Impact of Manipulated Perceived Efficacy and Self-Affirmation on Measures of Risk, Efficacy, and Intention

by

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Abstract

In the developed world, the principal sources of morbidity and mortality are diseases of lifestyle, and one of the central goals of health promotion is the encouragement of risk-reducing behaviour. In a series of 3 studies, the present program of research examined the effect of self-affirmation and manipulated perceived efficacy on perceptions of efficacy, risk perception, and risk-reducing behavioural intentions. Participants were undergraduate students who were randomly assigned to a self-affirmation manipulation (self-affirmation versus no self-affirmation) and a perceived efficacy manipulation (high versus low versus baseline), followed by exposure to negative health risk information (risk of a heart attack or colorectal cancer). Across the 3 studies, factor analyses indicated 3 distinct categories of risk-reducing intentions: intentions associated with maintaining an active lifestyle, seeking medical advice and assessment, and maintaining a healthy diet. There was little evidence that self-affirmation affected efficacy, risk, or intentions. Structural equation modeling and meta-analytic analyses suggested the presence of a suppression effect for risk perception: a manipulation designed to increase perceived efficacy had direct positive effects on risk, but also had indirect negative effects on risk, with measured efficacy acting as a mediator. These analyses also showed that the 3 categories of behavioural intentions had distinct (and often different) antecedents. These results highlight the complexity of variables in health risk behaviour. Implications and future directions are discussed.

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Chapter 1: Introduction

Over the past century we have seen a sharp increase in many preventable diseases such as cancer and cardiovascular disease (Taylor, 2006). Because of the clear role of behaviour in the development of these common and preventable diseases (e.g., smoking, diet, and alcohol consumption), individuals who have these diseases or who are at risk of developing them are generally advised by physicians to alter their health behaviours. In addition, public health campaigns are frequently conducted to alter people's health-relevant behaviours. Unfortunately, campaigns and physician advice often fail to alter the behaviours of the general public and the at-risk individual. Given the seriousness of these diseases, a great deal of health behaviour research has attempted to elucidate the reasons for this failure.

Perceived Risk

Perceived risk, one's belief about the likelihood of personal harm, is a central concept in most theories of health behaviour (for reviews see Sutton, 1987; Weinstein, 1993). Within the health domain, most theories of behavioural change identify perceived risk (also called perceived *probability*, *likelihood*, *susceptibility*, or *vulnerability*) as an important factor in explaining the behaviour change process. Theses theories include the health belief model (Becker, 1974; Janz & Becker, 1984), the subjective expected utility theory (Edwards, 1954; Ronis, 1992; Sutton, 1982), protection motivation theory (Maddux & Rogers, 1983; Prentice-Dunn & Rogers, 1986; Rogers, 1975; Rogers, 1983), and the theory of reasoned action (Fishbein & Azjen, 1975).

According to these theories, when individuals perceive their risk of some adverse health outcome to be high (or any other adverse outcome), they are more likely to take

preventative action to reduce their risk. This premise is supported by the large number of studies reporting significant correlations between perceived risk and behaviour. In meta-analyses, the effect sizes found for risk perceptions tend to be significant but small. Harrison, Mullen, and Green (1992) examined the predictive validity of the health belief model, and reported an effect size *r* of .15 for the relationship of perceived likelihood to health behaviour, and an effect size *r* of .08 for the relationship of perceived illness severity (i.e., seriousness of the risk) to health behaviour across three categories of health behaviours and two categories of study designs. Janz and Becker (1984) reviewed studies based on the health belief model and found significant results for 30 of 37 examining perceived likelihood, and for 24 of 30 studies examining perceived severity. In their review of studies related to protection motivation theory, Floyd, Prentice-Dunn, and Rogers (2000) reported an effect size *r* of .20 for studies measuring perceived likelihood and an effect size *r* of .19 for studies examining perceived severity.

Taken together, these studies show a clear relationship between perceived risk and behaviour. Thus, it appears that some benefit could be accrued if risk perceptions could be influenced. Specifically, one might be able to produce positive behavioural changes in health behaviour simply by altering individuals' perceptions of their risk.

Unrealistic Optimism

The results of studies examining the relationship between risk perception and behaviour would suggest that enhanced perceptions of risk should cause an increase in risk-reducing behaviours. In an ideal world, the simple act of providing individuals with facts regarding their risk of a particular hazard would be enough to alter their risk

perceptions and subsequent risk-reducing behaviours. However, the process of increasing both risk perception and risk-reducing behaviour is not as simple as it seems.

People do not always accept risk information at face value. One reason for this tendency is to disregard risk information is the propensity of individuals to engage in self-serving biases. Research has demonstrated that people tend to believe they are better than others (e.g., Goethals, Messick, & Allison, 1991; Hoorens, 1993), and self-serving or self-enhancing biases are well-documented phenomenon. The perception of self that most individuals hold is heavily weighted toward the positive end of the scale (Taylor & Brown, 1988). Individuals believe that they are more trustworthy, moral, and physically attractive than others and that they are above-average teachers, managers, and leaders (Sedikides, Campbell, Reeder, & Elliot, 1998). For example, Brown (1986) examined the relationship between self-appraisals and appraisals of others, and found that participants displayed a pronounced "self-other bias", such that positive attributes were rated as more descriptive of the self than of others, whereas negative attributes were rated as less descriptive of the self than of others. In many cases, these biases can be beneficial to the individual. For example, such illusions can promote mental health and the ability to be happy or contented, as well as the ability to engage in productive and creative work (Taylor, 1988; Taylor & Brown, 1988).

Self-serving biases can have varied effects: not only do individuals tend to attribute desirable attributes to themselves, but they also tend to assert that they are less likely than others to experience negative events, and more likely to experience positive events. Research has demonstrated that people will estimate the likelihood that they will experience a wide variety of pleasant events, such as liking their first job, getting a good

salary, or having a gifted child, as higher than those of their peers (Weinstein, 1980). Conversely, when asked their chances of experiencing a wide variety of negative events, including having an automobile accident (Robertson, 1977), having trouble finding a job, getting divorced a few years after marriage (Weinstein), or being a crime victim (Perloff & Fetzer, 1986) most people believe that they are less likely than their peers to experience such negative events. Weinstein has termed this underestimation of perceived risk as an *optimism bias*, or *unrealistic optimism*. Both children and adults overestimate the degree to which they will do well on future tasks (e.g., Crandall, Solomon, & Kelleway, 1955; Irwin, 1953; Marks, 1951; Taylor & Brown, 1988), and they are more likely to provide such overestimates the more personally important the task is (Frank, 1953).

Unrealistic Optimism and Perceived Risk

While at times beneficial, self-serving biases such as unrealistic optimism can often be more harmful than helpful. One important domain where such biases can produce negative effects is in perceived susceptibility to health problems. Numerous studies show that persons tend to underestimate their risk of health-related problems (e.g., Prokhorov et al., 2003; Weinstein, 1980; Weinstein, 1982; Weinstein, 1987). Studies examining the concept of unrealistic optimism have demonstrated optimistic biases for conditions such as heart disease, lung cancer, and alcoholism, as well as for motor vehicle crashes. This bias has important implications, as those who believe that, relative to their peers, they are unlikely to suffer future adverse events across a number of different life event domains are thought to be at an increased risk for later health

difficulties as a result of their decreased motivation to engage in health-protective behaviours (Davidson & Prkachin, 1997).

Even though unrealistic optimism is a well-documented phenomenon, its origins are less clear. In his review of the bias, Weinstein (1996) purported that, as with other self-serving beliefs, there appear to be multiple determinants. One of the most prominent explanations for unrealistic optimism are motivated distortions, where the goal is to protect self-esteem, project a positive social image, or reduce anxiety about a health risk (e.g., Gerrard, Gibbons, & Warner, 1991; Hoorens, 1993; Taylor et al., 1992; Weinstein, 1980). For example, Weinstein (1980; 1983; 1984) has suggested that one reason people underestimate their risk because of selective focus. That is, they tend to focus primarily on their risk-reducing attributes, including factors such as genetic predispositions and preventative behaviors. He has also argued that people tend to ignore their own risk-increasing behaviours and fail to recognize that others may engage in risk-reducing behaviours as well. Another motivational factor thought to affect risk perception is the perceived undesirability of the event. This means that the more undesirable the event, the more likely vulnerability to it will be underestimated (Gerrard et al., 1991; Weinstein, 1980). Finally, unmotivated or cognitive errors in calculating risk constitute another type of explanation. That is, individuals who are unrealistically optimistic display this bias because they are making errors in determining their risk of a particular hazard. These errors are not made consciously, and may be due to either motivational or cognitive factors (Weinstein, 1996).

In health risk studies, researchers have used these explanations of unrealistic optimism to develop interventions designed to reduce the bias. Their methods generally

fall into one of four types of strategies: giving participants information about the self-protective action of others; drawing participants' attention to the risk factors of a particular hazard; giving participants objective, individually-tailored risk feedback; and using self-affirmation to promote acceptance.

Influence of Others. A number of studies have examined the influence of others on one's level of unrealistic optimism. Researchers (e.g., Weinstein, 1980; Weinstein & Lachendro, 1982) suggest that individuals may be unrealistically optimistic because in making their comparative judgments, they are comparing themselves to an inaccurate standard, a person who has only high-risk characteristics (e.g., an overweight smoker). One possibility is that such individuals may be unaware of the risk-reducing factors others have in their favour, and tend to underestimate them. A second possibility is that individuals are aware of information regarding others' risk factors, but egocentric tendencies tend to make individuals think only about themselves. Thus, by giving individuals information about the average other, they are able to compare themselves to an accurate standard, and perhaps a person who has lower-risk characteristics (e.g., a physically active non-smoker). Within this category of intervention, one tactic has involved forcing participants to think about others by giving them information about *types* of risk factors generated by others (e.g., participants are shown risk factors and told they were generated by similar others), which has shown some effectiveness in reducing unrealistic optimism (e.g., Weinstein, 1982; Weinstein & Lachendro, 1982). A second tactic has been to give participants information about the actual *responses* of similar others on relevant risk factors (e.g., Weinstein, 1983), which has also been shown to significantly reduce the optimism bias in negative health events.

Attention to Risk Factors. A second strategy used to try and reduce unrealistic optimism has been to draw participants' attention to risk factors associated with a given negative event. This approach to bias reduction assumes that optimistic biases result from unsystematic or incomplete attention to risk factor information. By taking an educational approach and providing risk factor information it is thought that one can correct individuals' misunderstandings. Within this category of intervention, one tactic has been to reduce the bias by simply having participants rate their standing on associated risk factors prior to making comparative risk judgments for health and safety events. In his study examining the use of this tactic, Weinstein (1983) produced unexpected (and opposite) results, and demonstrated that rating oneself on risk factors substantially *increased* optimistic biases. In a similar study, Weinstein and Klein (1995) showed that having participants think about their standing on relevant risk factors, without providing clear feedback about what constituted favourable or unfavourable standing on these factors, did not significantly affect unrealistic optimism.

A second tactic has been to present risk factors in such a way that participants would see their own attributes as less than ideal, for example, focusing participants' attention on the positive end of a risk factor (e.g., indicating whether they have never gotten drunk with the risk of developing alcoholism), having participants form a mental image of a low-risk individual, and having participants generate their own list of risk factors that that would keep them from developing (risk-decreasing) health problems. In studies examining this tactic, such manipulations have generally produced negligible results (e.g., Weinstein & Klein, 1995).

Objective Risk Feedback. A third category of intervention has been to provide each participant with individualized, objective feedback regarding their risk of a particular negative event, as a way of correcting any unmotivated or cognitive errors in risk calculation. Avis, Smith, and McKinlay (1989) examined the effectiveness of this strategy in adults' perceptions of heart attack risk, and showed that the effect of objective risk feedback on risk perceptions did not have consistent effects on risk perception, but the effects were greatest for those who were objectively above average in risk. Kreuter and Strecher (1995) looked at the effect of providing objective risk feedback on individuals' perceived risk of several negative events, and once again, results were inconsistent. Results showed that objective risk feedback was effective in increasing perceived stroke risk among participants who had initially underestimated their risk, but it did not increase risk perceptions in those who had underestimated their risk of heart attack, cancer, and motor vehicle crash.

Taken together, it appears as though studies attempting to decrease unrealistic optimism through the tactics of using the influence of others, drawing attention to risk factors, and providing objective risk feedback have been largely ineffective. Some significant effects have been found, but in many cases these effects have been small and/or inconsistent. Thus, researchers have had to turn to other types of interventions for reducing unrealistic optimism. One such intervention has been the use of self-affirmation.

Self-Affirmation

Self-affirmation involves thinking about one's "affirming and sustaining valued self-images" (Steele, 1988, p. 291), and self-affirmation theory proposes that thoughts and actions are motivated by a desire to maintain a self-image as moral, adaptive, and

capable (Sherman, Nelson, & Steele, 2000). Researchers have hypothesized that individuals respond defensively when they receive threatening information (e.g., negative health information) as a means of maintaining their positive self-image. Threatening information can create dissonance, which can lead to defensiveness because the person's self-image is threatened. To restore one's self-image as positive, an individual may deny the risk and refuse to perform adaptive behaviours to reduce their risk (Fry & Prentice-Dunn, 2005).

Self-affirmation theory predicts that if an individual's self-image can be affirmed through some other means, the need to respond defensively to the threatening information should be reduced (Sherman et al., 2000). Thus, according to the theory, an individual does not have to deal with the threatening information directly, and can respond by self-affirming on any unrelated aspect of him or herself. For example, an individual who fails academically does not have to deal with that threat directly to maintain their positive self-image. Instead, the individual can preserve this positive image by affirming on something completely unrelated, such as, for example, reflecting on past acts of kindness or thinking about an important value. Given this relationship, self-affirmation theory would also predict that if one self-affirms *prior* to receiving threatening information about him or herself, he or she might be more likely to accept that information. Research has supported this, with a several studies suggesting that self-affirmations can reduce defensive processing of risk information (e.g., Reed & Aspinwall, 1998; Sherman et al., 2000). Self-affirmation theory has been applied to a number of domains, including stereotyping (Fein & Spencer, 1997), intellectual performance (Martens, John, Greenberg, & Schimel, 2006), as well as health risk – including its effect on risk acceptance and risk-reducing

behavioural intentions and behaviours. Within the health risk domain, several different types of self-affirmation manipulations have been used, including reflecting on past acts of kindness (e.g., Armitage, Harris, Hepton, & Napper, 2008; Reed & Aspinwall), writing or answering questions about a highly important value (e.g., Harris & Napper; Sherman et al., 2000; van Koningsbruggen & Das, 2009), and writing down one's desirable characteristics (e.g., Harris, Mayle, Mabbott, & Napper, 2007).

Self-Affirmation, Perceived Risk, and Behaviour Change

Several studies have examined the use of self-affirmation on risk perception, and a few of these studies have shown that self-affirmed participants are more accepting of their risk and show greater intention to reduce their risk. For example, Sherman et al. (2000) had participants self-affirm after watching a video about HIV risks. Self-affirmed participants saw themselves at higher risk for HIV and showed greater intention to take precautions (i.e., purchased more condoms in the HIV paradigm). Similarly, Harris and Napper (2005) had participants self-affirm prior to reading about the link between alcohol and breast cancer. Consistent with the findings of Sherman et al., results demonstrated that higher-risk self-affirmed participants had higher ratings of both risk and intention to reduce alcohol consumption. However, the changes in intention did not translate into actual behavioural change – no significant effects were found between self-affirmed and non-affirmed participants on alcohol consumption at follow-up.

