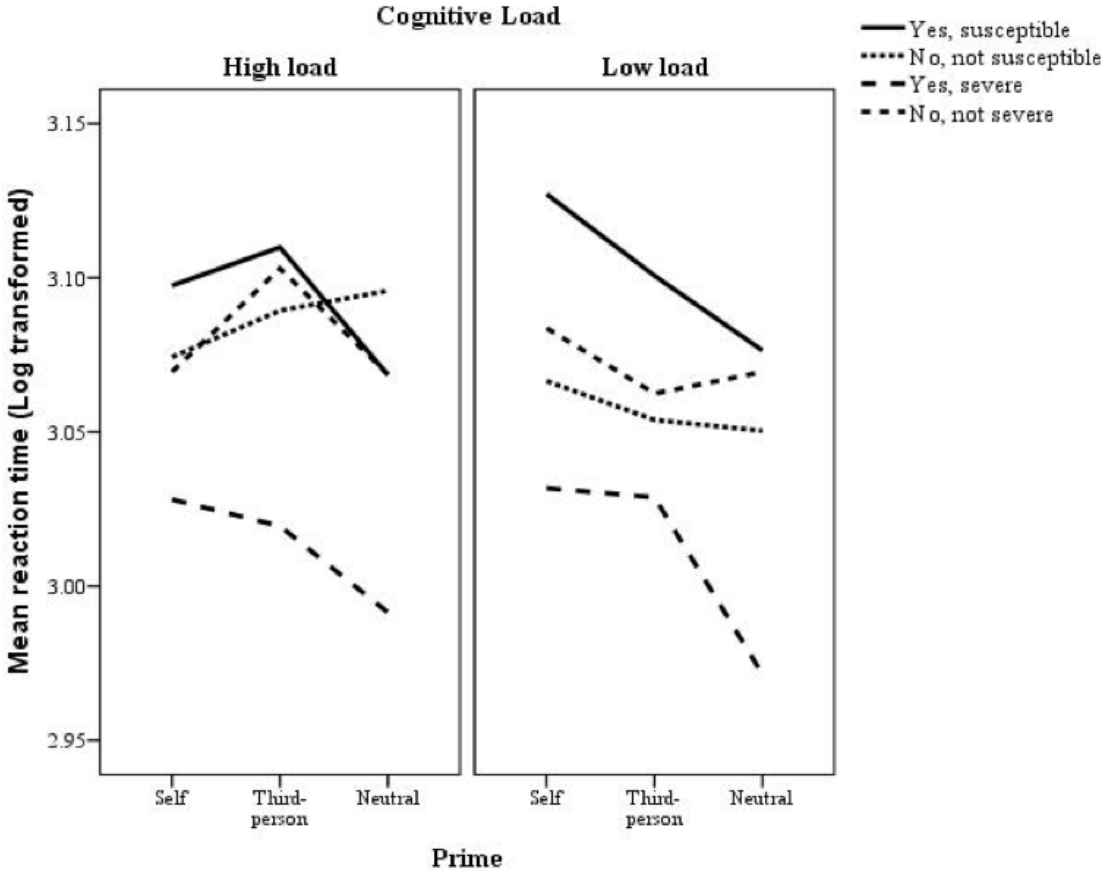


Figure 17. Mean reaction time for susceptibility to and severity of Cluster 2 health conditions split by *yes* and *no* responses (Experiment 2)

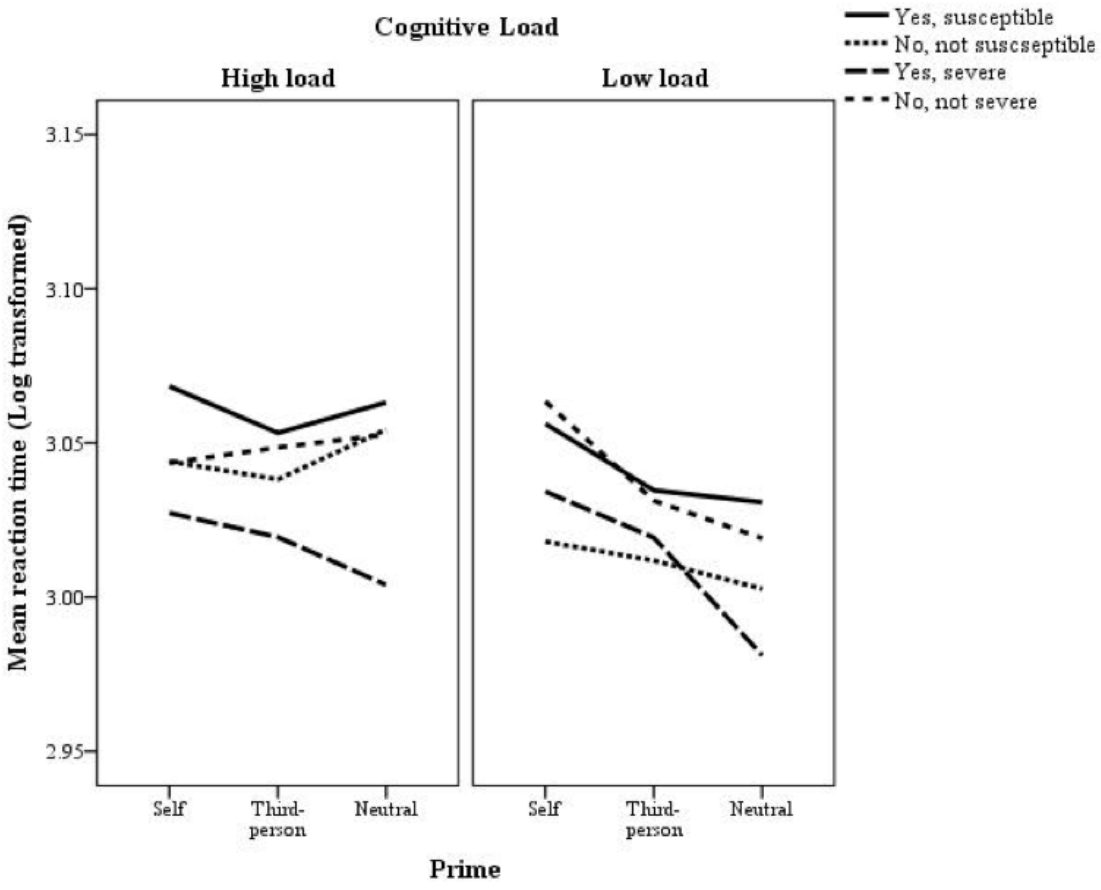


Figure 18. Mean reaction time for susceptibility to and severity of Cluster 3 health conditions split by *yes* and *no* responses (Experiment 2)