Even though there have been several studies showing positive effects of self-affirmation on risk perception, these effects have not been consistent. In their study examining the use of self-affirmation in cigarette smokers, Harris et al. (2007) had participants view graphic images depicting the health consequences of smoking after self-

affirming. Contrary to the findings of Sherman et al. (2000) and Harris and Napper (2005), results showed that self-affirmation did not affect perceived vulnerability to smoking-related disease. However, self-affirmed participants did report greater intentions to quit smoking. In addition, at follow-up, self-affirmed participants were more desirous of cutting down smoking, but there were no differences in self-reported cigarette consumption.

Other studies have not examined the effect of self-affirmation on risk perception directly, but have examined its effects on other related variables. One such variable has been message acceptance. Sherman et al. (2000) had female participants self-affirm after receiving information about the link between caffeine consumption and breast cancer. The authors found that coffee-drinking, self-affirmed participants were more accepting of the message (i.e., whether there was a link between caffeine and breast cancer) and showed greater intention to reduce caffeine consumption. Armitage and colleagues (2008) found similar results in their study examining the use of self-affirmation in a sample of adult cigarette smokers. After self-affirming, participants read a threatening message, and results showed that self-affirmation significantly increased message acceptance (i.e., how important it is that people stop smoking), intentions to quit smoking, and behaviour (i.e., whether or not participants took an antismoking leaflet).

Several studies have examined the effect of self-affirmation on message derogation and message processing, and these studies have generally shown positive effects. van Koningsbruggen and Das (2009) examined the use of self-affirmation in type 2 diabetes, and found that in at-risk participants, self-affirmation decreased the derogation of threatening type 2 diabetes information, increased intentions to do an

online risk test, and promoted online risk test-taking. In low-risk participants, self-affirmation decreased intentions and online risk test-taking. Reed and Aspinwall (1998) found that self-affirmed, higher risk caffeine consumers showed evidence of less biased processing of a message describing the link between caffeine and breast cancer. Interestingly, these participants also had significantly *lower* intentions to reduce their caffeine consumption, but at follow-up there was no evidence that these participants were less likely to decrease their caffeine consumption.

Finally, Epton and Harris (2008) did not examine the effects of self-affirmation on risk perception or on any aspect of message acceptance: they only examined the effects of self-affirmation on behavioural change. These authors had participants read a message outlining the health benefits of fruit and vegetable consumption following self-affirmation. They found no effect on intentions to eat the recommended number of fruits and vegetables, which was inconsistent with the results of most of the aforementioned studies. However, results did show that self-affirmed participants ate significantly more portions of fruits and vegetables in the week following the study.

Status of the Literature

Over the past two decades, the unrealistic optimism literature has made many advances. The bias has been demonstrated in a variety of populations, and for a wide range of health conditions. Unfortunately, most bias reduction techniques have demonstrated negligible effects. In the handful of studies that exist, self-affirmation is generally demonstrated to produce greater message acceptance and risk perception, but it is not clear whether this effect translates into the desired behaviour changes. Studies that have examined the relationship between self-affirmation and behaviour change have

generally produced mixed results, with both significant and nonsignificant effects for risk-reducing behaviours. A greater number of studies have examined risk-reducing behavioural intentions, but this research has again, produced mixed results, with both significant and nonsignificant effects.

Thus, the question remains: why do changes in risk perception in self-affirmed participants not result in greater behavioural change or, at least, in greater intention to change? In health, most theories of behavioural change identify perceived risk as an important factor in explaining the behavioural change process (e.g., Becker, 1974; Fishbein & Azjen, 1975). These theories premise that when individuals perceive their risk of a negative health event to be high, they are more likely to take preventative action to reduce their risk. Self-affirmation has been demonstrated to increase individuals' perception of risk, but at the same time, behavioural change is negligible.

There could be several reasons for the mixed behavioural intentions and behaviour change results. First, perhaps self-affirmation researchers have not considered the potential dimensionality of behavioural intentions (and subsequent behaviours). The aforementioned studies all measured very specific (and in most cases single-item) behavioural intentions (e.g., intention to quit smoking; intention to reduce alcohol) and behaviours (e.g., number of cigarettes smoked; number of leaflets taken), and these intentions and behaviours differ from study to study. However, it may be that self-affirmation affects certain intentions and/or behaviours more than others. There are numerous domains of health promoting behaviour one could consider, for example, dietary (e.g., modifying diet or alcohol consumption) or information-seeking (e.g., risk assessment for a given disease). Considering the self-affirmation studies reviewed above,

the intentions and behaviours measured in each of the studies could fall into different domains of health promoting behaviour, and it may be that self-affirmation's effects on these domains differs. For example, dietary changes could be affected more than information seeking behaviour, or vice versa. Thus, the inconsistent and mixed results on intentions and behaviours outlined above may in part be due to a lack of consideration of the potential dimensionality of health promoting behaviour, and to the differences in the types of intentions and behaviours measured.

A second reason for the mixed behavioural change results could be because of the self-affirmation itself. Self-affirming oneself may reduce defensiveness and increase acceptance, but at the same time, it may inhibit the behavioural change one would expect following an increase in perceived susceptibility (e.g., Becker, 1974; Fishbein & Azjen, 1975; Weinstein, 1988). Researchers have speculated that reactions to negative health threat information, particularly those associated with risk-increasing behaviours, may be directed in part to bolstering self-concept and reducing perceived inconsistency (Boney-McCoy, Gibbons, & Gerrard, 1999). According to self-affirmation theory, actions are guided by a need to maintain global self-worth (Aronson, Cohen, & Nail, 1999). So, while discontinuing a risk-increasing behaviour or adopting a healthy behaviour would address both the need to reduce health risk and the need to reaffirm oneself as a rational, responsible person, it is not the simplest solution. Individuals who are confronted with the acknowledgement of risky behaviours may look for a quicker solution to the emotional component of their distress (Boney-McCoy et al.). Research has demonstrated that when people are faced with a threat to their self-image, they may reduce discomfort by being allowed to reaffirm their worth in other areas (Aronson et al; Steele & Liu,

1983). Thus, in self-affirmation manipulations, individuals who have self-affirmed and become more accepting of negative health information may not produce any behaviour change because any discomfort produced by the acceptance of health risk has been reduced by self-affirmation, and there is subsequently no need to change their behaviour. Thus, it may be the case that self-affirmation produces contradictory effects, in that it increases an individual's acceptance of negative health risk information, but at the same time, it undermines an individual's need to take steps to decrease their risk.

A third reason for the inconsistencies in intentions and behaviours may be related to perceived risk. As described previously, individuals are more likely to take preventative action to reduce their risk when they perceive their risk of an adverse health outcome to be high. But perhaps we need to consider more than just perceived risk's effects on intentions and behaviours. It may be that an individual's perceived risk for a given health problem also affects other variables, which in turn can have an effect on risk-reducing behavioural intentions and behaviours, in a mediation-like relationship. Moreover, there may be other variables that could influence intentions and behaviours, independent of perceived risk. Acceptance of risk represents only one step in the reduction of risky health behaviours (Hochbaum, 1958; Rosenstock, 1966). Thus, to fully understand behaviour change, one must consider the influence of other variables.

Perceived Efficacy

One important variable to consider is *perceived efficacy* or *perceived behavioural control*. In their meta-analysis of fear appeals, Witte and Allen (2000) note that there are three key variables involved in effective fear appeals: fear (a negative emotion accompanied by high levels of arousal), perceived threat (perceived susceptibility to and

severity of the threat), and perceived efficacy. Perceived efficacy is composed of two dimensions, *self-efficacy* (one's beliefs about his or her ability to perform the recommended response) and *response efficacy* (one's beliefs about whether the recommended response is effective in averting the threat) (Witte & Allen). Thus, not only is the adoption of a particular health behaviour affected by an individual's perceived threat, but also by the extent to which that person believes he or she can actually do the behaviour, and the extent to which he or she believes that behaviour will decrease the perceived threat.

Of the two dimensions of perceived efficacy, self-efficacy has received more attention within the general psychological literature. Self-efficacy theory (Bandura, 1977) maintains that all processes of psychological and behavioural change operate through the alteration of an individual's sense of personal mastery or self-efficacy. Within the domain of health psychology, beliefs about personal control or efficacy are featured prominently in each of the major models of health-related behaviour change, including the health belief model (Rosenstock, 1974), protection motivation theory (Maddux & Rogers, 1983; Rogers, 1975), and the theory of planned behaviour (Ajzen & Madden, 1996). Response efficacy is also a feature of such models (Maddux & Rogers; Rogers; Rosenstock).

Perceived efficacy is demonstrated to be an important variable in determining whether people practice a particular health behaviour (e.g., Hochbaum, 1958; Murphy, Stein, Schlenger, & Maibach, 2001; Rosenstock, 1966): as self-efficacy and response efficacy increase, so does the probability of engaging in the recommended preventative behaviour (Maddux, Brawley, & Boykin, 1995). Moreover, self-efficacy has been shown to enhance both the adoption and maintenance of a variety of health-related behaviours

(e.g., DiClemente, Fairhurst, & Piotrowski, 1995; Goldman & Harlow, 1993; Maddux et al.; Solomon & Annis, 1990; Young, Connor, Ricciardelli, & Saunders, 2006). Response efficacy has also been shown to be related to the adoption of positive health behaviours (e.g., Straughan & Seow, 2000; Zak-Place & Stern, 2004). Thus, perceived efficacy is a well-established predictor of health-related behaviour. However, most studies examining perceived efficacy have only looked at its relationship with the performance of or the intended performance of these health-related behaviours (e.g., Bishop, Marteau, Hall, Kitchener, & Hajek, 2005; Goldman & Harlow, 1993; McMath & Prentice-Dunn, 2005; Prentice-Dunn, Floyd, & Flournoy, 2001). The impact of this variable may be broader; in addition to affecting health behaviours, perceived efficacy may also affect the *acceptance* of health risk information. To date however, this relationship has not been thoroughly explored.

Perceived Efficacy and Risk Perception

A number of studies have examined the relationship between perceived efficacy and risk perception, the majority of which are correlational in nature. Most of the studies examining the perceived efficacy-risk perception relationship focus on unrealistic optimism, and examine the relationship between comparative risk judgments and perceived efficacy. The findings of this research generally show a negative relationship between the two variables, that is, as perceived controllability of a given risk increases, risk perception decreases.

This relationship has been shown for both unrealistic optimism and comparative optimism, and for a diverse set of negative events including motor vehicle accidents, food-related hazards, and illnesses. Moreover, the relationship has been demonstrated

using ratings of both personal (e.g., event-specific) and general (e.g., health locus) perceived control. For example, Weinstein (1980; 1982; 1987) compared ratings of perceived behavioural control of a number of both health-related and non-health-related negative events, and demonstrated that in general, comparative risk perceptions were lower in participants who felt that the likelihood of an event occurring was within their control. DeJoy (1989) found a similar relationship in his study of unrealistic optimism in traffic accidents. Harris (1996) reviewed the literature on the relationship between optimism and perceived efficacy, and concluded that people are optimistically biased about negative events they perceive to be controllable. Using a more general and non-specific measure of perceived control, Hoorens and Buunk (1993) demonstrated the same relationship in their study of unrealistic optimism in various health problems.

One interpretation of the relationship between perceived efficacy and perceived risk has been proposed by Perloff (1987): if people believe that they possess important skills and abilities to a higher degree than others, then they will also believe that their efforts to reduce their health risks will be more effective than other people's. To the degree that health risks are perceived as controllable, this belief must lead to unrealistic optimism. A second interpretation is that individuals who believe that they are in control of their own health show more health-protective behaviours than people who believe that things are controlled by natural forces or powerful others (e.g., Hoorens & Buunk, 1993; Seeman & Seeman, 1983; Stickland, 1978).

Given the aforementioned literature, there is a clear relationship between risk perception and perceived control: higher perceptions of controllability are associated with lower perceptions of risk. This finding is interesting, and in considering the issues with

risk acceptance and behaviour change raised previously, it may have significant implications. This relationship may be causal, such that increases in controllability lead to decreases in risk perception. If this were true, this relationship would perhaps be troubling, as it would suggest that attempts at increasing perceived control (as often seen in public health campaigns) actually create negative (and opposite than intended) effects on perceptions of risk. In attempts to increase healthy behaviour, many health interventions and public health campaigns target an individual's perception of their ability to control their health risk. And while this may act to increase the likelihood of behaviour change, if there is a causal relationship between efficacy and risk, these attempts to increase perceived efficacy could also *decrease* an individual's perception of risk.

The negative relationship between risk perception and perceived efficacy is consistent, and has been shown across a variety of negative events, with both specific and general indices of control. However, this relationship has only been demonstrated in correlational studies: I could find no studies that examined the effect of experimentally manipulated perceived efficacy on risk perception. Thus, one cannot make any inferences regarding a causal relationship between perceived efficacy and risk perception. This places a caveat on these findings, as one could interpret the relationship in several causal ways. First, one could interpret the relationship as implying that changes in perceived efficacy will affect an individual's perception of risk. That is, if perceived efficacy increases, their risk perception will subsequently decrease. This interpretation is not illogical; it could be the case that when an individual feels in control of a negative event, they see themselves as being at a decreased risk because they have a sense that they can

control their risk at any time. If this is the case, then attempts at increasing one's perceived efficacy (as one often sees in public health campaigns) would produce opposite effects than would be intended (i.e., decreased perceptions of risk).

A second causal interpretation of this relationship is the reverse; one could interpret the relationship as implying that changes in risk perception will affect an individual's perceived efficacy. That is, if risk perception increases, their perceived efficacy will subsequently decrease. If this is the case, then attempts at increasing perceived efficacy would have no effects on perception of risk.

Finally, a third interpretation of the relationship between risk and perceived efficacy is that there is no causal relationship between the variables. Instead, there may be a third, unmeasured variable (e.g., a personality trait) that influences both perceived efficacy and risk perception. If this were the case, it would mean that perceived efficacy and perceived risk do not influence one another, and any relationship that exists between perceived efficacy and risk is due to the influence of this third variable. If this were true, changes in perceived efficacy would have no effect on risk perceptions, and vice versa.

Given the three potential causal paths accounting for the association between risk perception and perceived efficacy, manipulations of efficacy could have very different effects on risk perception. If the first interpretation is true, then making individuals feel more efficacious will be problematic because it results in decreased risk perception. If the second or third interpretations are true, then making individuals feel more efficacious will not be problematic because the manipulation should not affect risk perception. However, one must also consider the possibility that attempts to increase perceived efficacy may

have additional effects. That is, manipulations designed to increase perceptions of control over a given health risk could have direct effects on other related variables.

Self-Affirmation and Perceived Efficacy

Taken together, the literature examining the effects of self-affirmation and perceived efficacy has demonstrated different results for each variable. The use of self-affirmation is demonstrated to produce significant effects in increasing message acceptance and risk perception. But, it is not clear whether changes in risk perceptions will result in changes in intentions and behaviours: the use of self-affirmation has produced negligible and inconsistent results in producing changes in behavioural intentions and changes in behaviours. Conversely, perceived efficacy has not been demonstrated to produce significant effects in increasing message acceptance and risk perception. In fact, a negative relationship between perceived control and risk perception has been shown, but this research is only correlational. Therefore, the causal effect of perceived efficacy on risk perception is not clear. However, the use of perceived efficacy is demonstrated to produce significant effects in increasing behavioural intentions and behavioural change to reduce risk.

Given these findings, it appears that self-affirmation and perceived efficacy have contrasting effects on risk perceptions and health promoting behaviours. Each of these variables (and their effects on risk and behaviour) has largely been studied in isolation. But in the real world, complex systems of variables exist that operate simultaneously. It would be reasonable to assume that self-affirmation and perceived efficacy might work in combination with one another. Given their differing effects on risk and health promoting behaviour, these variables should be examined in conjunction with one another, to

examine how they interact to affect risk perception, and how this subsequently translates to changes in risk-reducing behavioural intentions and behaviours.