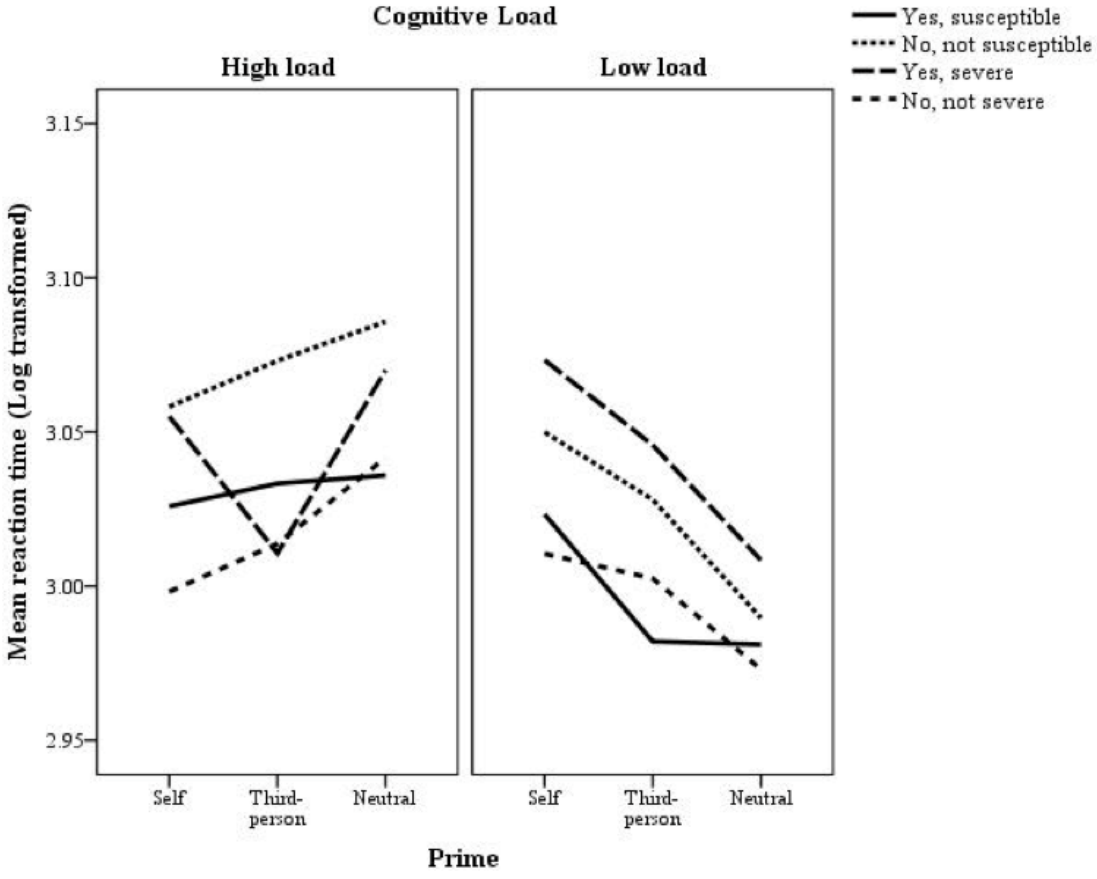
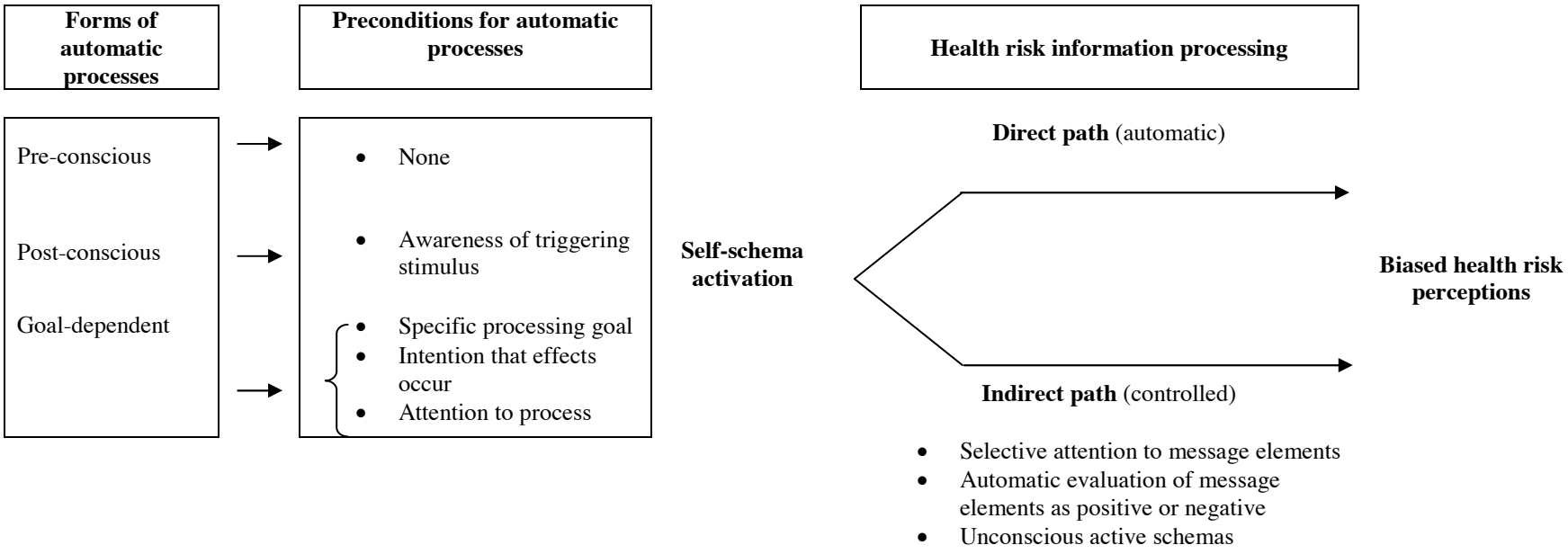


Figure 19. Routes of automatic self-schema activation and its influence on health risk information processing and risk perceptions



APPENDIX 2:

MANIPULATIONS AND MEASURES

Independent Variables

Self-schema activation (manipulated).

Subliminal primes.
Self primes. I, me, my, mine, myself, self.
Other primes. He/she, him/her, his/her, his/hers, himself/herself, other.
Neutral primes. It, its, itself.

Supraliminal primes.[91]
(I/he/it) go(es) to Chicago [above].
Anticipation fills (me/him/it) all-over [between].
(I/he/it) can see skyscrapers [after].
(I/he/it) want(s) to explore [against].
Attractions always escape (me/him/it) [within].
(my/his/its) voice is everywhere [despite].
(I/he/it) see(s) the sights [during].
(I/he/it) always window shop(s) [since].
(my/his/its) television is black [front].
(I/he/it) almost always linger(s) [addition].
(my/his/its) time is over [front].
(I/he/it) must leave now [except].
Boston belongs to (me/him/it) [despite].
(I/he/it) will return back [into].
(I/he/it) am/is almost there [beside].
This photograph is (mine/his/its) [like].
(my/his/its) ride is here [into].
(I/he/it) is taking lessons [underneath].
(I/he/it) typed a letter [addition].
(I/he/it) opened a bottle [toward].
(I/he/it) stared for hours [off].
(I/he/it) got a grade [without].
The pen is (mine/his/its) [above].
(I/he/it) moved a chair [beyond].
(I/he/it) ran two miles within.

Psychological reactance (measured).[92]

[91] Participants were instructed to form grammatically correct sentences using only four of the five words presented. Words in brackets represent those that do not belong to the sentence. Words in parentheses are the primes.

[92] $\alpha = .80$.

Response scale: 1= *strongly disagree*, 2 = *disagree*, 3 = *neither agree nor disagree*, 4 = *agree*, and 5 = *strongly agree*.

1. Regulations trigger a sense of resistance in me.
2. I find contradicting others stimulating.
3. When something is prohibited, I usually think "that's exactly what I am going to do."
4. The thought of being dependent on others aggravates me.
5. I consider advice from others to be an intrusion.
6. I become frustrated when I am unable to make free and independent decisions.
7. It irritates me when someone points out things which are obvious to me.
8. I become angry when my freedom of choice is restricted.
9. Advice and recommendations induce me to do just the opposite.
10. I am content only when I am acting of my own free will.
11. I resist the attempts of others to influence me.
12. It makes me angry when another person is held up as a model for me to follow.
13. When someone forces me to do something, I feel like doing the opposite.
14. It disappoints me to see others submitting to society's standards and rules.

Control Variables

Depression inventory.[93]

On this questionnaire are groups of statements. Please read each group of statements carefully. Then pick out one statement in each group that best describes the way you have been feeling the PAST WEEK, INCLUDING TODAY! Circle the number beside the statement you picked. If several statements in the group seem to apply equally well, circle each one. Be sure to read all the statements in each group before making your choice.

A: Mood
0. I do not feel sad.
1. I feel sad.
2. I am sad all the time and I can't snap out of it.
3. I am so sad or unhappy that I can't stand it.

B: Pessimism
0. I am not particularly discouraged about the future.
1. I feel discouraged about the future.
2. I feel I have nothing to look forward to.
3. I feel that the future is hopeless and that things cannot improve.

C: Sense of failure
0. I do not feel like a failure.
1. I feel I have failed more than the average person.
2. As I look back on my life all I can see is a lot of failures.
3. I feel I am a complete failure as a person.

[93] Spilt-half reliability= .86.

D: Lack of satisfaction
0. I get as much satisfaction out of thins as I used to.
1. I don't enjoy things the way I used to.
2. I don't get real satisfaction out of anything any more.
3. I am dissatisfied or bored with everything.

E: Guilty feeling
0. I don't feel particularly guilty.
1. I feel guilty a good part of the time.
2. I feel quite guilty most of the time.
3. I feel guilty all the time.

F: Sense of punishment
0. I don't feel I am being punished.
1. I feel I may be punished.
2. I expect to be punished.
3. I feel I am being punished.

G: Self hate
0. I don't feel disappointed in myself.
1. I am disappointed in myself.
2. I am disgusted with myself.
3. I hate myself.

H: Self accusations
0. I don't feel I am worse than anybody else.
1. I am critical of myself for my weaknesses or mistakes.
2. I blame myself all the time for my faults.
3. I blame myself for everything bad that happens.

I: Self-punitive wishes
0. I don't have any thoughts of killing myself.
1. I have thoughts of killing myself, but I would not carry them out.
2. I would like to kill myself.
3. I would kill myself if I had the chance.

J: Crying spells
0. I don't cry any more than usual.
1. I cry more now than I used to.
2. I cry all the time now.
3. I used to be able to cry but now I can't cry even though I want to.

K: Irritability
0. I am no more irritated now than I ever am.
1. I get annoyed or irritated more easily than I used to.
2. I feel irritated all the time now.

3. I don't get irritated at all by the things that used to irritate me.

L: Social withdrawal
0. I have not lost interest in other people.
1. I am less interested in other people than I used to be.
2. I have lost most of my interest in other people.
3. I have lost all my interest in other people.

M: Indecisiveness
0. I make decisions about as well as I ever could.
1. I put off making decisions more than I used to.
2. I have greater difficulty in making decisions than before.
3. I can't make any decisions at all anymore.

N: Body image
0. I don't feel I look any worse than I used to.
1. I am worried that I am looking old or unattractive.
2. I feel that there are permanent changes in my appearance and they make me look unattractive.
3. I believe that I look ugly.

O: Work inhibition
0. I can work about as well as before.
1. It takes extra effort to get started at doing something.
2. I have to push myself very hard to do anything.
3. I can't do any work at all.

P: Sleep disturbance
0. I can sleep as well as usual.
1. I don't sleep as well as I used to.
2. I wake up 1-2 hours earlier than usual and find it hard to get back to sleep.
3. I wake up several hours earlier than I used to and cannot get back to sleep.

Q: Fatigability
0. I don't get more tired than usual.
1. I get tired more easily than I used to.
2. I get tired from doing almost anything.
3. I get too tired to do anything.