If one considers perceived efficacy in conjunction with self-affirmation, a number of possible effects could occur, including both main effects and interaction effects. First, one would expect an effect of self-affirmation on risk perception. Although its effect has been examined in only a handful of health-risk studies, self-affirmation has been demonstrated to increase risk perception. However, the effect of self-affirmation on intentions and behaviours is less clear, with inconsistent effects on intentions and behaviours across studies. Thus, it is difficult to predict what the effect of the manipulation would be on intentions and behaviour.

Second, one would expect an effect of perceived efficacy on intentions and behaviours. Again, the effect of increasing perceived efficacy is demonstrated to increase behavioural intentions and behavioural change in numerous experimental studies. The effect of perceived efficacy on perceived risk however, is unclear. Correlational research indicates that increased mastery is associated with decreased perceptions of risk. If this is a causal path, then one would predict that manipulations that increase perceived efficacy would cause individuals to view themselves as being less at risk. However, if it is a reverse causal pathway, or, if there is a third, unmeasured variable accounting for the relationship between the variables, one would expect that perceived efficacy would have no effect on risk perception.

One might also expect interaction effects between self affirmation and perceived efficacy. First, it may be that perceived efficacy is the “missing piece” that acts, in conjunction with self-affirmation, to turn acceptance into action. If this is so, the

manipulation of an individual's perceived efficacy in conjunction with self-affirmation may produce greater acceptance of risk and greater behavioural change, with increased perceived efficacy perhaps increasing (or creating) the drive to turn risk acceptance into action. On its own, self-affirmation may act to increase risk acceptance, but if individuals do not have a sense of mastery or efficacy, the acceptance may not translate into behavioural change. For example, Harris and Napper (2005) note that even though their self-affirmation manipulation resulted in greater measure acceptance, the fact that it was not able to produce behaviour change may have been because participants were not provided with strategies to reduce the health risk. Thus, under conditions of increased perceived efficacy, self-affirmed individuals might be more accepting of their risk, and with this increased mastery they may show increased behavioral intentions and increased behavioural change to reduce risk.

On the other hand, the inability of self-affirmation to produce behaviour changes may not be due to a missing piece hypothesis. Instead, changes in behaviour may not occur because of motivational factors. Self-affirmation is demonstrated to reduce defensive processing, and to increase individuals' ability to tolerate negative information. It could be that self-affirmation causes individuals to become more comfortable with being at risk of a negative event, and are thus not motivated to engage in behaviours to reduce their risk. The manipulation of perceived efficacy may or may not act to increase motivation to engage in risk-reducing behaviours. It could be that increasing self and response efficacy incites the motivation sapped by self-affirmation. If it does, then the combination of self-affirmation and increased perceived efficacy would increase both risk perception and behavioural change. On the other hand, if perceived efficacy does not

have such motivational effects, then the decrease in motivation created by self-affirmation will not be reversed, and manipulating perceived efficacy in combination with self affirmation would only increase perception of risk.

Overview of Current Research

Broadly, the overarching goal of this program of research was to examine the effects of manipulating self-affirmation and perceived efficacy on several dependent variables: perceptions of efficacy, perceived risk, and risk-reducing behavioural intentions. However, within this research paradigm, there were a number of methodological and conceptual goals.

Methodologically, I wanted to experimentally manipulate both self-affirmation and perceived efficacy. In health psychology, previous studies have examined the effect of manipulating self-affirmation, however, very few have manipulated perceived efficacy, and those that have, have not examined its effects on risk perception. In addition to these manipulations, my research sought to determine whether risk-reducing behavioural intentions were multidimensional, or whether there were one-dimensional, as previous studies have treated them. Thus, my study included a comprehensive measure of risk-reducing intentions that assessed several different types of health promoting behaviour.

Conceptually, I had number of goals. First, I sought to further explore the potential behavioural effects of self-affirmation, as only a handful of studies have examined the role of self-affirmation in a health-risk context. A second goal was to explore the use of perceived efficacy in both increasing perception of risk and in the adoption of positive behaviours to decrease that risk (i.e., risk-reducing behavioural

intentions). Third, I set out to examine the effects of self-affirmation and perceived efficacy in conjunction with one another, which has never been examined in the context of risk acceptance and subsequent behavioural change. I also wondered whether any effects of self-affirmation or manipulated perceived efficacy on behavioural intentions were general across a wide range of risk-reducing behavioural intentions, or whether only certain domains were affected.

Finally, I had one additional conceptual goal. My program of research examined the effects of two experimental manipulations – self-affirmation and perceived efficacy – on several dependent variables: perceptions of efficacy, risk perception, and risk-reducing behavioural intentions. In isolation, these variables are generally well-studied, and the results of these studies have provided important information regarding risk perception and risk-reducing behaviour. However, rarely have these studies considered the effects of these independent variables together, and rarely have these dependent variables all been analyzed in a single study, including how they might influence and affect one another. For example, as highlighted previously, perceived efficacy has been shown to have positive effects on intentions and behaviours, but a negative correlation with risk. I wondered whether the manipulation of perceived efficacy could have differing effects on my dependent variables, and whether manipulating self-affirmation could have differing effects as well. Thus, the final conceptual goal of my study was to examine the dynamics among the dependent variables, and the effects of the manipulated variables on these dependent variables.

Chapter 2: Experiment 1

Method

Participants

Participants were 273 undergraduate students (100 men, 173 women). All participants were between the ages of 17 and 24, with a mean age of 18.28 years (*SD* = 0.83). Because the nature of my study involved future health outcomes (i.e., risk of heart attack prior to the age of 50) one participant aged 43 was removed from the data set. Participants received either course credit or $5 for their participation.

Experimental Design and Procedure

All participants received the same order of manipulations and dependent measures: the self-affirmation manipulation, followed by the perceived efficacy manipulation, followed by negative health risk information, and finally the dependent measures[1]. Participants were randomly assigned to both a self-affirmation manipulation and a perceived efficacy manipulation using a computer program that generated random sequences of numbers. The self-affirmation manipulation had two levels: no self-affirmation and self-affirmation. The perceived efficacy manipulation had three levels: high manipulated perceived efficacy, low manipulated perceived efficacy, and baseline manipulated perceived efficacy. Thus, the study consisted of a 2 x 3 between-subjects factorial design. The study was completed on a computer using MediaLab (2008).

[1] I conducted pilot testing where the order of the self-affirmation and perceived efficacy manipulations were counterbalanced, and found no differences between the two orders of manipulation.

At the outset of the study, participants were told that the purpose of the study was ostensibly to determine the predictors of heart attack and to validate a risk assessment questionnaire. They were shown the following message:

> Our lab has been measuring university-aged students on certain behavioural characteristics and then following them over time to track who develops heart disease and/or suffers a heart attack. By using ordinary least-squares multiple regression we've developed a mathematical formula that can predict whether or not an individual is at an increased risk of having a heart attack, and how high an individual's risk is, based on these behavioural characteristics. Using these results, we have developed a risk assessment questionnaire that can estimate an individual's risk of having a heart attack prior to the age of 50. Today, you are going to be taking part in a study further validating this risk assessment questionnaire.

Following the study introduction, participants were told that prior to the main risk assessment study, they would be asked to complete two brief studies for other researchers associated with the lab. These studies were actually the two experimental manipulations: participants were told that the self-affirmation manipulation was an attitudes-related study examining values, and that the perceived efficacy manipulation was a memory study related to the lab's research examining variables associated with heart attacks.

Self-affirmation manipulation. I used a self-affirmation manipulation commonly used by self-affirmation researchers (e.g., Harris & Napper, 2005; Sherman et al., 2000), adapted from Spencer and Fein (1997; see Appendix A for Experiment 1 materials). In both the self-affirmation and the no self-affirmation conditions, all participants were

given a list of nine values, including *business*, *economics*, *art*, *music*, *theatre*, *social life*, *relationships*, *science*, and *pursuit of knowledge*. Participants in the self-affirmation condition were asked to choose the value that was *most* important to them personally, and to write an essay describing why the value was important to them and how they used it in everyday life. Participants in the no self-affirmation condition were asked to choose the value that was *least* important to them personally, and to write and an essay describing why the value might be important to someone else.

Perceived efficacy manipulation. The perceived efficacy manipulation had three levels, high manipulated perceived efficacy, low manipulated perceived efficacy, and baseline manipulated perceived efficacy. Participants in the *high manipulated efficacy* condition read a passage about heart attack risk emphasizing that the prevention of a heart attack is easier than one might think. To increase response efficacy, the passage emphasized that behaviour and lifestyle were significant predictors of heart attack risk, with diet, physical activity, smoking status, and weight being the most important factors in the prevention of a heart attack. Participants in this condition were also told that background factors such as genetics and familial history of heart disease were only minor predictors of heart attack. To increase self-efficacy, the passage emphasized that heart attack prevention was well within participants' control, and that they could substantially reduce their risk with simple and effective behavioural changes.

Participants in the *low manipulated efficacy* condition read a passage about heart attack risk emphasizing that the prevention of a heart attack is difficult. To decrease response efficacy, the passage emphasized that behaviour and lifestyle were not as important as (uncontrollable) factors such as genetics and familial history of heart

disease. This passage described research showing that genetics and uncontrollable situational factors such as stress were the most important predictors of heart attack risk. To decrease self-efficacy, participants were told that individuals may not have as much control over their risk of having a heart attack as they have been led to believe.

Participants in the *baseline manipulated efficacy* condition served as a control group for the perceived efficacy manipulation. Participants read a paragraph about the heart, which included information about the structure of the heart and the history of its discovery. This paragraph was designed to be innocuous, with no reference to heart attacks or heart attack risk.

Following each of the perceived efficacy manipulations, all participants completed multiple choice questions testing their memory for the paragraph they had just read. Within these filler questions was a manipulation check designed to ensure that perceptions of perceived efficacy were properly manipulated.

Negative Risk Feedback. Following the two experimental manipulations, the next phase of the study was to expose all participants to negative health risk information. Participants were given comparatively extreme risk feedback in order to gauge the level at which they accepted this information. All participants were told that they were at the same, elevated risk for heart attack. This exposure was accomplished through the use of negative risk feedback from an ostensible computerized risk assessment questionnaire (RAQ).

The RAQ comprised 31 questions regarding participants' health behaviours such as their diet, physical activity, and knowledge of their heart health (e.g., blood pressure). Questions also assessed participants' familial history of heart disease. When possible,

questions were worded in such as manner such that most participants would give responses that could be interpreted as "risky" (e.g., "Do you eat fast food at least once per month?"; "In a given month, how many days do you miss planned exercise?"; "Do you eat the recommended 5-10 servings of fruits and vegetables every day?"). After completing the RAQ items, participants were shown the following message: "By clicking below you will submit your responses and our computer algorithm will compute a probability for the likelihood of a heart attack prior to the age of 50." Once participants "submitted" their responses to the computer, there was a delay of 10 seconds, at which point participants were given the negative risk feedback. While they waited, the following message appeared on the computer screen: "Please be patient. The calculation of your risk may take a few seconds". This delay was designed to increase the realism of the RAQ and the feedback.

Participants were given risk feedback regarding their risk of a heart attack prior to the age of 50. However, risk feedback was *not* based on participants' responses to the RAQ questions; instead, all participants received the same, high-risk feedback. Each participant was told that they were likely to have a heart attack prior to the age of 50, and that the percentage likelihood of this occurring was 35%. The percentage likelihood value was determined through pretesting; 30 undergraduate students were asked to estimate their likelihood of having a heart attack prior to the age of 50, and I chose a value that was extreme (i.e., double) relative to this mean.

After participants were given their risk feedback, they completed the dependent measures: risk perception, risk reducing behavioural intentions, and risk-reducing behaviors. To introduce the dependent measures, participants were told that in addition to

finding predictors of heart attacks, the lab was also interested in individuals' own expectations regarding their likelihood of having a heart attack, as well as their plans over the next year.

Measures

Perceived Efficacy Manipulation Check: This manipulation check consisted of four questions. Two questions (items 2 and 4) assessed participants' general perceptions of control over their risk of a heart attack (see Appendix B), and two questions (items 1 and 3) assessed more specific aspects of control, including the effects of background factors and situational factors on risk. Responses to each of the four items were made on a 7-point scale, and items 1 and 3 were reverse-coded, such that higher scores indicated higher perceptions of control on all items. Reliability analyses conducted on the four items showed poor reliability (alpha = .58). Item-total statistics indicated that reliability would be improved if items 1 and 3 were removed. Based on these results the two items were eliminated, and the final scale comprised items 2 and 4 and had adequate reliability (alpha = .77). Responses to the two items were summed to produce the measured perceived efficacy scale, with possible scores ranging from 2 to 14.

Self Risk Perception. Participants' estimates of their own risk were assessed using three questions: "Prior to the age of 50, what is the likelihood that you will have a heart attack?"; "Prior to the age of 50, what is the likelihood that you will have heart disease?"; and "Prior to the age of 50, what is the likelihood that you will have high blood pressure?". Responses were given on an 11-point Likert scale ranging from 1 (*extremely unlikely*) to 11 (*extremely likely*). Responses to the three questions were totaled to produce a self risk score, and this score could range from 3 to 33. Higher scores indicated

higher perceptions of risk. Reliability analyses showed the self risk items to have excellent reliability (alpha = .90).

Others Risk Perception. Participants were also asked to estimate the average Queen's student's risk using three questions similar to the self risk perception items: "Prior to the age of 50, what is the likelihood that the average Queen's University student of your age and sex will have a heart attack?"; "Prior to the age of 50, what is the likelihood that the average Queen's University student of your age and sex will have heart disease?"; and "Prior to the age of 50, what is the likelihood that the average Queen's University student of your age and sex will have high blood pressure?". Responses were given on an 11-point Likert scale ranging from 1 (*extremely unlikely*) to 11 (*extremely likely*). Responses to the three questions were totaled to produce an others risk score, and this score could range from 3 to 33. Reliability analyses showed the others risk items to have excellent reliability (alpha = .94).

Risk-Reducing Behavioural Intentions. Behavioural intentions to reduce the risk of heart attack were assessed using responses to 19 items (see Appendix B). Behavioural intentions included questions related to diet (e.g., "Over the next year I intend to eat the recommended 5-10 servings of fruit and vegetables per day"), physical activity (e.g., "Over the next year I intend to maintain a regular schedule of physical activity"), and heart function behaviour (e.g., "Over the next year I intend to get my blood pressure checked at least once"). Responses were made on an 11-point Likert scale ranging from 1 (*strongly disagree*) to 11 (*strongly agree*).

Risk-Reducing Behaviours. Participants were given information regarding opportunities to ostensibly participate in three programs designed to reduce the risk of

having a heart attack prior to the age of 50 (see Appendix B for full descriptions). These programs were fictitious, and to avoid ceiling effects, each of the three programs was designed to be intensive and involve a significant time commitment. The first program was nutritional counseling for a healthy heart, which involved meetings with a counselor and the completion of a food diary. The second program offered was a heart function and heart history assessment, which involved an examination of participants' current heart health and an assessment of their familial heart disease. The third program offered was a workshop designed to provide participants with information regarding the prevention of a heart attack. Participants indicated their decision to participate in each program by selecting 1 *(Yes! Please sign me up!*) or 0 (*No, I am not interested.*)[2]. See Appendix C for the correlation matrix for the dependent variables.