R: Loss of appetite
0. My appetite is no worse than usual.
1. My appetite is not as good as it used to be.
2. My appetite is much worse now.
3. I have no appetite at all anymore.

S: Weight loss*

I am purposely trying to lose weight by eating less. Yes/ No.
0. I haven't lost much weight, if any, lately.
1. I have lost more than 5 pounds.
2. I have lost more than 10 pounds.
3. I have lost more than 15 pounds.

T: Somatic preoccupation
0. I am no more worried about my health than usual.
1. I am worried about physical problems such as aches and pains; *or* upset stomach; *or* constipation.
2. I am very worried about physical problems and it's hard to think of much else.
3. I am so worried about my physical problems, that I cannot think about anything else.

U: Loss of libido
0. I have not noticed any recent change in my interest in sex.
1. I am less interested in sex than I used to be.
2. I am much less interested in sex now.
3. I have lost interest in sex completely.

* Reverse-coded item.

Life orientation test.[94]

Response scale: 0 = *strongly disagree*, 1= *disagree*, 2 = *neutral*, 3 = *agree*, and 4 = *strongly agree*.

1. In uncertain times, I usually expect the best.
2. If something can go wrong for me, it will.*
3. I'm always optimistic about my future.
4. I hardly ever expect things to go my way.*
5. I rarely count on good things happening to me.*
6. Overall, I expect more good things to happen to me than bad.

* Reverse-coded items.

Health locus of control.[95]

Response scale: 1 = *strongly disagree*, 2 = *disagree*, 3 = *slightly disagree*, 4 = *slightly agree*, 5 = *agree*, 6 = *strongly agree*.

1. If I take care of myself, I can avoid illness (I)
2. Whenever I get sick it is because something I've done or not done (I)
3. Good health is largely a matter of good fortune (E)

---

[94] $\alpha = .78$. Test-retest reliability: $\alpha = .68$ (4 months), $\alpha = .60$ (12 months), $\alpha = .56$ (24 months), and $\alpha = .79$ (28 months).

[95] $\alpha = .72$.

4. No matter what I do, if I am going to get sick I will get sick (E)
5. Most people do not realize the extent to which their illnesses are controlled by accidental happenings (E)
6. I can only do what my doctor tells me to do (E)
7. There are so many strange diseases around that you can never know how or when you might pick one up (E)
8. When I feel ill, I know it is because I have not been getting the proper exercise or eating right (I)
9. People who never get sick are just plain lucky (E)
10. People's ill health results from their own carelessness (I)
11. I am directly responsible for my health (I)

I = internally worded, E = externally worded. The scale is scored in the external direction with each item scored from 1 = strongly disagree to 6 = strongly agree for the externally worded items and reverse scored for the internally worded items.

Self consciousness.[96]

Response scale: 0 = *extremely uncharacteristic* to 4 = *extremely characteristic*.

1. I'm always trying to figure myself out.
2. I'm concerned about my style of doing things.
3. Generally, I'm not very aware of myself.*
4. It takes me time to overcome my shyness in new situations.
5. I reflect about myself a lot.
6. I'm concerned about the way I present myself.
7. I'm often the subject of my own fantasies.
8. I have trouble working when someone is watching me.
9. I never scrutinize myself.*
10. I get embarrassed very easily.
11. I'm self-conscious about the way I look.
12. I don't find it hard to talk to strangers.*
13. I'm generally attentive to my inner feelings.
14. I usually worry about making a good impression.
15. I'm constantly examining my motives.
16. I feel anxious when I speak in front of a group.
17. One of the last things I do before I leave my house is look in the mirror.
18. I sometimes have the feeling that I'm off somewhere watching myself.
19. I'm concerned about what other people think of me.
20. I'm alert to changes in my mood.
21. I'm usually aware of my appearance.
22. I'm aware of the way my mind works when I work through a problem.
23. Large groups make me nervous.

[96] Test-retest correlations for the subscales were: public self-consciousness = .84; private self-consciousness = .79; social anxiety = .73; and total score = .80.

* Items are reverse scored.

Self-efficacy.

Response scale: 1 = *strongly agree*, 2 = *agree*, 3 = *neutral*, 4 = *disagree*, 5 = *strongly disagree*.

1. When I make plans, I am certain I can make them work.*
2. One of my problems is that I cannot get down to work when I should.
3. If I can't do a job the first time, I keep trying until I can.*
4. When I set important goals for myself, I rarely achieve them.
5. I give up on things before completing them.
6. I avoid facing difficulties.
7. If something looks too complicated, I will not even bother to try it.
8. When I have something unpleasant to do, I stick to it until I finish it.*
9. When I decide to do something, I go right to work on it.*
10. When trying to learn something new, I soon give up if I am not initially successful.
11. When unexpected problems occur, I don't handle them well.
12. I avoid trying to learn new things when they look too difficult for me.
13. Failure just makes me try harder.*
14. I feel insecure about my ability to do things.
15. I am a self-reliant person.*
16. I give up easily.
17. I don't seem capable of dealing with most problems that come up in life.

* Items are reverse scored.

Self-esteem scale.[97]

Response scale: 1 = *strongly agree*, 2 = *agree*, 3 = *disagree*, 4 = *strongly disagree*.

1. On the whole, I am satisfied with myself.*
2. At times I think I am no good at all.
3. I feel that I have a number of good qualities.*
4. I am able to do things as well as most other people.*
5. I feel I do not have much to be proud of.
6. I certainly feel useless at times.
7. I fell that I'm a person of worth.*
8. I wish I could have more respect for myself.
9. All in all, I am inclined to think that I am a failure.
10. I take a positive attitude toward myself.*

* Items are reverse scored.

Dependent Measures

[97] α ranges from .74 to .84.

Affect/ arousal.

Please click on the number that best describes how you feel on the following items.

Bad	-5	-4	-3	-2	-1	0	1	2	3	4	5	Good
Disappointed	-5	-4	-3	-2	-1	0	1	2	3	4	5	Satisfied
Sad	-5	-4	-3	-2	-1	0	1	2	3	4	5	Happy
Displeased	-5	-4	-3	-2	-1	0	1	2	3	4	5	Pleased
Calm	-5	-4	-3	-2	-1	0	1	2	3	4	5	Excited
Energetic*	-5	-4	-3	-2	-1	0	1	2	3	4	5	Tired
Down	-5	-4	-3	-2	-1	0	1	2	3	4	5	Elated
Sedate	-5	-4	-3	-2	-1	0	1	2	3	4	5	Aroused

* Item reverse coded.

APPENDIX 3:

PILOT WORK

Pilot Study 1: Selection of health conditions

List of health conditions presented to rating groups for initial screening. Clusters 1, 2, and 3 denote health conditions familiar to 90% or more of student population based on Pilot Studies 1 and 2 and the cluster to which the disease belongs. Positively valenced health conditions used in Experiment 1 appear in parentheses.

Cluster 1	Cluster 2	Cluster 3
Bone cancer (Healthy bones)	Alcoholism	Binge drinking
Brain damage (Healthy brain)	Allergies	Broken bone
Brain injury	Appendicitis	Constipation
Brain tumors	Arthritis	Diarrhea
Gum disease (Healthy gums)	Asthma	Flu
Heart abnormalities	Bladder infection	Stress
Hepatitis B	Cardiac arrest	Sunburn
HIV/ AIDS	Chlamydia	
Human papillomavirus	Depression	
Infertility (Fertility)	Diabetes	
Leukemia	Genital warts	
Malaria	Hearing loss (Healthy hearing)	
Organ transplant	Heart attack	
Pancreatic cancer	Heart failure (Healthy heart)	
Personality disorder (Healthy personality)	Herpes	
Schizophrenia	Kidney stones (Healthy kidneys)	
Seizure	Lung cancer (Healthy lungs)	
	Melanoma	
	Migraine	
	Nervous breakdown	
	Obesity (Healthy weight)	
	Pneumonia	
	Skin cancer	
	Stomach ulcer	
	Stroke	
	Suicide	

Familiarity:[98] Have you encountered the name of this health condition before this study?

- Yes
- No[99]

Population prevalence: Estimate the percentage of students at the university to whom this event could occur.
----- % (Write a number between 0 and 100. For example: 50%).

Stereotype salience: How easy is it to imagine the kind of person who typically suffers from this event?
Very hard to imagine 1 2 3 4 5 6 7 Very easy to imagine

Perceived risk controllability: Can a person control whether this health condition happens to them?
Very hard to control 1 2 3 4 5 6 7 Very easy to control

Exempt beliefs: If an individual had not yet experienced this health condition, how likely is it to happen for them?
Impossible 0 1 2 3 4 5 6 7 Extremely likely

Personal experience: Do you know of anyone to whom this health condition has happened?[100]
0 Has not happened to anyone I know before
1 Has happened to few acquaintances
2 Has happened to many acquaintances
3 Has happened to few friends or close relatives
4 Has happened to many friends or close relatives
5 Has happened to me once
6 Has happened to me more than once

Personal susceptibility: How likely is it for you to encounter this health condition?
Impossible 0 1 2 3 4 5 6 7 Extremely likely

Personal severity: If you encounter this health condition, how severe or serious will it be?
Not severe at all 1 2 3 4 5 6 7 Extremely severe

---

[98] Words in bold are variable names and did not appear on the questionnaire.