Following the completion of the dependent measures, all participants were thoroughly debriefed and informed of the true nature of the study (See Appendix D for information sheet, consent form, and debriefing materials). Participants were given both an oral debriefing, as well as a written debriefing sheet. To dispel any concerns participants may have had about their risk of heart attack, they were told that the risk feedback they received was fabricated and that all participants in the study received the same high-risk feedback. In addition, to address any misconceptions about heart attack risk presented in the study, all participants received information regarding heart-healthy behaviours from the Heart and Stroke Foundation of Canada.

[2] The rates of sign-up for the three behaviour items were very low across all participants: 34 of the 273 participants (12.5%) signed up for nutritional counseling, 27 (9.9%) signed up for a heart assessment, and 12 (4.4%) signed up for the workshop. Such low rates made it difficult to perform any meaningful analyses of the behaviours items and I found no significant effects. I included these items in two subsequent studies, and similar issues arose. Thus, I chose not to include analyses for the behaviour items.

Results

Factor Structure of Behavioural Intentions Measure

As noted above, many researchers have treated risk-reducing behavioural intentions as a single factor, by either using only one item to measure behavioural intentions (thereby implicitly assuming one-dimensionality), or, by using several items and aggregating them into one total score. However, for any given health risk, one could argue that there are a number risk-reducing behavioural intentions that are associated with it, and assuming that a single factor underlies behavioural intentions is somewhat questionable. If in fact there are several factors underlying risk-reducing behavioural intentions for a given health risk, interventions designed to increase behavioural intentions (and subsequent behaviours) may affect each factor in different ways. My measure of behavioural intentions comprised multiple items, thus, in order to determine whether the items were measuring a single factor or multiple factors, I first conducted a factor analysis of the 19 behavioural intentions items.

Factor Analysis Method

Exploratory factor analytic (EFA) methods were used to identify the dimensions underlying the 19 behavioural intention items. Maximum likelihood was chosen to fit the common factor model to the data, primarily because it allows for computation of a wide range of indexes of the goodness of fit model. Solutions were rotated using an oblique rotation (direct oblimin). This type of rotation was selected because oblique rotations allow for correlations among factors, although they do not require that the factors be correlated.

To determine the appropriate number of factors for the measures, four procedures recommended by Fabrigar, Wegener, MacCallum, and Strahan (1999) and Fabrigar and Wegener (in press) were used in conjunction with one another. These factor-number procedures included examination of the scree plot of eigenvalues from the reduced correlation matrix, parallel analysis, the model fit procedure using Root Mean Square Error of Approximation (RMSEA) as the fit index, and interpretability of the solution.

Scree plot. With scree plot analysis, the eigenvalues of the reduced correlation matrix are computed and then plotted in descending order of the values. Each eigenvalue corresponds to the variance accounted for by a common factor. Thus, the appropriate number of factors corresponds to the last occasion where a substantial drop occurs in the magnitude of the eigenvalues.

The scree plot of eigenvalues from the reduced correlation matrix for the behavioural intentions items is presented in Figure 1. With this plot, the substantial drops

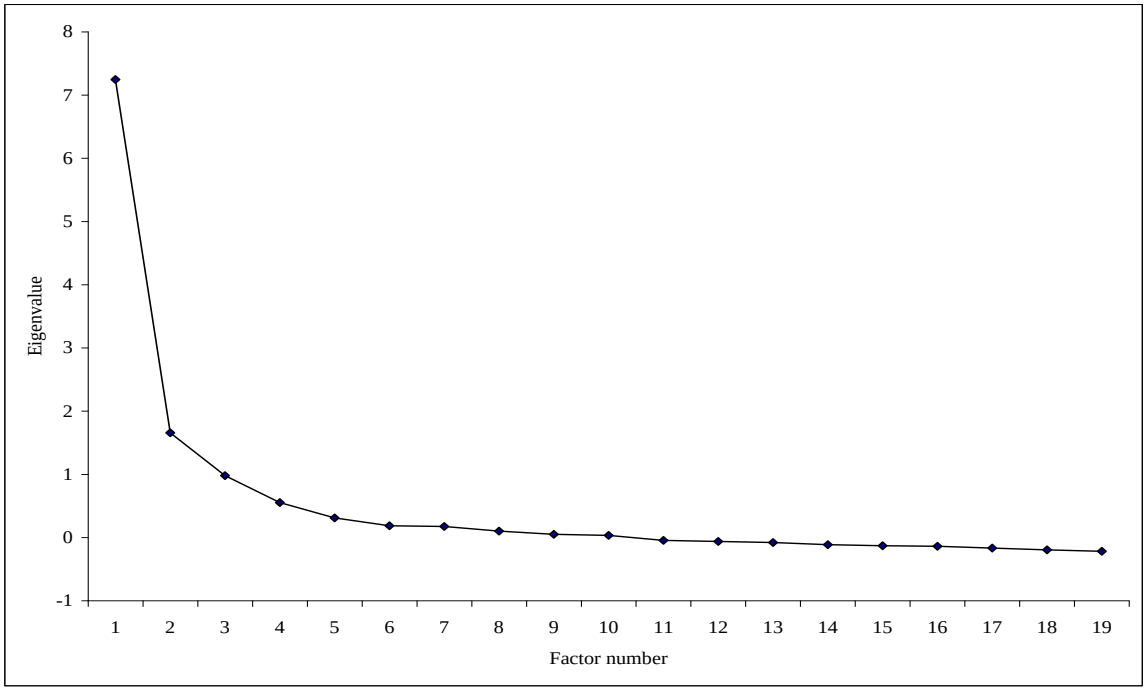


Figure 1. Experiment 1 behavioural intentions scree plot of eigenvalues.

in the curve following the first, second, and third eigenvalues provide support for a three-factor solution. There are also noticeable dips following the fourth and fifth eigenvalues, suggesting that a four- and five-factor solution is plausible. Thus, this scree plot supports a model with a minimum of three factors, with some support for four and five factors.

Parallel analysis. The second factor-number procedure used was parallel analysis. Parallel analysis involves the comparison of eigenvalues from the reduced correlation matrix to eigenvalues one would expect to obtain from completely random data (i.e., the predicted means of eigenvalues produced by repeated sets of random data; Humphreys & Montanelli, 1975; Montanelli & Humphreys, 1976). This procedure is based on the notion that sample eigenvalues greater than corresponding random data eigenvalues represent non-random effects (i.e., latent factors). The number of factors in the model is determined by the number of eigenvalues that are greater than the eigenvalues predicted from random data.

Two sets of expected values are generated in parallel analysis. The first represents the mean eigenvalue predicted from sets of random data. Observed values are compared to the mean, and any value greater than the mean is considered to be a non-random effect. However, this criterion of using the mean eigenvalue may be too lenient. Though this criterion may well identify non-random effects, the effects may be trivial and using the results could lead to overfactoring. Because of this issue, eigenvalues that fell at the 95th percentile were also used. These upper-bound values represent a stricter criterion, and may not be as prone to overfactoring as simply using the mean eigenvalue.

The observed eigenvalues, the mean eigenvalues, and the eigenvalues that fell at the 95th percentile corresponding to the behavioural intentions item factor analysis are

presented in Table 1. The observed eigenvalues were compared to both the mean eigenvalues and the eigenvalues that fell at the 95th percentile. For the mean eigenvalues, the observed eigenvalue was greater than the mean value until the six-factor solution.

Table 1

Experiment 1 Behavioural Intentions Observed Eigenvalues, Mean Eigenvalues, and 95th Percentile Eigenvalues

Factor	Observed	Mean	95th Percentile
1	7.246	0.573	0.661
2	1.656	0.473	0.554
3	0.979	0.397	0.477
4	0.552	0.335	0.393
5	0.310	0.272	0.322
6	0.187	0.221	0.267
7	0.175	0.169	0.212
8	0.102	0.123	0.175
9	0.051	0.084	0.133
10	0.032	0.043	0.074
11	-0.047	0.001	0.036
12	-0.062	-0.037	-0.007
13	-0.080	-0.079	-0.038
14	-0.113	-0.117	-0.086
15	-0.131	-0.158	-0.124
16	-0.139	-0.197	-0.162

17	-0.167	-0.239	-0.206
18	-0.196	-0.279	-0.245
19	-0.219	-0.332	-0.296

Thus, using the mean values, a maximum of five factors is supported. For the 95th percentile eigenvalues (a more conservative criterion), the observed eigenvalue was greater for every factor until the five-factor solution, and a maximum of four factors was supported.

Model fit The third factor-number procedure used was the examination of model fit. RMSEA was used as an index of model fit. RMSEA is an estimate of the discrepancy between the model and the data per degree of freedom for the model. RMSEA values of 0.050 or less indicate good model fit, those between .051 to .080 indicate acceptable model fit, those between.081 to.100 indicate marginal model fit, and an RMSEA value greater than .100 indicates poor model fit (Browne & Cudeck, 1992; MacCallum, Browne, & Sugawara, 1996).

In the investigation of model fit, a series of models increasing in the number of factors is examined. Using RMSEA, the fit of the simplest model is examined first (one-factor), followed by the fit of a two-factor model, and so on. The optimum model is a model where having one fewer factor produces much poorer model fit, and having one additional factor does not increase model fit substantially. In addition, the magnitude of improvement in RMSEA between models was also considered. Though small changes in RMSEA from one model to another do indicate improvement in model fit, choosing a model based on relatively minor improvements in fit can result in overfactoring. The

ultimate goal is to arrive at the most parsimonious model that adequately accounts for the data.

With the behavioural intentions items RMSEA values and corresponding confidence intervals were compared for a series models, from one to six factors. RMSEA values and 90% confidence intervals are presented in Table 2.

The one-factor model demonstrated poor model fit. The two-factor model decreased in RMSEA by 0.037 and represented a substantial improvement, but the fit of the model remained in the poor range. The three-factor model decreased in RMSEA by 0.026 and represented a substantial improvement as well, and the fit of the model moved to the marginal range. The four-factor model improved model fit substantially as well, with a 0.022 decrease in RMSEA indicating acceptable model fit. The five-factor model decreased in RMSEA by 0.013 and represented a substantial improvement as well, and

Table 2

Experiment 1 RMSEA Values and Corresponding 90% Confidence Intervals for the Behavioural Intentions Items

Number of factors	RMSEA	90% CI
1-factor	0.145	0.137-0.154
2-factor	0.108	0.099-0.117
3-factor	0.082	0.072-0.093
4-factor	0.060	0.048-0.073
5-factor	0.053	0.039-0.067
6-factor	0.045	0.028-0.061

the fit of the model remained in the acceptable range. Finally, the six-factor model decreased RMSEA by .008 and represented minimal improvement, however, the fit of the model moved to the good range. Interestingly, even though model fit appeared to improve with increasing factors, the precision of the estimate (i.e., the confidence intervals) became progressively worse after the three-factor solution. Based on this model fit assessment, one could argue for a three- or four-factor solution for the behavioural intentions items.

Interpretability of solutions The final factor number procedure used was the interpretability of the model. When deciding on the appropriate number of factors, factor interpretability is an important criterion to consider. Even if all other criteria indicate a particular number of factors, if the model is not easily interpretable or if it is not conceptually sensible, it is of little value to the researcher. Thus, one must always consider relevant theory and previous research when determining the appropriate number of factors.

With the behavioural intentions items, the three factor-number procedures used supported a three, four-, or five-factor solution. To further investigate the utility of these models, their interpretability was examined. To identify the items that primarily loaded on each factor, a few general guidelines were used. Factor loadings that equaled or exceeded 0.40 were considered substantial loadings, and loadings between 0.20 and 0.39 were considered marginal loadings. Values less than 0.20 were considered low.

The rotated solution for the three-factor solution was examined first. Factor loadings and communalities are presented in Table 3. Factor loadings that are bolded represent substantial loadings. The first factor appeared to represent behavioural

intentions related to maintaining a healthy diet. Eight items had substantial loadings on this first factor, with low loadings on the second and third factors. The second factor

Table 3

Experiment 1Pattern Matrix of Rotated Three-Factor Solution of the Behavioural Intentions Items

Item	Pattern Coefficients			Communalities
	1	2	3	
Decrease fat	**0.82**	-0.02	0.002	0.67
Decrease saturated fat	**0.75**	0.14	-0.009	0.70
Eat 5-10 servings of fruits & vegetables	**0.63**	-0.03	0.23	0.62
Eat more salads	**0.60**	0.11	0.07	0.51
Decrease fast food	**0.58**	0.03	0.06	0.40
Decrease red meat	**0.57**	0.04	-0.12	0.33
Eat more plant-based protein	**0.41**	0.16	0.08	0.39
Alcohol in moderation	**0.40**	0.11	-0.10	0.23
Get cholesterol checked	0.001	**0.94**	0.04	0.66
Speak with physician	0.08	**0.59**	0.18	0.54
Have blood pressure checked	0.23	**0.59**	0.08	0.62
Go to the gym	-0.03	0.02	**0.86**	0.68
Weight training	-0.12	0.17	**0.77**	0.61
Engage in regular physical activity	0.14	-0.03	**0.77**	0.69
Engage in cardiovascular activity	0.06	0.05	**0.76**	0.64
Physical activity with friends	-0.03	0.09	**0.75**	0.60

Avoid smoking cigarettes	0.37	-0.08	0.04	0.24
Maintain a healthy diet	0.46	-0.12	0.46	0.59
Walk/bike/rollerblade when possible	0.35	0.03	0.32	0.42

appeared to represent behavioural intentions related to seeking medical advice and assessment. Three items loaded substantially loaded on this second factor. Finally, the third factor appeared to best represent intentions related to maintaining an active lifestyle, with five items loading substantially. One item loaded substantially and another loaded marginally on both Factors 1 and 2. Factors 1 and 2 were moderately correlated (r = .45); as were Factors 2 and 3 (r = .35); and Factors 1 and 3 (r = .45).

The rotated solutions for the four- and five-factor solutions were examined next. With the four-factor solution, there was one factor where only one item loaded substantially, and with the five-factor solution, there were two factors were only one item loaded substantially. Factors comprised of only one item can indicate over-factoring (Fabrigar et al., 1999; Fabrigar & Wegener, in press), thus, because of this issue I concluded that neither the four- or the five-factor solutions best represented the behavioural intentions item data.

Conclusion. Parallel analysis indicated that both four- and five-factor models were plausible solutions, while model fit suggested that three- and four-factor solutions were plausible. Scree plot examination suggested that three-, four-, and five-factor solutions were possible. However, when examining the interpretability of the three models, the four- and five-factor models fell short because the additional factors in each of these solutions comprised only one item. The three-factor model was readily

interpretable and made more sense conceptually. Moreover, only one of the nineteen items did not load substantially. Thus a three-factor solution, reflecting behavioural intentions related to maintaining an active lifestyle, seeking medical advice and assessment, and maintaining a healthy diet was concluded as best representing the behavioural intentions data.

The results of these factor analyses of the behavioural intentions items indicated that there were, in fact, multiple factors underlying the items. This finding is contrary to the manner in which risk-reducing behavioural intentions have been treated in previous studies (i.e., as comprising a single factor). My analyses revealed three distinct behavioural intentions factors, and this finding raised the possibility that the manipulation of self-affirmation and perceived efficacy could have differing effects on each of these factors.

Based on the results of the factor analyses, three behavioural intentions scales were created by summing the items: medical behavioural intentions (items 4, 11, and 18; scores could range from 3 to 33; See Appendix B for items and corresponding item numbers), activity behavioural intentions (items 5, 6, 7, 8, and 9; scores could range from 5 to 55), and dietary behavioural intentions (items 2, 3, 13, 15, and 17; scores could range from 5 to 55)[3]. Reliability analyses showed the activity behavioural intentions scale to have excellent reliability (alpha = .90); the medical behavioural intentions scale to have good reliability (alpha = .84); and the dietary behavioural intentions scale to have adequate reliability (alpha = .73). The medical and dietary behavioural intentions scales

[3] Even though three additional items loaded substantially on the dietary behavioural intentions factor, these items were not included in the final scale. A factor analysis of the behavioural intentions scale in Experiment 2 also supported a three-factor solution, but these three items did not load substantially. In order to maintain consistency between the two studies, only those items that loaded substantially in both studies were included in the final scales.

were moderately correlated (r = .51); as were the medical and activity scales (r = .49), and the dietary and activity scales (r = .45).

Univariate ANOVAs

Manipulation Check

Next, I conducted a manipulation check to ensure that the perceived efficacy manipulation was effective in altering perceptions of measured efficacy related to heart attack risk[4]. To examine these effects, a 2 (self-affirmation condition: self-affirmation versus no self-affirmation) X 3 (manipulated perceived efficacy: high versus low versus baseline) analysis of variance (ANOVA) was conducted. The measured perceived efficacy scale was the dependent variable. The ANOVA showed a significant main effect for manipulated efficacy, $F(2, 267) = 53.55$, $p < .001$. Significant differences among the three conditions were evaluated using the 95% confidence intervals. Participants in the high manipulated efficacy condition reported significantly higher ratings of measured efficacy (M = 12.20; CI = 11.76-12.65), compared to participants in the baseline manipulated efficacy condition (M = 10.86; CI = 10.40-11.32); and participants in the baseline manipulated efficacy condition (M = 8.86; CI = 8.41-9.32) reported significantly higher ratings of measured efficacy compared to those in the low manipulated efficacy condition. These significant findings were critical, as it suggested that the manipulation of perceived efficacy was successful in influencing participants' measured perceptions of perceived efficacy related to their heart attack risk.

As expected, the ANOVA indicated no significant main effect for self-affirmation $F(1, 267) = 0.03$, p = .86. I did not expect a significant interaction between manipulated

[4] A manipulation check for the self-affirmation manipulation was not conducted, as there is no established method of determining whether a participant has self-affirmed.

efficacy and self-affirmation, but this interaction was marginally significant $F(2, 267) = 2.40$, $p = .09$, and I felt that this warranted further investigation. The estimated marginal means for measured efficacy as a function of the two independent variables are presented in Table 4. As can be seen by the 95% confidence intervals, under no self-affirmation, the low perceived efficacy condition was significantly different from both the baseline and high manipulated efficacy conditions. Under self-affirmation, there were significant differences among all three of the manipulated efficacy conditions. These results suggest that the perceived efficacy manipulation was effective with both affirmed and non-affirmed participants; however, it appears that the effect was somewhat stronger in participants that self-affirmed.

Table 4

Experiment 1 Estimated Marginal Means and 95% Confidence Intervals for Measured Efficacy

Self-Affirmation	Manipulated Efficacy	Estimated Marginal Mean	95% Confidence Interval
No self-affirmation	Low	8.72	8.08-9.38
	Baseline	11.29	10.67-11.91
	High	11.98	11.34-12.62
Self-affirmation	Low	9.00	8.36-9.64
	Baseline	10.43	9.76-11.09
	High	12.43	11.81-13.05

Unrealistic Optimism

Before examining the effects of the independent variables on risk perception, I wanted to confirm that I did in fact, replicate the classic unrealistic optimism effect; that is, that individuals see themselves as less at risk than similar others. The unrealistic optimism effect for health problems has been clearly demonstrated in previous literature, which shows individuals consistently view themselves as being at less risk than similar others for numerous health (and non-health) risks. Thus, I expected a similar effect for heart attack risk in my study. To examine this relationship, I conducted a paired sample *t* test. As predicted, the results showed that individuals' perceptions of others risk (M = 19.58, SD = 5.50) were significantly greater than perceptions of self risk (M = 16.21, SD = 7.15), t (272) = 7.62, $p < .001$, thus replicating the unrealistic optimism effect.

Self Risk Perception

Having confirmed that the manipulation of perceived efficacy was effective in producing the intended effects, and that the classic unrealistic optimism effect was demonstrated, the next set of analyses tested the study's core hypotheses. The first analysis examined the effects of the independent variables on self risk perception. I conducted a 2 (self-affirmation condition: self-affirmation versus no self-affirmation) X 3 (manipulated perceived efficacy: high versus low versus baseline) ANOVA, with the self risk score as the dependent variable.

Based on the results of previous studies, I might have expected a main effect of self-affirmation on self risk perception, with self-affirmed participants indicating higher risk perceptions than non-affirmed participants. However, in the present study this effect did not emerge. The ANOVA showed no significant main effect for self-affirmation, F(1,

267) = 0.09, p = .76, indicating there were no differences in risk perception for participants who self-affirmed versus those who did not self-affirm.

I might have also expected a main effect of manipulated efficacy on self risk perception, given that correlational literature has suggested that higher perceived efficacy is associated with lower perceptions of health risk. In the present study, I found no significant main effect for manipulated efficacy, $F(2, 267) = 0.94$, p = .39, indicating there were no significant differences in self risk perception among participants in the low, baseline, and high manipulated efficacy conditions. The finding that the manipulation of perceived efficacy had virtually no impact on risk perception raises the possibility that the manipulation of perceived efficacy exerts no causal effect on individuals' perception of health risk.

Previous studies have not examined the effects of self-affirmation on risk perception in conjunction with manipulated perceived efficacy, thus, it was difficult to predict exactly how the two variables would combine to influence risk perception. The ANOVA showed a significant interaction between self-affirmation and manipulated efficacy $F(2, 267) = 3.75$, p = .03. The estimated marginal means and 95% confidence intervals for self risk perception as a function of the two independent variables are presented in Table 5.

There were several aspects of the interaction that warranted further comment. First, it is worthwhile to examine differences in risk perception for the baseline manipulated condition under self-affirmation and no self-affirmation. As mentioned earlier, previous self-affirmation studies have demonstrated that self-affirmation increases acceptance of health risk. Looking at the differences in self risk perception

Table 5

Experiment 1 Estimated Marginal Means and 95% Confidence Intervals for Self Risk Perception

Self-Affirmation	Manipulated Efficacy	Estimated Marginal Mean	95% Confidence Interval
No self-affirmation	Low	17.89	15.78-19.99
	Baseline	13.90	11.88-15.91
	High	17.36	15.28-19.44
Self-affirmation	Low	15.47	13.39-17.55
	Baseline	16.93	14.78-19.08
	High	15.96	13.97-17.95

between the two baseline manipulated efficacy conditions provided the most meaningful and "pure" examination of the self-affirmation effect, as perceived efficacy is not manipulated *per se* in these conditions. Examination of the 95% confidence intervals show a significant difference between the two conditions, such that baseline participants who self-affirmed provided significantly higher perceptions of risk perception relative to those baseline participants who did not self-affirm. This finding suggests that self-affirming did affect perception of risk in a way that is consistent with previous research.

Having examined the effects of self-affirmation on the baseline conditions, my next step was to consider the manipulated efficacy simple main effects. Once again, 95% confidence intervals were used as the basis for determining significant differences among

the means. I first examined differences in risk perception among the three manipulated efficacy conditions under no self-affirmation, which allowed me to investigate the effects of manipulated efficacy on risk perception, in the absence of self-affirmation. Thus, these analyses provided the most meaningful and "pure" examination of the effect of manipulated efficacy on risk. Again, given previous research showing a negative relationship between risk perception and perceived efficacy, I might have expected that participants in the low manipulated efficacy condition would show higher estimates of risk relative to participants in the baseline condition. This relationship was found to be true: when participants did not self-affirm, participants in the low manipulated efficacy condition provided significantly higher estimates of risk relative to participants in the baseline condition. Given this finding, one might be tempted to conclude that the negative relationship suggested by correlational research is correct. However, this finding was contradicted when I compared the high manipulated efficacy condition to the baseline condition. I found the same relationship: participants in the high condition also provided significantly higher estimates of risk relative to participants in the baseline condition. There were no significant differences in risk perception estimates for participants in the high manipulated efficacy condition versus the low manipulated efficacy condition. Thus, the results of these contrasts suggest that attempts at both increasing and decreasing perceived efficacy can increase perceptions of risk.

I next considered the effects of manipulated efficacy when participants had self-affirmed; this allowed me to examine my major hypothesis – the effects of combining self-affirmation and manipulated efficacy on estimates of perceived risk. Self-affirmation has been demonstrated to increase risk perception in experimental studies, while the

negative relationship between perceived efficacy and risk perception has only been demonstrated in correlational studies. One might have expected a number of different results. First, it could be that the effects of self-affirmation on risk perception are so strong that changes in perceived efficacy produce negligible effects. On the other hand, if there is a true causal relationship between perceived efficacy and risk perception, attempts at increasing perceived efficacy may cancel out any increase in risk perception that self-affirmation produces, while decreasing perceived efficacy may augment the effects of self-affirmation on risk perception. However, examination of the 95% confidence intervals indicates that there were no significant differences in risk perception among the three manipulated efficacy conditions.

Behavioural Intentions

The next set of analyses examined the effects of the independent variables on risk-reducing behavioural intentions. I conducted a series of three 2 (self-affirmation condition: self-affirmation versus no self-affirmation) X 3 (manipulated perceived efficacy: high versus low versus baseline) ANOVAs, with each of the three behavioural intentions scales (activity; medical, and dietary) as the dependent variable.

Previous studies have shown mixed results for the effects of self-affirmation on behavioural intentions, with some studies demonstrating that self-affirming can increase risk-reducing behavioural intentions, with other studies showing null effects. Thus, one might have predicted a main effect of self-affirmation on self risk perception, such that self-affirmed participants would show greater intentions to reduce their risk of a heart attack. However, in the present study this effect did not emerge. The ANOVA showed no significant self-affirmation main effect for activity behavioural intentions, $F(1, 267) =$

0.69, $p = .41$; medical behavioural intentions, $F(1, 267) = 0.92$, $p = .34$; or dietary behavioural intentions, $F(1, 267) = 0.09$, $p = .76$, indicating there were no differences in behavioural intentions for participants who self-affirmed versus those who did not.

The effect of perceived efficacy on behavioural intentions is well-demonstrated in the health literature, with numerous experimental studies showing that higher perceptions of control over a health risk are associated with greater intentions to reduce that risk. Thus, I did expect a main effect of manipulated efficacy for behavioural intentions. Given previous research, one would predict that individuals in the high manipulated efficacy condition would report the greatest intentions to reduce their risk, and that individuals in the low condition would report the lowest intentions to reduce their risk, with participants in the baseline condition falling somewhere in the middle. The estimated marginal means and 95% confidence intervals for each of the three behavioural intentions scales are presented in Table 6. The ANOVA did show a significant manipulated efficacy main

Table 6

Experiment 1 Estimated Marginal Means and 95% Confidence Intervals for Behavioural Intentions

	Activity Behavioural Intentions (95% CI)	Medical Behavioural Intentions (95%CI)	Dietary Behavioural Intentions (95%CI)
Low Manipulated Efficacy	40.99 (38.73-43.25)	20.52 (18.90-22.94)	40.13 (38.05-42.20)
Baseline Manipulated Efficacy	44.00 (41.76-46.26)	22.61 (21.00-24.22)	41.66 (39.56-43.73)
High Manipulated Efficacy	44.70 (42.50-46.90)	23.07 (21.50-24.65)	42.41 (40.38-44.43)

effect for activity behavioural intentions, $F(2, 267) = 3.00$, $p = .05$, indicating there were significant differences in activity intentions to reduce risk among the three manipulated efficacy conditions. This effect approached significance for the medical behavioural intentions, $F(2, 267) = 2.79$, $p = .06$, but was nonsignificant for dietary behavioural intentions, $F(2, 267) = 1.24$, $p = .29$.

Examination of the means and 95% confidence intervals for activity behavioural intentions showed that there was a significant difference in intentions between the high and low manipulated efficacy conditions, such that high manipulated efficacy participants indicated greater intentions to reduce their risk compared to low manipulated efficacy participants. There was also a significant difference between the low and baseline conditions, but not between the baseline and high conditions. Even though only one of the behavioural intentions scales showed a significant main effect for manipulated efficacy, one of the scales approached significance, and all of the means trended in the expected direction. For each of the scales, participants in the high manipulated efficacy condition showed the greatest intentions to reduce their risk, followed by participants in the baseline, and then low manipulated efficacy conditions.

My next step was to examine the interactive effects of self-affirmation and manipulated perceived efficacy on behavioural intentions. There were a number of possibilities. On one hand, manipulated efficacy might have acted as the "missing piece" that acted in conjunction with self-affirmation to turn acceptance into action. If this were true, one would expect the highest intentions to be reported by self-affirmed participants in the high manipulated efficacy condition. On the other hand, it may be that self-affirmation causes individuals to become more comfortable with being at risk of a

negative event, and are thus not motivated to engage in behaviours to reduce their risk. If this were true, one would expect to see the lowest intentions in self-affirmed participants, with non-self-affirmed participants showing differing intentions, with high manipulated efficacy participants showing the greatest, and low manipulated efficacy participants showing the lowest intentions. Finally, it may be that self-affirmation's effect on risk perception is unrelated to intentions to reduce risk. If this were true, one would expect no interaction, and predict that for both self-affirmed and non-self-affirmed participants, the highest intentions would be reported by the high manipulated efficacy participants, and the lowest intentions would be reported by the low manipulated efficacy participants (i.e., the same pattern of means shown in the perceived efficacy main effect. This last prediction was confirmed. Results of the ANOVA showed no significant interaction between self-affirmation and manipulated efficacy for activity behavioural intentions, $F(2, 267) = 0.26$, $p = .77$; medical behavioural intentions, $F(2, 267) = 0.87$, $p = .42$; or dietary behavioural intentions, $F(2, 267) = 0.47$, $p = .63$.

Structural Equation Modeling

Model Specification and Model Fitting

Up to this point, I have examined each of the dependent variables – measured efficacy, perceived risk, and risk-reducing behavioural intentions – in isolation of one another. However, given previous literature and theories of health behaviour one would expect these dependent variables to be related and interplay with one another. Thus, the final set of analyses sought to examine the dynamics among the dependent variables, and the effects of the manipulated variables on these dependent variables. To examine these relationships, I specified a structural equation model (SEM) of relationships, and this

proposed model is presented in Figure 2. It is important to note that I did examine this model under conditions of self-affirmation and no self-affirmation. However, there was no evidence of any differences between the two conditions, so I collapsed the models across self-affirmation.

I created two dummy coded variables to represent assignment to manipulated perceived efficacy condition. The first dummy coded variable, high versus baseline efficacy variable, represented high manipulated efficacy relative to baseline manipulated efficacy. The second dummy coded variable, low versus baseline efficacy variable, represented low manipulated efficacy relative to baseline manipulated efficacy.
In the structural model, given the fact that my manipulation of perceived efficacy was designed to affect participants' perceptions of perceived efficacy (i.e., measured efficacy), I specified the influence of the two dummy coded variables, the high efficacy dummy variable and the low efficacy dummy variable, on measured efficacy. Because the manipulation of perceived efficacy may have affected other dependent variables (perceived risk and the three behavioural intentions scales), I specified those paths as well. I specified the influence of measured efficacy and perceived risk, given the previous correlational research demonstrating a negative relationship between the two variables. Previous literature has also demonstrated a positive relationship between measured efficacy and behavioural intentions, and a positive relationship between perceived risk and behavioural intentions, thus, these paths were also specified.