[99] If a participant were unfamiliar with a specific health condition, she would skip over the questions pertaining to disease (e.g., prevalence, personal experience).

[100] Personal experience was a multiple-choice question, where participants could choose more than one option. The response that reflected the strongest personal experience was selected for data analysis purposes. For example, if a participant circled 2 (has happened to many acquaintances) and 5 (has happened to me once), a score of 5 was selected for personal experience.

Pilot Study 2: Pretesting health risk controllability manipulation
Stimulus material: Balamuthia mandrillaris ameba infection

Version 1: Controllable (191 words)

Balamuthia infection
It can be stopped!

Balamuthia is a free-living ameba. Anyone can be exposed to Balamuthia, people who are healthy or those with weakened immune systems. It causes a serious infection of the brain and spinal cord called granulomatous amebic encephalitis (GAE) that is usually fatal with a death rate of 95%.

Why is it stoppable?

Much is known about how a person becomes infected. It infects the body through skin wounds and cuts or when dust or water containing Balamuthia is breathed in or forced through the nose or mouth.

Preventing infection includes avoiding contact with stagnant waters, refraining from water-related activities in bodies of warm fresh water, hot springs, and thermally-polluted water such as water around power plants, and avoiding digging in or stirring up the sediment while taking part in water-related activities in shallow, warm fresh water areas.

It takes only days to develop the first symptoms of Balamuthia after exposure. Diagnosis of Balamuthia is easy, and it may be easily distinguished from other neurological and non-infectious diseases. Tests to identify Balamuthia are widely available.

Treatment options are available and almost all patients have survived with treatment.

Version 2: Uncontrollable (193 words)

Balamuthia infection
It cannot be stopped!

Balamuthia is a free-living ameba. Anyone can be exposed to Balamuthia, people who are healthy or those with weakened immune systems. It causes a serious infection of the brain and spinal cord called granulomatous amebic encephalitis (GAE) that is usually fatal with a death rate of 95%.

Why is it unstoppable?

Balamuthia infection can occur at any time of year. Little is known about how a person becomes infected and what specific factors make people more susceptible to infection and disease from Balamuthia.

There are no known ways to prevent infection with Balamuthia since it occurs worldwide in soil, dust, and fresh water. Everyday activities like gardening, playing with dirt, breathing in soil carried by wind, and water-related activities increase the risk for infection.

It takes weeks to months, even 2 or more years, to develop the first symptoms of Balamuthia after exposure. Diagnosis of Balamuthia is difficult, as it may be easily confused with other neurological and non-infectious diseases. Tests to identify Balamuthia are not widely available.

The best treatment options are still unknown and only a handful of patients have survived with treatment.

Version 3: Neutral (192 words)

Balamuthia infection

Balamuthia is a free-living ameba. Anyone can be exposed to Balamuthia -- people who are healthy or those with weakened immune systems. It causes a serious infection of the brain and spinal cord called granulomatous amebic encephalitis (GAE) that is usually fatal with a death rate of 95%.

Early symptoms might include a combination of the following: severe headache; stiff neck, or neck pain with neck movement; sensitivity to light; nausea and vomiting; unusual fatigue; fever; difficulty walking or talking; sudden one-sided weakness; behavioral changes; seizures; unusual skin lesions that persist over months.

Early diagnosis and treatment may increase the chances for survival.

Infections with Balamuthia can be diagnosed on biopsies of skin, brain, and other infected tissues through routine pathologic testing.

There are several recorded cases of Balamuthia infection where the patients survived after long-term treatment with multiple drugs. In some of those cases, the patients were able to return to normal, functioning lives.

Drugs used in treating GAE caused by Balamuthia have included a combination of flucytosine, pentamidine, fluconazole, sulfadiazine and either azithromycin or clarithromycin. Recently, miltefosine in combination with some of these other drugs has shown some promise.

Familiarity:[101] Have you encountered the name of this health condition before this study?

- Yes
- No

Population prevalence: Estimate the percentage of students at the university to whom this event could occur.
----- % (Write a number between 0 and 100. For example 50%).

Stereotype salience: How easy it is to imagine the kind of person who typically suffers from this event?
Very hard to imagine 1 2 3 4 5 6 7 Very easy to imagine

Exempt beliefs: If an individual had not yet experienced this health condition, how likely it is to happen for them?
Impossible 0 1 2 3 4 5 6 7 Extremely likely

Personal experience: Do you know of anyone to whom this health condition has happened?
0 Has not happened to anyone I know before
1 Has happened to few acquaintances
2 Has happened to many acquaintances
3 Has happened to few friends or close relatives
4 Has happened to many friends or close relatives
5 Has happened to me once
6 Has happened to me more than once

Personal susceptibility control: Can you control whether this health condition happens to you?
Very hard to control 1 2 3 4 5 6 7 Very easy to control

Personal severity control: Can you control how severe or serious will the disease be if you encounter it?
Very hard to control 1 2 3 4 5 6 7 Very easy to control

Personal progression rate control: Can you control the rate of progress of this health condition if you encounter it?
Very hard to control 1 2 3 4 5 6 7 Very easy to control

Personal treatment effectiveness control: Can you control the effects of treatment of this health condition if you encounter it?
Very hard to control 1 2 3 4 5 6 7 Very easy to control

Personal general control: In general, can you control this health condition?
Very hard to control 1 2 3 4 5 6 7 Very easy to control

[101] Words in bold are variable names and did not appear on the questionnaire.

Third-person susceptibility control: Can a person control whether this health condition happens to them?

Very hard to control 1 2 3 4 5 6 7 Very easy to control

Third-person severity control: Can a person control how severe or serious will the disease be if they encounter it?

Very hard to control 1 2 3 4 5 6 7 Very easy to control

Third-person progression rate control: Can a person control the rate of progress of this health condition if they encounter it?

Very hard to control 1 2 3 4 5 6 7 Very easy to control

Third-person treatment effectiveness control: Can a person control the effects of treatment of this health condition if they encounter it?

Very hard to control 1 2 3 4 5 6 7 Very easy to control

Third-person general control: In general, can a person control this health condition?

Very hard to control 1 2 3 4 5 6 7 Very easy to control

Part 2: Replication of Pilot Study 1. Participants in each group rated 33 health conditions.

Familiarity: Have you encountered the name of this health condition before this study?

- Yes
- No

Pilot Study 3: Associative strength between the self and health as a trait.

List of personality traits and health- and sickness-related words presented to participants. Numbers next to traits appear in Anderson's (1968) article and reflect trait likeability, where 1 is the most likeable. Numbers were not shown to the participants. Asterisks indicate health- or sickness-related words that were added to the list for the purposes of this pilot but were not in Anderson's original list or were not randomly selected from it.

Positive	**Negative**	**Health**	**Sickness**
1 sincere	475 gossipy	* healthy	* unhealthy (365)
7 intelligent	478 irritating	* able-bodied	* unwell
8 dependable	483 egotistical	* bouncing	* sickly
10 thoughtful	491 discourteous	* fit	* ill
14 reliable	499 irresponsible	* hale	* diseased
16 warm	502 jealous	* hearty	* sick
21 happy	503 unpleasant	* strong	* unfit
27 humorous	504 unreliable	* robust	* unsound
28 responsible	509 quarrelsome	* vigorous (130)	* indisposed
29 cheerful	514 boring	* sound	* weak (429)
30 trustful	522 unfriendly	* well	* feeble
42 courteous	539 conceited	* well-conditioned	
47 imaginative	540 greedy	* whole	
53 polite	543 insincere	* wholesome (113)	
56 forgiving	544 unkind		
62 efficient	545 untrustworthy		
67 alert	548 malicious		
69 witty	550 untruthful		
73 patient	551 dishonest		
83 capable	554 phony		

REFERENCES

Aarts, H., & Dijksterhuis, A. (2003). The silence of the library: Environment, situational norm, and social behavior. *Journal of Personality and Social Psychology, 84*, 18–28.

Ajzen, I. (1991). The theory of planned behaviour. *Organisational Behaviour and Human Decision Processes, 50*, 179-211.

Ajzen, I., & Fishbein, M. (1973). Attitudinal and normative variables as predictors of specific behaviors. *Journal of Personality & Social Psychology, 27*, 41–57.

Ajzen, I., & Fishbein, M. (2000). Attitudes and the attitude-behavior relation: Reasoned and automatic processes. In W. Stroebe, & M. Hewstone (Eds.), *European Review of Social Psychology* (Vol. 11, pp. 1–33). Chichester, England: John Wiley & Sons.

Albarracin, D., McNatt, P. S., Klein, C. T. F., Ho, R. M., Mitchell, A. L., & Kumkale, G. (2003). Persuasive communications to change actions: An analysis of behavioral and cognitive impact in HIV prevention. *Health Psychology, 22,* 166–177.

Allport, G. W. (1961). *Pattern and growth in personality*. NY: Holt, Rinehart and Winston.

Anderson, N. H. (1968). Likableness ratings of 555 personality-trait words. *Journal of Personality and Social Psychology, 9*(3), 272–279.

Bandura, A. (1977). Self-efficacy: Toward a unifying theory of behavioral change. *Psychological Review, 84*(2), 191–215.

Bandura, A. (1998). Health promotion from the perspective of social cognitive theory. *Psychology and Health, 13,* 623–649.