Maximum likelihood was chosen as the model-fitting method, and the model was fit to a covariance matrix of the 20 measured variables. For each of the seven latent variables, the scale of measurement was set by specifying one of the measured variables

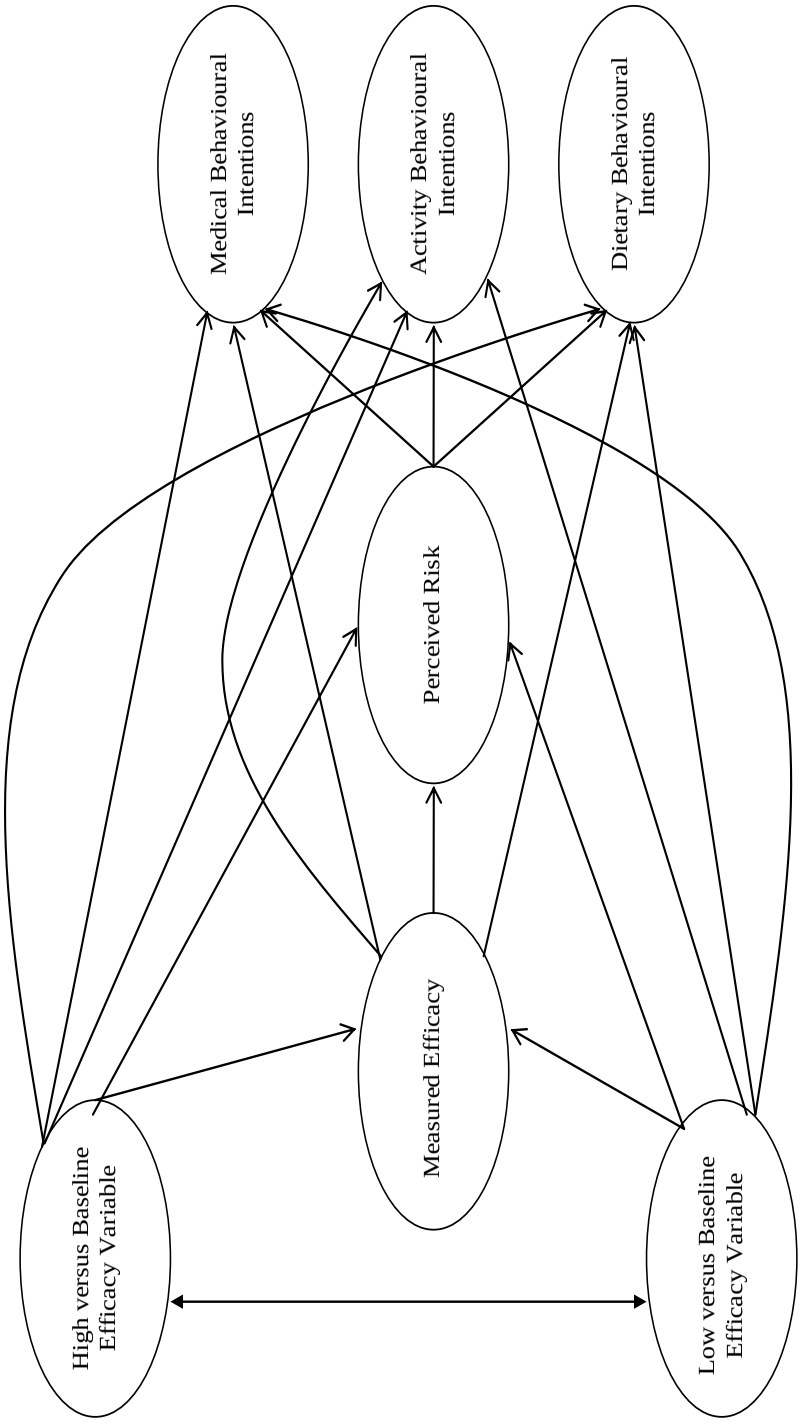


Figure 2. Proposed structural model

as a reference indicator by fixing its factor loading to one. I allowed the residuals of the three behavioural intentions scales to correlate with one another. The analysis was performed using LISREL 8.50 software (Joreskog & Sorbom, 2001).

Evaluation of Model

Model fit. Model fit was assessed using multiple goodness-of-fit indices, including RMSEA, Standardized Root Mean Square Residual (SRMR), and Non-Normed Fit Index (NNFI). These three indices were chosen because they have been demonstrated to function well in the detection misspecified models (Hu & Bentler, 1998). For completeness, the χ^2 goodness-of-fit test is also reported.

First, I considered the χ^2 test of model fit. For the proposed model, the test was significant, χ^2 (151, N = 273) = 264.78, $p < .001$, indicating that the model was not a perfect representation of the data. This test assumes that the model holds *perfectly* in the population, and it is an unrealistic criterion upon which to assess a model. Methodologists have criticized the use of the χ^2 goodness-of-fit test in assessing model fit (Hu & Bentler, 1998) and have recommended the use of alternative indices of model fit. However, I chose to report this index because it is customary to do so.

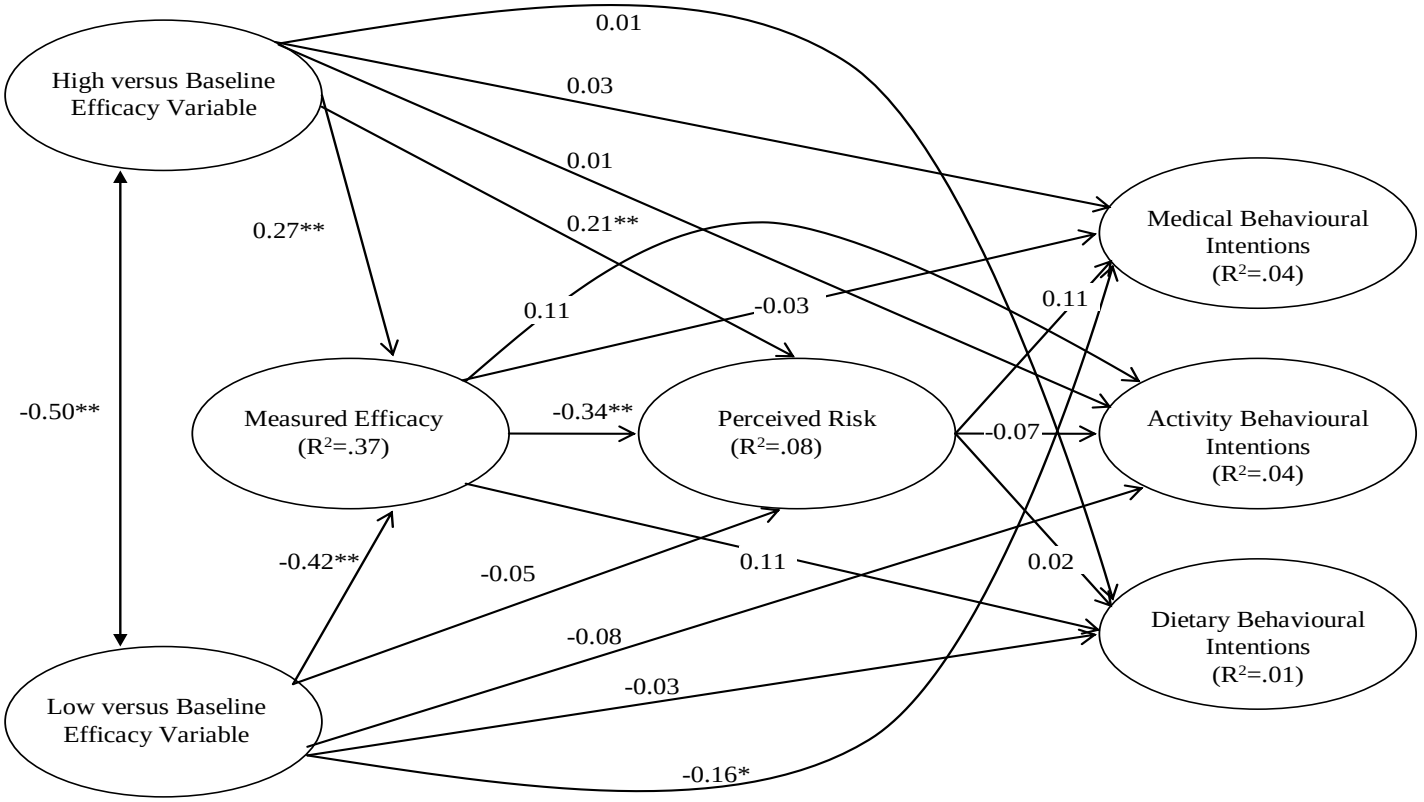
Next, I considered RMSEA. Recall that to interpret RMSEA, the following guidelines have been outlined: values of 0.050 or less indicate good model fit, .051 to .080 indicate acceptable model fit, .081 to .100 indicate marginal model fit, and a RMSEA greater than .100 indicates poor model fit (Browne & Cudeck, 1992). For the proposed model, RMSEA = 0.051 (90% CI = 0.040-0.061) indicating acceptable model fit. The third index examined was SRMR. In interpreting SRMR, values of 0.08 or lower are generally regarded as indicative of good fit. For the proposed model, SRMR = 0.045,

indicating good model fit. Finally, I considered NNFI (also referred to as the Tucker-Lewis Index (TLI)). In interpreting the NNFI, larger values reflect better fit. Traditionally, values of 0.90 have been considered to indicate good fit; however, more recent research suggests that values of 0.95 or higher may be a more appropriate guideline. For the proposed model, NNFI = 0.97, again indicating good model fit. Overall, these indices showed the model to have reasonably good model fit[5].

Path Estimates. The path estimates for the model are presented in Figure 3. All of the 20 measured variables loaded significantly (ps < .05) on their hypothesized latent variables. For the sake of simplicity, only the structural paths are presented in the figure.

First, these results show that assignment to manipulated efficacy condition had a significant effect on measured efficacy: paths for both the high efficacy dummy variable and the low efficacy dummy variable were significant. There was a strong positive relationship between the high efficacy dummy variable and measured efficacy, which indicates that participants in the high manipulated efficacy condition had higher measured efficacy relative to participants in the baseline condition. Conversely, there was a strong negative relationship between the low efficacy dummy variable and measured efficacy, which indicates that participants in the low manipulated efficacy condition had lower measured efficacy relative to participants in the baseline condition. These results support the earlier ANOVAs which indicated that my manipulation of perceived efficacy worked quite well in affecting measured efficacy.

[5] It is often customary to include an estimate of the fit of the overall measurement model, separate from the fit of the full model that includes both the measurement and structural models. Given that the indicators used in my SEM model were based on prior reliability analyses and factor analyses, I expected that the measurement model would show good fit. This was confirmed: when fit separately, the measurement showed good fit in Experiment 1 and in each of the two subsequent studies. However, for the sake of conciseness I have chosen not to report the results of these analyses of the measure models, but instead simply to report fit indices for the full model.



+ $p \leq .10$, * $p \leq .05$, ** $p \leq .01$

Figure 3. Experiment 1 path diagram

Next, measured efficacy significantly influenced perceptions of risk, and this relationship was again, in the expected direction. There appears to be a strong negative relationship between these two variables, such that higher measured efficacy caused lower perceptions of risk. This finding is consistent with previous correlational literature, which suggests that individuals who perceive themselves as more efficacious in their ability to control their risk have lower perceptions of risk. This finding suggests that my attempts at increasing perceived efficacy did in fact increase measured efficacy, but that as a result of this increased measured efficacy, participants felt less at risk.

Next, and perhaps most interestingly, the high efficacy dummy variable had a direct effect on perceived risk. There was a moderate and positive relationship between these two variables, which indicates that attempts at experimentally increasing efficacy caused increased perceptions of risk. This finding suggests the presence of a suppression effect. The manipulation to increase perceived efficacy caused increased perceptions of risk. But on the other hand, these attempts at increasing perceived efficacy also appeared to work against this effect of increasing risk, because as described above, increasing perceived efficacy caused increased measured efficacy, which in turn, led to *decreased* perceptions of risk. This finding raises an important issue, and suggests that attempts at increasing perceived efficacy could have both positive and negative effects on individuals' perceptions of risk.

The low efficacy dummy variable did not have a significant influence on perceived risk, which one might have expected given the direct relationship between high manipulated efficacy and perceived risk. The low efficacy dummy variable did, however, significantly affect medical behavioural intentions. Examination of the standardized

coefficient for this path shows a moderate negative relationship, which suggests that attempts at experimentally decreasing perceived efficacy caused decreased intentions to seek medical advice and assessment to reduce risk. This finding is in line with previous research, which shows participants who feel less in control of a given health risk are less likely to take steps to reduce that risk. Moreover, it also supports my previous supposition that interventions aimed at influencing risk-reducing behaviour may affect different types of health risk behaviours differently.

Discussion

Overall, there were a number of interesting findings in Experiment 1. First, my analyses indicated that I successfully manipulated perceived efficacy, and this was demonstrated in both the ANOVAs and the SEM analyses. The nature of the self-affirmation manipulation itself makes it difficult to determine whether it was successfully manipulated, as there is no well-established self-affirmation manipulation check. However, the self-affirmation manipulation used in Experiment 1 is commonly used in the literature (e.g., Harris & Napper, 2005; Sherman et al., 2000), and there was some evidence that I successfully manipulated self-affirmation: my results showed that I replicated the self-affirmation effect on risk perception found in previous studies. However, given the fact that I found few other effects of self-affirmation on the dependent variables, I cannot be certain that this variable was successfully manipulated.

Next, Experiment 1 also provided evidence that risk-reducing behavioural intentions are not one-dimensional, and instead can fall into one of several domains of health-promoting behaviour that should be treated as distinct categories. My study showed three behavioural intentions factors: behavioural intentions associated with

maintaining an active lifestyle; seeking medical advice and assessment; and maintaining a healthy diet. My analyses also showed that to some degree, my manipulations affected these distinct factors of behavioural intentions differently. With the factor analyses, overall, I generally found that manipulations to increase perceived efficacy were associated with greater intentions to engage in risk-reducing behaviours relative to manipulations to decrease perceived efficacy, which is consistent with previous literature. However, the effect was significant for only activity behavioural intentions, approached significance for medical behavioural intentions, and was nonsignificant for dietary behavioural intentions. When I examined the causal relationships among the variables using SEM, my analyses also indicated that the manipulation of perceived efficacy directly affected the behavioural intentions scales differently. Attempts at decreasing perceived efficacy caused increased behavioural intentions to seek medical advice and assessment, but did not appear to affect either of the other two types of intentions significantly. Thus, the results of the ANOVAs and the SEM support previous literature showing a positive relationship between perceived efficacy and behavioural intentions, but more importantly, these results also showed that my manipulations affected the domains of behavioural intentions differently. This finding may explain some of the inconsistencies in the results of previous studies' – which have generally treated behavioural intentions as having a single factor – examinations of self-affirmation on behavioural intentions and behaviours.

With regards to perceived risk, my study was the first to examine the influence of both self-affirmation and perceived efficacy on this variable. Research has suggested that in isolation, both variables are related to perceptions of risk, but the ANOVAs showed no

significant effect of either self-affirmation or perceived efficacy on risk perception, which was surprising. However, SEM analyses suggested that perhaps there was a suppression effect. That is, I found that my manipulation designed to increase perceived efficacy had direct positive effects on perceptions of risk, but also, that this manipulation had indirect *negative* effects on perceptions of risk, with participants' measured efficacy acting as a mediator. These results may explain my null findings shown in the ANOVAs, as the increase in risk perception caused by manipulating perceived efficacy may have been offset by the mediating effects of measured efficacy on risk perception. My findings are interesting, but at the same time somewhat puzzling. Previous correlational research has shown a negative relationship between perceived efficacy and perceived risk; individuals with a greater sense of efficacy related to a health risk demonstrate decreased perception of being at risk. However, what is puzzling is why my attempts at increasing perceived efficacy caused increased perceptions of risk. Overall, my findings suggest that manipulations of perceived efficacy may directly influence more than just perceptions of efficacy (i.e., measured efficacy). These findings suggest that the direct effects of this manipulation on the targeted variable (i.e., measured efficacy) may have additional, residual effects on other related variables. Moreover, these results highlight the value of exploring the dynamics of such variables in one coherent model. Previous research has not examined these variables in such a manner, and my results suggest that simple manipulations such as perceived efficacy can have complex effects on several variables.