Bargh, J. A., & Chartrand, T. L. (2000). The mind in the middle: A practical guide to priming and automaticity research. In H. Reis, & C. Judd (Eds.), *Handbook of Research Methods in Social Psychology* (pp. 253–285). New York: Cambridge University Press.

Bargh, J. A. (1982). Attention and automaticity in the processing of self-relevant information. *Journal of Personality and Social Psychology, 43*(3), 425-436.

Bargh, J. A. (1989). Conditional automaticity: Varieties of automatic influence in social perception and cognition. In J. S. Uleman, & J. A. Bargh (Eds.), *Unintended Thought* (pp. 3–51). New York: Guilford.

Bargh, J. A. (1994). The four horsemen of automaticity: Awareness, intention, efficiency,

and control in social cognition. In R. S. Wyer, & T. K. Srull (Eds.), *Handbook of social cognition* (pp. 1–40). Hillsdale, NJ: Erlbaum.

Bargh, J. A. (1997). The automaticity of everyday life. In R. S. Wyer (Ed.), *The automaticity of everyday life: Advances in social cognition* (Vol. X, pp. 1–62). New Jersey: Erlbaum.

Bargh, J. A. (2008). Free will is un-natural. In J. Baer, J. Kaufman, & R. Baumeister (Eds.), *Are we free? Psychology and free will* (pp. 128–154). New York: Oxford University Press.

Bargh, J. A., & Ferguson, M. J. (2000). Beyond Behaviorism: On the automaticity of higher mental processes. *Psychological Bulletin, 126*(6), 925–945.

Bargh, J. A., Chaiken, S., Govender, R., & Pratto, F. (1992). The generality of the automatic attitude activation effect. *Journal of Personality and Social Psychology, 62*, 893–912.

Baumeister, R. (1999). The nature and structure of the self: An overview. In R. F. Baumeister (Ed.), *The self in social psychology* (pp. 1–20). Philadelphia: Psychology Press.

Baumeister, R. F., & Cairns, K. J. (1992). Repression and self-presentation: When audiences interfere with self-deceptive strategies. *Journal of Personality and Social Psychology, 62*(5), 851–862.

Baumeister, R. F., Bratslavsky, E., Muraven, M., & Tice, D. M. (1998). Ego depletion: Is the active self a limited resource? *Journal of Personality and Social Psychology, 74*, 1252–1265.

Baumeister, R. F., Dale, K., & Sommer, K. L. (1998). Freudian defense mechanisms and empirical findings in modern social psychology: Reaction formation, projection, displacement, undoing, isolation, sublimation, and denial. *Journal of Personality, 66*(6), 1081–1124.

Beck, A. T., Rush, A. J., Shaw, B. F., & Emery, G. (1979). *Cognitive therapy of depression*. NY: The Guilford Press.

Becker, M. H. (1974). The health belief model and personal health behavior. *Health Education Monographs, 2,* 324–473.

Belk, R. (1988). Possessions and the extended self. *Journal of Consumer Research, 15,* 139–168.

Bradley, G. W. (1978). Self-serving bias in the attribution process: A reexamination of the fact or fiction question. *Journal of Personality and Social Psychology, 36*(1), 56–71.

Bränström, R., Kristjansson, S., & Ullén, H. (2005). Health-related behaviours: Risk perception, optimistic bias, and readiness to change sun related behaviour. *European Journal of Public Health, 16*(5), 492–497.

Brehm, J. W. (1996). *A theory of psychological reactance*. New York: Academic Press.

Brewer, N. T**.**, & Hallman, W. K. (2006). Subjective and objective risk as predictors of influenza vaccination during the vaccine shortage of 2004-2005. *Clinical Infectious Diseases, 43*, 1379–1386.

Brewer, N. T., & Rimer, B. K. (2008). Perspectives on health behavior theories that focus on individuals. In K. Glanz, B. K. Rimer, & K. Viswanath (Eds.), *Health behavior and health education: Theory, research, and practice* (pp. 149–166). San Francisco: John Wiley & Sons.

Brewer, N. T., Chapman, G. B., Gibbons, F. X., Gerrard, M., McCaul, K. D., & Weinstein, N. D. (2007). Meta-analysis of the relationship between risk perception and health behavior: The example of vaccination. *Health Psychology, 26*(2), 136–145.

Brinol, P., & Petty, R. E. (2006). Fundamental processes leading to attitude change: Implications for cancer prevention communications. *Journal of Communication, 56S*, 81–104.

Brockner. J. & Elkind, M. (1985). Self-esteem and reactance: Further evidence of attitudinal and motivational consequences. *Journal of Experimental Social Psychology, 21,* 346–361.

Bruner, J. S. (1957). On perceptual readiness. *Psychological Review, 64*, 123–152.

Burger J. M., & Burns, L. (1988). The illusion of unique invulnerability and the use of effective contraception. *Personality and Social Psychology Bulletin, 14*(2), 264–270.

Bushman, B. J. (1998). Effects of warning and information labels on consumption of full-fat, reduced-fat, and no-fat products. *Journal of Applied Psychology, 83*(1), 97–101.

Cameron, G. T., & Frieske, D. A. (1994). The time needed to answer: Measurement of memory response latency. In A. Lang (Ed.), *Measuring psychological responses to media* (pp. 149–164). Hillsdale, NJ: Lawrence Erlbaum.

Centers for Disease Control and Prevention (2010). Retrieved from http://www.cdc.gov/

Champion, V. L., & Skinner, C. S. (2008). The health belief model. In K. Glanz, B. K. Rimer, & K. Viswanath (Eds.), *Health behavior and health education: Theory, research, and practice* (pp. 45–66). San Francisco: John Wiley & Sons.

Chang, E. C., Asakawa, K., & Sanna, L. J. (2001). Cultural variations in optimistic and pessimistic bias: Do Easterners really expect the worst and Westerners really expect the best when predicting future life events? *Journal of Personality and Social Psychology, 81*(3), 476–491.

Chapman, G. B. (2004). The psychology of medical decision making. In D. J. Koehler, & N. Harvey (Eds.), *Blackwell handbook of judgment and decision making* (pp. 585–603). Malden, MA: Blackwell Publishing.

Chartrand, T. L., Dalton, A. N., & Fitzsimons, G. J. (2007). Nonconscious relationship reactance: When significant others prime opposing goals. *Journal of Experimental Social Psychology, 43,* 719–726.

Chen, S., & Chaiken, S. (1999). The heuristic-systematic model in its broader context. In: S. Chaiken, & Y. Trope (Eds.), *Dual-process theories in social psychology* (pp. 73–96). New York: The Guilford Press.

Cho, H., & Salmon, C. T. (2007). Unintended effects of health communication campaigns. *Journal of Communication. 57*, 293–317.

Christensen-Szalanski, J. J. J., Brck, D. E., Christensen-Szalanski, C. M., & Koepsell, T. D. (1983). Effects of expertise and experience on risk judgments. *Journal of Applied Psychology, 68*(2), 278–284.

Comello, M. L., & Slater, M. D. (2011). The effects of drug-prevention messages on the accessibility of identity-related constructs. *Journal of Health Communication, 16*(5), 458–469.

Correll, J., Spencer, S. J., & Zanna, M. P. (2004). An affirmed self and an open mind: Self-affirmation and sensitivity to argument strength. *Journal of Experimental Social Psychology, 40,* 350–356.

County of LA Department of Public Health. Retrieved from http://reallycheckyourself.org/

Dillard, A. J., McCaul, K. D., & Klein, W. M. P. (2006). Unrealistic optimism in smokers: Implications for smoking myth endorsement and self-protective motivation. *Journal of Health Communication, 11,* 93–102.

Dillard, A. J., McCaul, K. D., Kelso, P. D., & Klein, W. M. P. (2006). Resisting good

news: Reactions to breast cancer risk communication. *Health Communication*, *19*(2), 115–123.

Dillard, J. P., & Shen, L. (2005). On the nature of reactance and its role in persuasive health communication. *Communication Monographs, 72*, 144–168.

Ditto, P. H., & Lopez, D. F. (1992). Motivated skepticism: Use of differential decision criteria for preferred and nonpreferred conclusions. *Journal of Personality and Social Psychology, 63*(4), 568–584.

Eagly, A. H., & Chaiken, S. (1993). *The psychology of attitudes*. Fort Worth, TX: Harcourt Brace Jovanovich College Publishers.

Eddy, D. M. (1984). Probabilistic reasoning in clinical medicine: Problems and opportunities. In D. Kahneman, P. Slovic, & A. Tversky (Eds.), *Judgment under uncertainty: Heuristics and biases* (pp. 249–267). Cambridge: Cambridge University Press.

Eichstaedt, J., & Silvia, P. J. (2003). Noticing the self: Implicit assessment of self-focused attention using word recognition latencies. *Social Cognition, 21*(5), 349–361.

Fazio, R. H. (1990). A practical guide to the use of response latency in social psychological research. In C. Hendrick, & M. S. Clark (Eds.), *Research methods in personality and social psychology* (pp. 74–97). Newbury Park, CA: Sage.

Fenigstein, A., Scheier, M. F., & Buss, A. H. (1975). Public and private self-consciousness: Assessment and theory. *Journal of Consulting and Clinical Psychology, 43*(4), 522–527.

Ferguson, M. (2007). The automaticity of evaluation. In J. A. Bargh (Ed.), *Social psychology and the unconscious: The automaticity of higher mental processes* (pp. 219–264). New York: Psychology Press.

Fernald, A. (1993). Approval and disapproval: Infant responsiveness to vocal affect in familiar and unfamiliar languages. *Child Development, 64,* 657–674.

Fischhoff, B., Slovic, P., & Lichtenstein, S. (1977). Knowing with certainty: The appropriateness of extreme confidence. *Journal of Experimental Psychology: Human Perception and Performance, 3*(4), 552–564.

Fitzsimons, G. J., & Lehmann, D. R. (2004). Reactance to recommendations: When unsolicited advice yields contrary responses. *Marketing Science, 23*(1), 82–94.

Fox, J., & Bailenson, J. N. (2009). Virtual self-modeling: The effects of vicarious reinforcement and identification on exercise behaviors. *Media Psychology, 12*(1), 1–25.

G Power software (2010). Retrieved from http://www.psycho.uni-duesseldorf.de/aap/projects/gpower/

Gerend, M. A., Aiken, L. S., West, S. G., & Erchull, M. J. (2004). Beyond medical risk: Investigating the psychological factors underlying women's perceptions of susceptibility to breast cancer, heart disease, and osteoporosis. *Health Psychology, 23*(3), 247–258.

Gerrard, M., Gibbons, F. X., & Warner, T. D. (1991). Effects of reviewing risk-relevant behavior on perceived vulnerability among women marines. *Health Psychology, 10*(3), 173–179.

Gerrard, M., Kurylo, M., & Reis, T. (1991). Self-esteem, erotophobia, and retention of contraceptive and AIDS information in the classroom. *Journal of Applied Social Psychology, 21*(5), 368–379.

Gibbons, F. X., Eggleston, T. J., & Benthin, A. C. (1997). Cognitive reactions to smoking relapse: The reciprocal relation between dissonance and self-esteem. *Journal of Personality and Social Psychology, 72*(1), 184–195.

Giner-Sorolla, R., & Chaiken, S. (1997). Selective use of heuristic and systematic processing under defense motivation. *Personality and Social Psychology Bulletin, 23*(1), 84–97.

Glanz, K., Rimer B. K., & Viswanath, K. (2008). The scope of health behavior and health education. In K. Glanz, B. K. Rimer, & K. Viswanath (Eds.), *Health behavior and health education: Theory, research, and practice* (pp. 3–22). San Francisco: John Wiley & Sons.

Greenwald, A. G., & Franham, S. D. (2000). Using the Implicit Association Test to measure self-esteem and self-concept. *Journal of Personality and Social Psychology, 79*(6), 1022–1038.

Harris, P. R., Griffin, D. W., & Murray, S. (2008). Testing the limits of optimistic bias: Event and person moderators in a multilevel framework. *Journal of Personality and Social Psychology, 95*(5), 1225–1237.

Harris, P. R., Mayle, K., Mabbot, L., & Napper, L. (2007). Self-affirmation reduces smokers' defensiveness to graphic on-pack cigarette warning labels. *Health Psychology, 26*(4), 437–446.

Higgins, E. T. (1987). Self-discrepancy: A theory relating self and affect. *Psychological Review, 94*(3), 319–340.

Higgins, E. T. (1989). Knowledge accessibility and activation: Subjectivity and suffering from unconscious sources. In J. S. Uleman, & J. A. Bargh (Eds.), *Unintended thought* (pp. 75–123). New York: Guilford.

Hong, S. M., & Faedda, S. (1996). Refinement of the Hong psychological reactance scale. *Educational and Psychological Measurement, 56*(1), 173–182.

Hoorens, V., & Buunk, B. P. (1993). Social comparison of health risks: Locus of control, the person-positivity bias, and unrealistic optimism. *Journal of Applied Social Psychology, 23*(4), 291–302.

Hornik, R. (2002). Public health communication: Making sense of contradictory evidence. In R. Hornik (Ed.), *Public Health Communication: Evidence for Behavior Change* (pp. 1–19). Mahwah, NJ: Lawrence Erlbaum.

Hull, J., Slone, L., Meteyer, K., & Matthews, A. (2002). The nonconsciousness of self-consciousness. *Journal of Personality and Social Psychology, 83*(2), 406–424.

Janz, N. K. (1984). The health belief model: A decade later. *Health Education Quarterly, 11,* 1–47.

Jemmott, J. B., Ditto, P. H., & Croyle, R. T. (1986). Judging health status: Effects of perceived prevalence and personal relevance. *Journal of Personality and Social Psychology, 50*(5), 899–905.

Jones, J. T., Pelham, B. W., & Carvallo, M. (2004). How do I love thee? Let me count the Js: Implicit egotism and interpersonal attraction. *Journal of Personality and Social Psychology, 87*, 665–683.

Kahlor, L., Dunwoody, S., & Griffin, R. J. (2002). Attributions in explanations of risk estimates. *Public Understanding of Science, 11,* 243–257.

Kahneman, D. (1991). Judgment and decision making: A personal view. *Psychological Science, 2*(3), 142–145.

Kaiser Family Foundation (2010). Retrieved from http://www.kff.org/

Kasprzyk, D., Montano, D., & Fishbein, M. (1998). Application of an integrated behavioral model to predict condom use: A prospective study among high HIV risk groups. *Journal of Applied Social Psychology, 28*(17), 1557–1583.

Katapodi, M. C., Facione, N. C., Humphreys, J. C., & Dodd, M. J. (2005). Perceived breast cancer risk: Heuristic reasoning and search for a dominance structure. *Social Science & Medicine, 60,* 421–432.

Kelley, W. M., Macrae, C. N., Wyland, C. L., Caglar, S., Inati, S., & Heatherton, T. F. (2002). Finding the self? An event-related FMRI study. *Journal of Cognitive Neuroscience, 14*(5), 785–794.

Kenen, R., Ardern-Jones, A., & Eeles, R. (2003). Family stories and the use of heuristics: Women from suspected hereditary breast and ovarian cancer (HBOC) families. *Sociology of Health & Illness, 25*(7), 838–865.

Keppel, G., & Wickens, T. D. (2004). *Design and analysis: A researcher's handbook* (4th ed.). Upper Saddle River, N.J.: Pearson Prentice Hall.

Keren, G., & Trigen, K. H. (2004). Yet another look at the heuristics and biases approach. In D. J. Koehler, & N. Harvey (Eds.), *Blackwell handbook of judgment and decision making* (pp. 89–109). Malden, MA: Blackwell Publishing.

Kircher, T. T. J., Brammer, M., Bullmore, E., Simmons, A., Bartels, M., & David, A. S. (2002). The neural correlates of intentional and incidental self processing. *Neuropsychologia, 40,* 683–692.

Klar, Y., & Ayal, S. (2004). Event frequency and comparative optimism: Another look at the indirect elicitation method of self-others risks. *Journal of Experimental Social Psychology, 40,* 805–814.

Klein, C. T. F., & Larsen, M. (2002). Perceived control and the optimistic bias: A meta-analytic review. *Psychology and Health, 17*(4), 437–446.

Klein, W. M. P., & Harris, P. R. (2009). Self-affirmation enhances attentional bias toward threatening components of a persuasive message. *Psychological Science, 20*(12), 1463–1467.

Knäuper, B., Kornik, R., Atkinson, K., Guberman, C., & Aydin, C. (2005). Motivation influences the underestimation of cumulative risk. *Personality and Social Psychology Bulletin, 31*, 1511–1523.

Koole, S. L., Dijksterhuis, A., & van Knippenberg, A. (2001). What's in a name: Implicit self-esteem and the automatic self. *Journal of Personality and Social Psychology, 80*, 669–685.

Kuiper, N. A., & Rogers, T. B. (1979). Encoding of personal information: Self-other differences. *Journal of Personality and Social Psychology, 37*(4), 499–514.

Kunda, Z. (1990). The case for motivated reasoning. *Psychological Bulletin, 108*(3), 480–498.

Larsen, M., & Shepperd, J. A. (2001). Do moderators of the optimistic bias affect personal or target risk estimates? A review of the literature. *Personality and Social Psychology Review, 5*(1), 74–95.

Larwood, L. (1978). Swine flu: A field study of self-serving biases. *Journal of Applied Social Psychology, 8*(3), 283–289.

Lavino, J. G., & Neumann, R. B. (2010). *Psychology of risk perception*. NY: Nova Science Publishers.

Leary, M. R., & Tangney, J. P. (2003). The self as an organizing construct in the behavioral and social sciences. In M. R. Leary, & J. P. Tangney (Eds.), *Handbook of self and identity* (pp. 3–14). New York; London: Guilford.

Lench, H. C., & Ditto, P. H. (2008). Automatic optimism: Biased use of base rate information for positive and negative events. *Journal of Experimental Social Psychology, 44,* 631–639.

Liberman, A., & Chaiken, S. (1992). Defensive processing of personally relevant health messages. *Personality and Social Psychology Bulletin, 19,* 669–679.

Libet, B., Gleason, C., Wright, E. W., & Pearl, D. K. (1983). Time of conscious intention to act in relation to onset of cerebral activity (readiness-potential). *Brian, 106,* 623–642.

Lin, Y., Lin, C., & Ragubir, P. (2003). Avoiding anxiety, being in denial, or simply stroking self-esteem: Why self-positivity? *Journal of Consumer Psychology, 13*(4), 464–477.

Liska, A. E. (1984). A critical examination of the causal structure of the Fishbein/ Ajzen attitude-behavior model. *Social Psychology Quarterly, 47,* 61–74.