There was a second interesting finding for the effects of the independent variables on perceived risk: there was a significant interaction between the two variables, but they did not interact in the way that I anticipated. I found that manipulated efficacy affected

risk perception only in those participants that did not self-affirm: attempts at both increasing perceived efficacy *and* decreasing perceived efficacy caused increased perceptions of risk. On the other hand, there were no differences in risk perception among the efficacy conditions in participants who did self-affirm. This was surprising, and did not make sense given prior literature. However, after considering my perceived efficacy manipulations, I concluded that this interaction may be due to a potential study confound. Participants in both the high and low manipulated efficacy conditions read passages that discussed heart attacks, while participants in the baseline condition did not – baseline participants read a passage that discussed only the history and structure of the heart muscle, with no mention of heart attacks. It may be that for the high and low perceived efficacy participants, simply reading a passage about heart attacks was enough to increase perceptions of risk. Now, this pattern only emerged in participants who had self-affirmed, thus it may be that because of the self-affirmation, baseline participants were less defensive and more willing to accept their risk. At any rate, because of this potential confound, I cannot be certain as to what effects my attempts at increasing and decreasing perceived efficacy had on risk perception, because the discussion of heart attacks may have increased risk perception regardless of measured efficacy. Consequently, in Experiment 2 I sought to correct this methodological issue.

Chapter 3: Experiment 2

Method

Participants

Participants were 243 undergraduate students (46 men, 197 women). All participants were between the ages of 17 and 24, with a mean age of 18.05 years (*SD* =

0.85). As in Experiment 1, because my manipulations involved future health outcomes (i.e., risk of heart attack prior to the age of 50) three participants aged 33, 41, and 53 were removed from the data set. Participants received either course credit or $5 for their participation.

Experimental Design and Procedure

Experiment 2 was the same design as Experiment 1: the study consisted of a 2 (self-affirmation condition: self-affirmation versus no self-affirmation) X 3 (manipulated perceived efficacy: high versus low versus baseline) between-subjects factorial design. Experiment 2 used exactly the same procedures and materials as Experiment 1, with the exception of one principal change: a modified baseline manipulated efficacy condition, such that the discussion of heart attacks was included. In Experiment 1, the baseline condition discussed the history and structure of the heart, with no mention of heart attacks. In Experiment 2, participants in the baseline manipulated efficacy condition read a information about the signs, symptoms, and physiology of a heart attack. In designing the passage, I was careful not to include any information about controllability and risk factors.

Measures

Perceived Efficacy Manipulation Check: This manipulation check consisted of the same four questions used in Experiment 1. Reliability analyses conducted on the four items showed adequate reliability (alpha = .76). As with Experiment 1, item-total statistics indicated that reliability would be improved if items 1 and 3 were removed. Based on these results the two items were eliminated, and the final scale comprised items 2 and 4, and had good reliability (alpha = .87). The final two-item measure of control

comprised the same items as those used in Experiment 1. Responses to the two items were summed to produce a measured efficacy score, with possible scores ranging from 2 to 14.

Self Risk Perception. Self risk perception was assessed using the same three questions used in Experiment 1. Reliability analyses of the three items showed them to have good reliability (alpha = .84). In addition to the three self risk items used in Experiment 1, an additional self risk item was added in Experiment 2, "Considering everything you know about yourself, what is the likelihood that YOU will have a heart attack prior to the age of 50? Please enter a number from 0 to 100", however, this item was not included in subsequent analyses[6].

Others Risk Perception. Others risk perception was assessed using the same three questions used in Experiment 1. Reliability analyses of the three items showed them to have excellent reliability (alpha = .93). In addition to the three others risk perception items used in Experiment 1, an additional others risk item was added, "Considering everything you know about Queen's students, what is the likelihood that the average Queen's University student of your age and sex will have a heart attack prior to the age of 50? Please enter a number from 0 to 100", however, this item was not included in subsequent analyses.

Risk-Reducing Behavioural Intentions. Behavioural intentions to reduce the risk of heart attack were assessed using the same 19 items used in Experiment 1 with some

[6] This item, along with the three self risk items were standardized and transformed into z scores to create a comparable metric, and these z scores were then summed to produce a total 4-item self risk score. An ANOVA was conducted using the 4-item self risk total as the dependent variable, with self-affirmation and perceived efficacy as the independent variables. Results of the ANOVA were similar to those using the three-item self risk total, thus to keep the self risk variables consistent among studies, the three-item self risk total was used. Similar results were obtained using the others risk items.

minor changes in wording (see Appendix E), and one additional item was added "I intend to speak with my family about my familial risk of heart disease and heart attacks". See Appendix C for the correlation matrix for the dependent variables.

Results

Factor Structure of Behavioural Intentions Measure

The results of Experiment 1 showed that there were three distinct factors underlying the risk-reducing behavioural intentions measure: behavioural intentions related to maintaining an active lifestyle; seeking medical advice and assessment; and maintaining a healthy diet. Given that the behavioural intentions measure used the same 19 items as Experiment 1 (plus one additional item) I expected that I would find a similar factor structure underlying the measure. My first set of analyses in Experiment 2 sought to examine the factor structure of the behavioural intentions items.

Factor Analysis Method

Experiment 2 used the same exploratory factor analytic methods as those used in Experiment 1 to identify the dimensions underlying the 20 behavioural intentions items.

Scree plot. The scree plot of eigenvalues from the reduced correlation matrix for the behavioural intentions items is presented in Figure 4. With this plot, the substantial drops in the curve following the first, second, and third eigenvalues provide support for a three-factor solution.

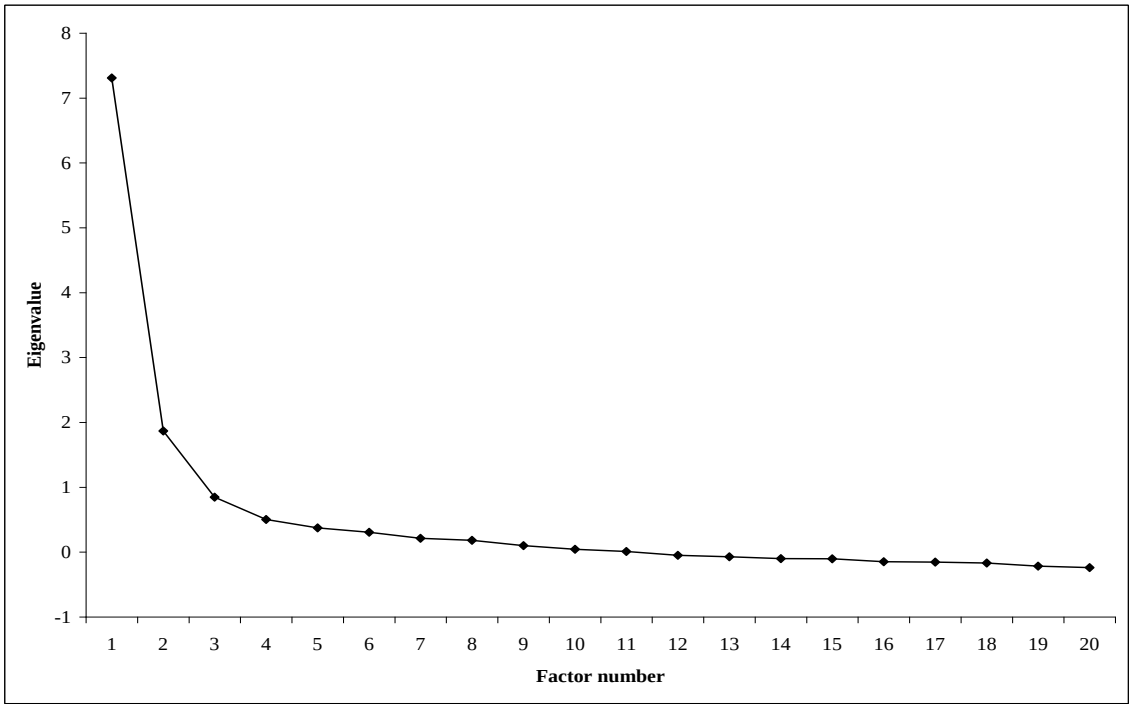


Figure 4. Experiment 2 behavioural intentions scree plot of eigenvalues.

Parallel analysis. The observed eigenvalues, the mean eigenvalues, and the 95th percentile eigenvalues corresponding to the behavioural intentions item factor analysis are presented in Table 7. Interestingly, even though there were very few differences between the behavioural intentions items in Experiments 1 and 2, the results of parallel analysis in Experiment 2 suggested that there were a greater number of factors compared to Experiment 1. For the mean eigenvalues, the observed eigenvalue was greater than the expected value until the nine-factor solution. Thus, using the mean values, a maximum of eight factors is supported. For the 95th percentile values, the observed eigenvalue was greater for every factor until the seven-factor solution, and a maximum of six factors was supported.

Table 7

Experiment 2 Behavioural Intentions Observed Eigenvalues, Mean Eigenvalues, and 95th Percentile Eigenvalues

Factor	Observed	Mean	95th Percentile
1	7.308	0.626	0.707
2	1.867	0.526	0.603
3	0.847	0.451	0.516
4	0.503	0.382	0.445
5	0.374	0.318	0.367
6	0.306	0.257	0.304
7	0.211	0.209	0.261
8	0.181	0.158	0.195
9	0.099	0.111	0.152
10	0.042	0.066	0.100
11	0.009	0.026	0.062
12	-0.049	-0.016	0.022
13	-0.073	-0.055	-0.016
14	-0.101	-0.096	-0.067
15	-0.104	-0.134	-0.097
16	-0.149	-0.177	-0.144
17	-0.155	-0.220	-0.195
18	-0.167	-0.257	-0.227
19	-0.216	-0.296	-0.267

20	-0.240	-0.343	-0.307

Model fit. RMSEA values and corresponding confidence intervals for the behavioural intentions items were compared for a series models, from one to eight factors. The magnitude of improvement in RMSEA between models was also considered. RMSEA values and 90% confidence intervals are presented in Table 8.

Table 8

Experiment 2 RMSEA Values and Corresponding 90% Confidence Intervals for the Behavioural Intentions Items

Number of factors	RMSEA	90% CI
1-factor	0.140	0.131-0.148
2-factor	0.098	0.089-0.108
3-factor	0.082	0.072-0.092
4-factor	0.062	0.050-0.074
5-factor	0.054	0.040-0.068
6-factor	0.048	0.031.-0.063
7-factor	0.042	0.021-0.060
8-factor	0.033	0.000.-0.054

The one-factor model demonstrated poor model fit. The two-factor model decreased in RMSEA by 0.042 and represented a substantial improvement, and the fit of the model moved to the marginal range. The three-factor model decreased in RMSEA by

0.016, but model fit remained in the marginal range. The four-factor model decreased in RMSEA by 0.020 and represented a substantial improvement as well, and the fit of the model moved to the acceptable range. Interestingly, the results of this model fit assessment look similar to those of Experiment 1. Once again, even though the four-factor model represented substantial improvement from the three-factor model, the confidence intervals begin to widen following the three-factor solution, which indicates that the precision of the estimate of model fit is decreasing. Next, the five-factor model decreased in RMSEA modestly (0.08), and the fit of the model remained in the acceptable range. The six-factor model decreased in RMSEA minimally (0.06), however, the fit of the model moved to the good range. With the seven-and eight-factor models, there were again only modest decreases in RMSEA (0.06 and 0.09, respectively). Given these results, it is difficult to justify the five-, six-, seven-, and eight-factor solutions. Overall, the model fit assessment of this data supports a four-factor solution, and potentially a three-factor solution.

Interpretability and stability of solutions Thus far, the factor-number procedures used in examining the factor structure of the behavioural intentions items supported a range of solutions, including three-, four-, six-, and eight-factor solutions. To further investigate the utility of the models, I examined their interpretability.

I examined the rotated solution for the three-factor solution first. Factor loadings and communalities are presented in Table 9, and factor loadings that are bolded represent substantial loadings. Recall that in Experiment 1, a three-factor model was concluded as best representing the behavioural intentions data. Here, the first factor appeared to represent behavioural intentions related to seeking medical advice and assessment. Four

items had substantial loadings on this first factor, with low loadings on the second and third factors. The second factor appeared to represent behavioural intentions related to maintaining an active lifestyle, with five items loading substantially. Finally, the third factor appeared to best represent intentions related to maintaining a healthy diet, with five

Table 9

Experiment 2 Pattern Matrix of Rotated Three-Factor Solution of the Behavioural Intentions Items

Item	Pattern Coefficients			Communalities
	1	2	3	
Get cholesterol checked	**0.96**	-0.01	-0.06	0.75
Have blood pressure checked	**0.77**	0.06	0.003	0.68
Speak with physician	**0.74**	0.12	0.05	0.74
Speak to family about risk	**0.67**	0.05	0.15	0.70
Engage in cardiovascular activity	0.05	**0.86**	-0.13	0.62
Go to the gym	-0.03	**0.84**	-0.06	0.59
Engage in regular physical activity	0.03	**0.80**	0.04	0.65
Weight training	0.11	**0.64**	0.06	0.54
Physical activity with friends	0.16	**0.60**	0.10	0.54
Eat more salads	0.12	0.14	**0.65**	0.58
Eat more plant-based protein	0.19	-0.07	**0.53**	0.40
Decrease fast food	0.12	0.13	**0.53**	0.49
Eat 5-10 servings of fruits & vegetables	0.12	0.23	**0.48**	0.51
Alcohol in moderation	0.13	-0.19	**0.46**	0.30

Maintain a healthy diet	-0.14	0.44	0.34	0.46
Decrease red meat	0.17	0.12	0.28	0.33
Decrease fat	0.21	0.23	0.33	0.42
Decrease saturated fat	0.36	0.16	0.39	0.57
Avoid smoking cigarettes	-0.11	0.05	0.34	0.17
Walk/bike/rollerblade when possible	0.18	0.37	0.24	0.45

items loading substantially. One item loaded substantially on Factor 2 and marginally on Factor 3, and five items failed to load substantially on any of the three factors. Factors 1 and 2 were moderately correlated (r = .32); as were Factors 2 and 3 (r = .44). Factors 1 and 3 were highly correlated (r = .51). This three-factor solution is quite similar to the three-factor solution shown in Experiment 1, and as will be seen in Experiment 3, the three-factor solution from a subsequent study. Thus, from both a stability and an interpretability standpoint, the three-factor solution appears to provide a good representation of the data.

The rotated solution for the four-factor solution was examined next. This solution appeared to be quite similar to that of the three-factor solution; however, the medical advice and assessment factor split into two factors, with two items loading on each factor. I decided that the four-factor solution did not best represent the data, for several reasons. First, the two medical intentions factors were highly correlated (r = .47), which suggested that keeping them as two separate factors would not provide much additional useful information. Second, from a stability and replicability standpoint, this four-factor solution did not emerge in Experiment 1, and as will be seen, did not emerge in Experiment 3.

I next considered the rotated solution for the six-and eight-factor solutions. For the six-factor solution, there was one factor where only one item loaded substantially. As noted previously, factors comprised of only one item can indicate over-factoring. There were several problems with eight-factor solution: several items loaded on multiple factors, a number of items failed to load substantially on any factor, and there was at least one factor where only one item loaded substantially. Thus, I concluded that none of the four-, six, or eight-factor solutions best represented the behavioural intentions items data.