Marks, G. (1984). Thinking one's abilities are unique and one's opinions are common. *Personality and Social Psychology Bulletin, 10*(2), 203–208.

Markus, H. (1977). Self-schemata and processing information about the self. *Journal of Personality and Social Psychology, 35*(2), 63–78.

Maruta, T., Colligan, R. C., Malinchoc, M., & Offord, K. P. (2002). Optimism-pessimism assessed in the 1960s and self-reported health status 30 years later. *Mayo Clinic Proceedings, 77,* 748–753.

McCoy, S., Gibbons, F. X., & Gerrard, M. (1999). Self-esteem, compensatory self-enhancement, and the consideration of health risk. *Personality and Social Psychology Bulletin, 25*(8), 954–965.

McNeil, B. J., Pauker, S. G., Sox, H. C., & Tversky, A. (2000). On the elicitation of preferences for alternative therapies. In T. Connolly, H. R. Arkes, & K. R. Hammond (Eds.), *Judgment and decision making: An interdisciplinary reader* (2nd ed., pp. 272–280). Cambridge, UK: Cambridge University Press.

Menon, G., Raghubir, P., & Agrawal, N. (2008). Health risk perceptions and consumer psychology. In C. P. Haugtvedt, P. M. Herr, & F. R. Kardes (Eds.), *Handbook of consumer psychology* (pp. 981–1010). New York: Lawrence Erlbaum Associates.

Meszaros J. R., Asch D. A., Baron J., Hershey, J. C., Kunreuther, H., & Schwartz-Buzaglo, J. (1996). Cognitive processes and the decisions of some parents to forego pertussis vaccination for their children. *Journal of Clinical Epidemiology, 49*(6), 697–703.

Milkman, K. L., Chugh, D., & Bazerman, M. H. (2009). How can decision making be improved? *Perspectives on Psychological Science, 4*(4), 379–383.

Miller, C. H., Burgoon, M., Grandpre, J. R., & Alvaro, E. M. (2006). Identifying principal risk factors for the initiation of adolescent smoking behaviors: The significance of psychological reactance. *Health Communication, 19*(3), 241–252.

Mischel, W., Ebbesen, E. B., & Zeiss, A. M. (1976). Determinants of selective memory about the self. *Journal of Consulting and Clinical Psychology, 44*(1), 92–103.

Montaño, D. E., & Kasprzyk, D. (2008). Theory of reasoned action, theory of planned behavior, and the integrated behavioral model. K. Glanz, B. K. Rimer, & K. Viswanath (Eds.), *Health behavior and health education: Theory, research, and practice* (pp. 67–98). San Francisco: John Wiley & Sons.

Moors, A., & Houwer, J. (2007). What is automaticity? An analysis of its component features and their interrelations. In J. A. Bargh (Ed.), *Social psychology and the unconscious: The automaticity of higher mental processes* (pp. 11–50). New York: Psychology Press.

Moray, N. (1959). Attention in dichotic listening: Affective cues and the influence of instructions. *Quarterly Journal of Experimental Psychology, 11,* 56–60.

Nabi, R. L., & Oliver, M. B. (2009). Introduction. In R. L. Nabi, & M. B. Oliver (Eds.), *The SAGE handbook of media processes and effects* (pp. 1–5). Los Angeles: Sage.

Nisbett, R. E., & Wilson, T. D. (1977). Telling more than we can know: Verbal reports on mental processes. *Psychological Review, 84*, 231–259.

Noar, S. M., Benac, C., & Harris, M. (2007). Does tailoring matter? Meta-analytic review of tailored print health behavior change interventions. *Psychological Bulletin, 133*(4), 673–693.

Nosek, B. A., Greenwald, A. G., & Banaji, M. R. (2007). The implicit association test at age 7: A methodological and conceptual review. In J. A. Bargh (Ed.), *Social psychology and the unconscious: The automaticity of higher mental processes* (pp. 265–292). New York: Psychology Press

O'Keefe, D. J. (2002). *Persuasion theory & research*. Thousand Oaks, CA: Sage.

Partnership for a drug-free America (2010). Retrieved from http://www.drugfree.org/

Paulhaus, D. L. (1993). Bypassing the will: The automatization of affirmations. In D. M. Wegner, & J. W. Pennebaker (Eds.), *Handbook of mental control* (pp. 573–587). NJ: Century Psychology Series.

Payne, B. K. (2001). Prejudice and perception: The role of automatic and controlled processes in misperceiving a weapon. *Journal of Personality and Social Psychology, 81*, 181–192.

Perse, E. M. (1990). Audience selectivity and involvement in the newer media environment. *Communication Research, 17*(5), 675–697.

Peters, E., McCaul, K. D., Stefanek, M., & Nelson, W. (2006). A heuristics approach to understanding cancer risk perception: Contributions from judgment and decision-making research. *Annuals of Behavioral Medicine, 31*(1), 45–52.

Petty, R. E., & Wegner, D. T. (1999). The elaboration likelihood model: Current status and controversies. In S. Chaiken, & Y. Trope (Eds.), *Dual-process theories in social psychology* (pp. 41–72). New York: The Guilford Press.

Radcliffe, N. M., & Klein, W. M. P. (2002). Dispositional, unrealistic, and comparative optimism: Differential relations with the knowledge and processing of risk information and beliefs about personal risk. *Personality and Social Psychology Bulletin, 28*(6), 836–846.

Raghubir, P., & Menon, G. (1998). AIDS and me, never the twain shall meet: The effects of information accessibility on judgments of risk and advertising effectiveness. *Journal of Consumer Research, 25,* 52–63.

Random.org (2010) Retrieved from http://www.random.org/

Reed, G. M., Kemeny, M. E., Taylor, S. E., Wang, H. J., & Visscher, B. R. (1994). Realistic acceptance as a predictor of decreased survival time in gay men with AIDS. *Health Psychology, 13*(4), 299–307.

Reed, M. B., & Aspinwall, L. G. (1998). Self-affirmation reduces biased processing of health-risk information. *Motivation and Emotion, 22*(2), 99–132.

Renner, B. (2004). Biased reasoning: Adaptive responses to health risk feedback. *Journal of Personality and Social Psychology Bulletin, 30*(3), 384–396.

Rimal, R. N. (2001). Perceived risk and self-efficacy as motivators: Understanding individuals' long-term use of health information. *Journal of Communication, 51*(4), 633–654.

Rimal, R. N., & Morrison, D. (2006). A uniqueness to personal threat (UPT) hypothesis: How similarity affects perceptions of susceptibility and severity in risk assessment. *Health Communication, 20*(3), 209–219.

Rimal, R. N., & Real, K. (2003). Perceived risk and efficacy beliefs as motivators of change. *Human Communication Research, 29*(3), 370–399.

Robberson, M. R., & Rogers, R. W. (1988). Beyond fear appeals: Negative and positive persuasive appeals to health and self-esteem. *Journal of Applied Social Psychology, 18*(3), 277–287.

Rogers, R. W. (1975). A protection motivation theory of fear appeals and attitude change. *Journal of Psychology, 91,* 93–114.

Rogers, R., & Prentice-Dunn, S. (1997). Protection motivation theory. *Handbook of Health Behavior Research, 1*, 113–132.

Rogers, T. B., Kuiper, N. A., & Kirker, W. S., (1977). Self-reference and the encoding of personal information. *Journal of Personality and Social Psychology, 35*(9), 677–688.

Rosenberg, M. (1979). *Conceiving the self*. NY: Basic Books.

Ross, L., Green, D., & House, P. (1977). The "false consensus effect": An egocentric bias in social perception and attribution processes. *Journal of Experimental Social Psychology, 13*(3), 279–301.

Salovery, P., & Birnbaum, D. (1989). Influence of mood on health-relevant cognitions. *Journal of Personality and Social Psychology, 57*(3), 539–551.

Scheier, M. F., & Carver, C. S. (1992). Effects of optimism on psychological and physical well-being: Theoretical overview and empirical update. *Cognitive Therapy and Research, 16*(2), 201–228.

Scheier, M. F., Carver, C. S. & Bridges, M. W. (1994). Distinguishing optimism from neuroticism (and trait anxiety, self-mastery, and self-esteem): A reevaluation of

the life orientation test. *Journal of Personality and Social Psychology, 67*(6), 1063–1078.

Sedikides, C., & Green, J. D. (2000). On the self-protective nature of inconsistency-negativity management: Using the Person Memory Paradigm to examine self-referent memory. *Journal of Personality and Social Psychology, 79*(6), 906–922.

Sedikides, C. (1993). Assessment, enhancement, and verification determinants of the self-evaluation process. *Journal of Personality and Social Psychology, 65*(2), 317–338.

Seibel, C. A., & Dowd, E. T. (1999). Reactance and therapeutic noncompliance. *Cognitive Therapy and Research, 23*(4), 373–379.

Sharot, T., Riccardi, A. M., Raio, C. M., & Phelps, E. A. (2007). Neural mechanisms mediating optimism bias. *Nature, 450,* 102–106.

Sheer, V. C., & Cline, R. J. (1994). The development and validation of a model explaining sexual behavior among college students: Implications for AIDS communication campaigns. *Health Communication Research, 21*(2), 280–304.

Shepperd, J. A., Helweg-Larsen, M., & Ortega, L. (2003). Are comparative risk judgments consistent across time and events? *Personality and Social Psychology Bulletin, 29*(9), 1169–1180.

Sherer, M., Maddux, J. E., Mercandante, B., Prentice-Dunn, S., Jacobs, B., & Rogers, R. W. (1982). The self-efficacy scale: Construction and validation. *Psychological Reports, 51,* 663–671.

Sherman, D. A. K., Nelson, L. D., & Steele, C. M. (2000). Do messages about health risks threaten the self? Increasing the acceptance of threatening health messages via self-affirmation. *Personality and Social Psychology Bulletin, 26*(9), 1046–1058.

Showers, C. J., & Zeigler-Hill, V. (2003). Organization of self-knowledge: Features, functions, and flexibility. In M. R. Leary, & J. P. Tangney (Eds.), *Handbook of self and identity* (pp. 47–67). New York; London: Guilford.

Shrauger, J. S., & Rosenberg, S. E. (1970). Self-esteem and the effects of success and failure feedback on performance. *Journal of Personality, 38*(3), 404–417.

Shrauger, J. S., & Sorman, P. B. (1977). Self-evaluations, initial success and failure, and improvement as determinants of persistence. *Journal of Consulting and Clinical Psychology, 45*(5), 784–795.

Simon, H.A. (1955). A behavioral model of rational choice. *Quarterly Journal of Economics, 69,* 7–19.

Slovic, P. (1987). Perception of risk. *Science, 236*(4799), 280–285.

Slovic, P., & Peters, E. (2006). Risk perception and affect. *Current Directions in Psychological Science, 15*(6), 322–325.

Slovic, P., Finucane, M. L., Peters, E., & MacGregor, D. G. (2004). Risk as analysis and risk as feelings: Some thoughts about affect, reason, risk, and rationality. *Risk Analysis, 24,* 311–322.

Slovic, P., Fischoff, B., & Lichtenstein, S. (1982). Facts versus fears: Understanding perceived risk. In D. Kahneman, P. Slovic, & A. Tversky (Eds.), *Judgment under uncertainty: Heuristics and biases* (pp. 463–489). Cambridge: Cambridge University Press.

Smith, G. E., Gerrard, M., & Gibbons, F. X. (1997). Self-esteem and the relation between risk behavior and perceptions of vulnerability to unplanned pregnancy in college women. *Health Psychology, 16*(2), 137–146.

Srull, T. K., & Wyer, R. S. (1979). Category accessibility and social perception: Some implications for the study of person memory and interpersonal judgments. *Journal of Personality and Social Psychology, 38*(6), 841–856.

Strecher, V. J., Kreuter, M. W., & Kobrin, S. C. (1995). Do cigarette smokers have unrealistic perceptions of their heart attack, cancer, and stroke risks? *Journal of Behavioral Medicine, 18*(1), 45–54.

Svenson, O. (1984). Cognitive processes in judging cumulative risk over different periods of time. *Organizational Behavior and Human Performance, 33*, 22–41.

Swann, W. B., Hixon, J. G., Stein-Seroussi, A., & Gilbert, D. T. (1990). The fleeting gleam of praise: Cognitive processes underlying behavioral reactions to self-relevant feedback. *Journal of Personality and Social Psychology, 59*(1), 17–26.

Symons, C. S., & Johnson, B. T. (1997). The self-reference effect in memory: A meta-analysis. *Psychological Bulletin, 121*(3), 371–394.

Tabachnick, B., & Fidell, L. S. (2007). *Using multivariate statistics*. Boston: Pearson/Allyn & Bacon.

Taylor, S. E., & Brown, J. D. (1988). Illusion and well being: A social psychological perspective on mental health. *Psychological Bulletin, 103*(2), 193–210.

Taylor, S. E., & Brown, J. D. (1994). Positive illusions and well being revisited: Separating fact from fiction. *Psychological Bulletin, 116*(1), 21–27.

Taylor, S. E., Kemeny, M. E., Aspinwall, L. G., Schneider, S. G., Rodriquez, R., & Herbert, M. (1992). Optimism, coping, psychological distress, and high-risk sexual behavior among men at risk for Acquired Immunodeficiency Syndrome (AIDS). *Journal of Personality and Social Psychology, 63*(3), 460–473.

Taylor, S. E., Lerner, J. S., Sherman, D. K., Sage, R. M., & McDowell, N. K. (2003). Are self-enhancing cognitions associated with healthy or unhealthy biological profiles? *Journal of Personality and Social Psychology, 85*(4), 605–615.

Tversky, A., & Kahneman, D. (1974). Judgment under uncertainty: Heuristics and biases. *Science, 185*(4157), 1124–1131.

Tversky, A., & Kahneman, D. (1986). Rational choice and the framing of decisions. *Journal of Business, 59*(4), S251–S278.

van Koningsbruggen, G. M., Das, E., & Roskos-Ewoldsen, D. R. (2009). How self-affirmation reduces defensive processing of threatening health information: Evidence at the implicit level. *Health Psychology, 28*(5), 563–568.

Velde, F. W., Hooykaas, C., & Pligt, J. (992). Risk perception and behavior: Pessimism, realism, and optimism about Aids-related health behavior. *Psychology and Health, 6,* 23–38.

Viswanath, K., Wallington, S. F., & Blake, K. (2009). Media effects and population health. In R. L. Nabi, & M. B. Oliver (Eds.), *The SAGE handbook of media processes and effects* (pp. 312–329). Los Angeles: Sage.

Wallston, B. S., Wallston, K. A., Kaplan, G. D., & Maides, S. A. (1976). Development and validation of the health locus of control (HLC) scale. *Journal of Consulting and Clinical Psychology, 44*(4), 580–585.

Wallston, K. A., & Wallston, B. S. (1981). Health locus of control scales. In H. M. Lefcourt (Ed.), *Research with the locus of control construct: Assessment methods* (Vol. 1, pp. 189–243). New York: Academic Press.

Watson, L. A., Dritschel, B., Obansawin, M. C., & Jentzsch, I. (2007). Seeing yourself in a positive light: Brain correlates of the self-positivity bias. *Brain Research, 1152,* 106–119.

Wegener, D. T., & Petty, R. E. (1995). Flexible correction processes in social judgment: The role of naïve theories in corrections for perceived bias. *Journal of Personality and Social Psychology, 68*, 36–51.

Wegner, D. M., & Wheatley, T. (1999). Apparent mental causation: Sources of the experience of will. *American Psychologist, 54*, 480–492.

Weinstein, N. D. (1980). Unrealistic optimism about future life events. *Journal of Personality and Social Psychology, 39*(5), 806–820.

Weinstein, N.D. (1984). Why it won't happen to me: Perceptions of risk factors and susceptibility. *Health Psychology, 3*(5), 431–457.

Weinstein, N. D. (1987). Unrealistic optimism about susceptibility to health problems: Conclusions from a community-wide sample. *Journal of Behavioral Medicine, 10*(5), 481–500.

Weinstein, N. D., & Klein, W. M. (2002). Resistance of personal risk perceptions to debiasing interventions. In T. Gilovich, D. Griffin, & D. Kahneman (Eds.), *Heuristics and biases: The psychology of intuitive judgment* (pp. 313–323). Cambridge, UK: Cambridge University Press.

Weinstein, N. D., Slovic, P., & Gibson, G. (2004). Accuracy and optimism in smokers' beliefs about quitting. *Nicotine & Tobacco Research, 6*(Supplement 3), S375–S380.

Wheeler, S. C., DeMarree, K. G., & Petty, R. E. (2007). Understanding the role of the self in prime-to-behavior effects: The active-self account. *Personality and Social Psychology Review, 11*, 234–261.

Wheeler, S. C., Morrison, K. R., DeMarree, K. G., & Petty, R. E. (2008). Does self-consciousness increase or decrease priming effects? It depends. *Journal of Experimental Social Psychology, 44,* 882–889.

Windschitl, P. D. (2002). Judging the accuracy of a likelihood judgment: The case of smoking risk. *Journal of Behavioral Decision Making, 15*, 19–35.

Witte, K. (1992). Putting the fear back into fear appeals: The extended parallel process model. *Communication Monographs, 59,* 329–349.

Witte, K., & Allen, M. (2000). A meta-analysis of fear appeals: Implications for effective public health campaigns. *Health Education and Behavior, 27,* 591–615.

Woloshin, S., Schwartz, L. M., Black, W. C., & Welch, H. G. (1999). Women's perceptions of breast cancer risk: How you ask matters. *Medical Decision Making, 19*, 221–229.

Wyer, R. S., & Frey, D. (1983). The effects of feedback about self and others on the recall and judgments of feedback-relevant information. *Journal of Experimental Psychology, 19,* 540–559.

Zeckhauser, R. J., & Viscusi, W. K. (2000). Risk with reason. In T. Connolly, H. R. Arkes, & K. R. Hammond (Eds.), *Judgment and decision making: An interdisciplinary reader* (2nd ed., pp. 465–478). Cambridge, UK: Cambridge University Press.

Zillmann, D. (2002). Exemplification: Theory of media influence. In J. Bryant, & D. Zillmann (Eds.), *Media effects: Advances in theory and research* (pp. 19–42). Mahwah, NJ: Lawrence Erlbaum Associates.

Zuckerman, M. (1979). Attribution of success and failure revisited, or: The motivational bias is alive and well in attribution theory. *Journal of Personality, 47*(2), 245–287.