Conclusion. Scree plot examination, parallel analysis, and model fit procedures all suggested different factor solutions for the behavioural intentions items. However, when examining the interpretability of the models, the three-factor solution appeared to best represent the data. This model was readily interpretable and made sense conceptually. In addition, the three-factor solutions for Experiments 1 and 2 were almost identical, with the exception of three items not loading substantially on the dietary behavioural intentions scale. Overall, there was stability of this three-factor model across multiple studies: this solution was consistent with the three-factor solution identified for the behavioural intentions items in Experiment 1, and as will be seen in Experiment 3, consistent with behavioural intentions items for a different health risk.

The results of these factor analyses replicated the results of Experiment 1, and, provided additional support for the premise that risk-reducing behavioural intentions are multidimensional and comprised of several distinct categories. Thus, consistent with Experiment 1, a three-factor solution, reflecting behavioural intentions related to maintaining an active lifestyle, seeking medical advice and assessment, and maintaining a healthy diet was concluded as best representing the behavioural intentions data. Based on

these results, three behavioural intentions scales were created using the same items as those in Experiment 1. Reliability analyses showed both the activity and medical behavioural intentions scales to have good reliability (alphas = .88 and .87, respectively) and the dietary behavioural intentions scale to have adequate reliability (alpha = .74). The medical and dietary behavioural intentions scales were strongly correlated (r = .56), and the medical and activity scales were moderately correlated (r = .39), as were the dietary and activity scales (r = .42).

Univariate ANOVAs

Manipulation Check

As with Experiment 1, prior to the main analyses I conducted a manipulation check to ensure that the perceived efficacy manipulation was effective in altering participants' measured efficacy relative to heart attack risk. To examine these effects, a 2 (self-affirmation condition: self-affirmation versus no self-affirmation) X 3 (manipulated perceived efficacy: high versus low versus baseline) ANOVA was conducted. Measured perceived efficacy was the dependent variable. The ANOVA showed a significant main effect for manipulated efficacy, $F(2, 238) = 141.84$, $p < .001$. Significant differences among the three conditions were evaluated using the 95% confidence intervals. Participants in the high manipulated efficacy condition reported significantly higher measured efficacy (M = 12.59, CI = 12.14-13.04) compared to participants in the baseline condition (M = 10.74, CI = 10.32-11.17), and participants in the baseline condition reported significantly higher ratings of control compared to those in the low condition (M = 7.28, CI = 6.84-7.73). These significant findings suggested that my manipulation of

perceived efficacy was once again successful in influencing participants' measured efficacy in relation to heart attack risk.

Experiment 1 showed no significant main effect for self-affirmation, and the results of Experiment 2 were consistent with this finding: there was no significant main effect for self-affirmation, $F(1, 238) = 0.03$, $p = .87$. Experiment 1 did, however, show a marginally significant interaction between manipulated efficacy and self-affirmation. This result was also true in Experiment 2, $F(2, 238) = 2.49$, $p = .09$. Once again, I felt that this interaction warranted further investigation. The estimated marginal means and 95% confidence intervals for measured efficacy as a function of the two independent variables are presented in Table 10. As can be seen by the 95% confidence intervals,

Table 10

Experiment 2 Estimated Marginal Means and 95% Confidence Intervals for Measured Efficacy

Self-Affirmation	Perceived Efficacy	Estimated Marginal Mean	95% Confidence Interval
No self-affirmation	Low	7.64	7.03-8.25
	Baseline	10.42	9.82-11.02
	High	12.49	11.85-13.12
Self-affirmation	Low	6.92	6.27-7.57
	Baseline	11.07	10.47-11.66
	High	12.69	12.06-13.33

under no self-affirmation, there were significant differences among all three of the manipulated efficacy conditions. Similarly, under self-affirmation, there were also significant differences among all three of the manipulated efficacy conditions; however, as with Experiment 1, these results suggest that the effects of the perceived efficacy manipulation were somewhat stronger in participants who self-affirmed.

Unrealistic Optimism

Prior to examining the effects of the independent variables on risk perception, I once again wanted to confirm that I did in fact, replicate the classic unrealistic optimism effect. Experiment 1 replicated the unrealistic optimism effect, showing that participants viewed themselves as being significantly less at risk relative to similar others. Thus, I expected a similar effect for participants in Experiment 2. To examine this relationship, I conducted a paired sample *t* test. As predicted, the results showed that individuals' perceptions of others risk ($M = 16.49$, $SD = 5.18$) were significantly greater than perceptions of self risk ($M = 12.58$, $SD = 5.18$), $t\ (242) = 11.78$, $p < .001$, again replicating the unrealistic optimism effect.

Self Risk Perception

The next set of analyses tested the study's core hypotheses. As with Experiment 1, the first analysis examined the effects of the independent variables on self risk perception. A 2 (self-affirmation condition: self-affirmation versus no self-affirmation) X 3 (manipulated perceived efficacy: high versus low versus baseline) ANOVA was conducted, with the self risk score as the dependent variable.

Experiment 1 showed no main effect of self-affirmation on risk perception, thus I did not expect a significant main effect for this manipulation in Experiment 2. My

hypothesis was confirmed; the ANOVA showed no significant main effect for self-affirmation, $F(1, 237) = 0.16$, $p = .69$, indicating there were no differences in risk perception for participants who self-affirmed versus those who did not self-affirm.

Experiment 1 also showed no significant main effect of manipulated efficacy, thus again, I did not expect a significant main effect in Experiment 2. My hypothesis was confirmed; the ANOVA showed no significant main effect for manipulated efficacy $F(2, 237) = 0.83$, $p = .44$, indicating there were no significant differences in risk perception among participants in the three manipulated efficacy conditions.

Finally, I considered the interaction between self-affirmation and manipulated efficacy. Experiment 1 showed a significant interaction between these two variables: when participants self-affirmed, manipulations of efficacy had no effect on perceptions of self risk; and when participants did not self-affirm, attempts at both increasing and decreasing efficacy were associated with increased perceptions of risk. However, recall that there was a potential confound of the baseline manipulated efficacy condition in Experiment 1, and that the baseline condition in Experiment 2 was altered to correct this. This correction would allow me to determine whether the significant interaction in Experiment 1 was due to meaningful effects, or whether it was due to the potential confound. Results of Experiment 2 showed no significant interaction between self-affirmation and manipulated efficacy $F(2, 237) = 1.16$, $p = .32$. This finding suggests that the significant interaction found in Experiment 1 may have been driven by the confounded baseline condition.

Behavioural Intentions

The next set of analyses examined the effects of the independent variables on risk-reducing behavioural intentions. As with Experiment 1, I conducted a series of three 2 (self-affirmation condition: self-affirmation versus no self-affirmation) X 3 (manipulated perceived efficacy: high versus low versus baseline) ANOVAs, with each of the three behavioural intentions scales as the dependent variable.

In Experiment 1, there was no significant main effect for self-affirmation on any of the three behavioural intentions variables. This result was replicated in Experiment 2: the ANOVA showed no significant self-affirmation main effect for activity behavioural intentions, $F(1, 237) = 0.28$, $p = .60$; medical behavioural intentions, $F(1, 237) = 0.004$, $p = .95$; or dietary behavioural intentions, $F(1, 237) = 0.27$, $p = .61$ indicating there were no differences in behavioural intentions for participants who self-affirmed versus those who did not self-affirm.

I next considered the main effect of manipulated efficacy. The ANOVAs showed no significant main effect for activity behavioural intentions, $F(2, 237) = 0.62$, $p = .54$; or medical behavioural intentions, $F(2, 237) = 1.20$, $p = .30$. However, the effect did approach significance for dietary behavioural intentions, $F(2, 237) = 2.62$, $p = .07$. The estimated marginal means and 95% confidence intervals for each of the three behavioural intentions scales are presented in Table 11. In Experiment 1, there was a significant manipulated efficacy main effect for activity behavioural intentions, and a marginally significant main effect for medical behavioural intentions, such that participants in the high condition showed the greatest intentions to reduce their risk, followed by participants in the baseline then low manipulated efficacy conditions. There was no

Table 11

Experiment 2 Estimated Marginal Means and 95% Confidence Intervals for Behavioural Intentions

	Activity Behavioural Intentions (95% CI)	Medical Behavioural Intentions (95%CI)	Dietary Behavioural Intentions (95%CI)
Low Manipulated Efficacy	39.60 (37.17-42.03)	19.18 (17.34-21.02)	38.66 (36.49-40.84)
Baseline Manipulated Efficacy	41.06 (38.76-43.35)	21.09 (19.35-22.83)	41.65 (39.60-43.70)
High Manipulated Efficacy	41.44 (39.01-43.86)	20.67 (18.83-22.50)	41.80 (39.63-43.96)

significant effect for dietary behavioural intentions in Experiment 1. Thus, the results of Experiment 2 are somewhat opposite to those of Experiment 1, which is somewhat puzzling. However, examination of the means shows that in general, the results trended in the expected direction, that is, high manipulated efficacy participants showed the greatest intentions, followed by those in the baseline, then low manipulated efficacy conditions.

My next step was to examine the interactive effects of self affirmation and manipulated efficacy on behavioural intentions. In Experiment 1, there was no significant interaction between the two independent variables. This result was replicated in Experiment 2: results of the ANOVAs showed no significant interaction for activity behavioural intentions, $F(2, 237) = 0.23$, $p = .79$; medical behavioural intentions, $F(2, 237) = 0.44$, $p = .65$; or dietary behavioural intentions, $F(2, 237) = 0.44$, $p = .64$.

Structural Equation Modeling

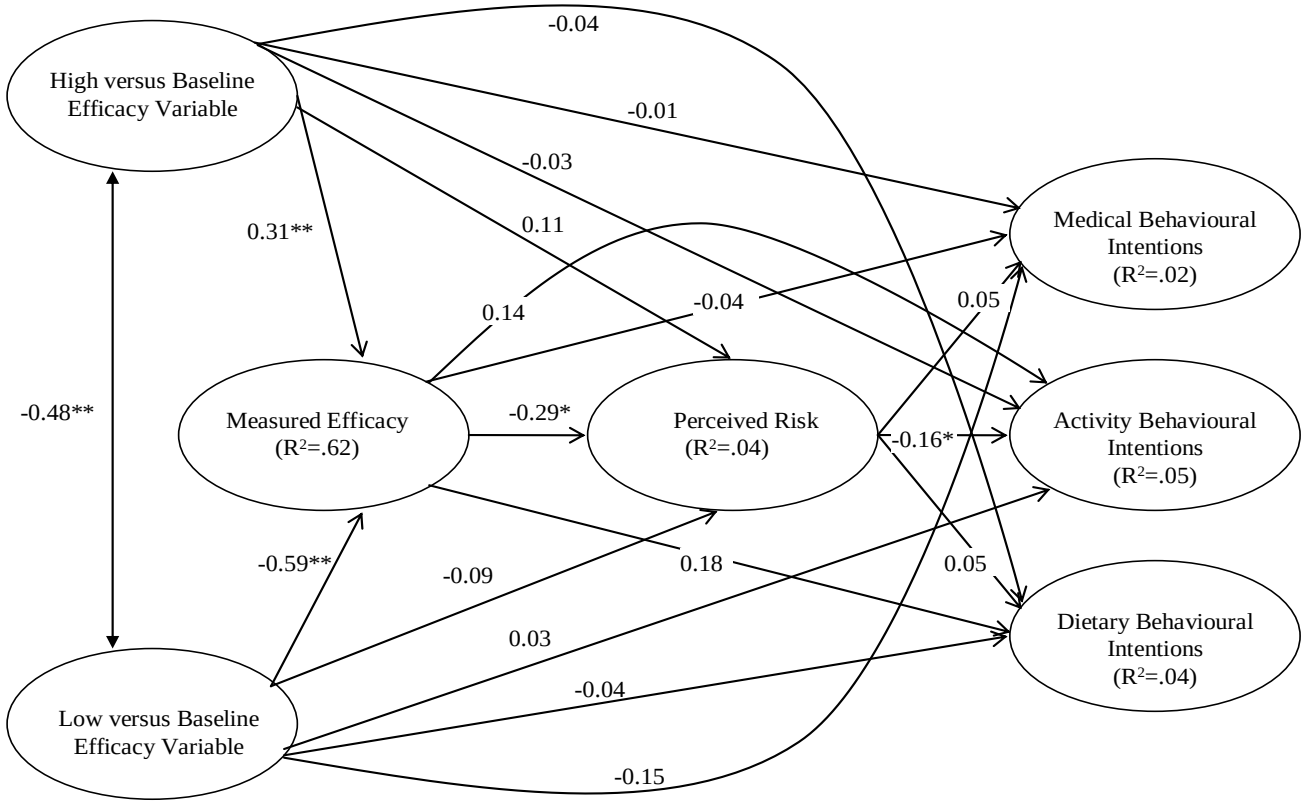
Model Specification and Model Fitting

The final set of analyses in Experiment 2 sought to examine the causal relationships among the independent and dependent variables. Once again, I sought to examine the dynamics among the dependent variables, as well as the effects of the manipulated variables on these dependent variables. Moreover, I wanted to determine whether the model of interrelationships proposed and evaluated in Experiment 1 would produce similar results in Experiment 2. I evaluated the same model and using the same SEM procedures as Experiment 1.

Model fit. Model fit was evaluated using the same four goodness-of-fit indices used in Experiment 1. For the proposed model, three of the four indices indicated good model fit: RMSEA = 0.049 (90% CI = 0.037-0.061); SRMR = 0.056; and NNFI = 0.97. The χ^2 Test of Goodness-of-Fit was significant χ^2 (151, N = 243) = 235.51, $p < .001$, indicating that the model was not a perfect representation of the data. However, for the reasons discussed in Experiment 1, a significant result is not sufficient grounds to reject a model.

Path Estimates. The path estimates for the model are presented in Figure 5. All of the 20 measured variables loaded significantly (ps < .05) on their hypothesized latent variables. For the sake of simplicity, only the structural paths are presented in the figure.

First, these results show that assignment to manipulated efficacy condition had a significant effect on measured efficacy: both the high efficacy dummy variable and the low efficacy dummy variable paths were significant, which is consistent with Experiment



+ $p \leq .10$, * $p \leq .05$, ** $p \leq .01$

Figure 5. Experiment 2 path diagram

1. There was a strong positive relationship between the high efficacy dummy variable and measured efficacy, which indicates that participants in the high manipulated efficacy condition had higher measured efficacy relative to participants in the baseline condition. Conversely, there was a strong negative relationship between the low efficacy dummy variable and measured efficacy, which indicates that participants in the low manipulated efficacy condition had lower measured efficacy relative to participants in the baseline condition. This result replicates Experiment 1's finding, and supports earlier ANOVAs which indicated that my manipulation of perceived efficacy worked quite well in affecting participants' measured efficacy.

The results of Experiment 1 suggested the presence of a suppression effect: the high efficacy dummy variable had a direct positive effect on perceived risk, as well as an indirect *negative* effect on perceived risk though a mediator, measured efficacy. That is, that the manipulation to increase perceived efficacy caused increased perceptions of risk, but that it also caused increased measured perceptions of efficacy which in turn, caused decreased perceived risk. In Experiment 2, I did replicate the strong negative relationship between measured efficacy and perceived risk: higher measured efficacy caused lower perceptions of risk. However, I did not find a significant direct effect of the high efficacy dummy variable on perceived risk in Experiment 2. This relationship was small and positive, and in the expected direction, but nonsignificant (p = .19).

Finally, perceptions of risk significantly influenced activity behavioural intentions in Experiment 2. Examination of the standardized regression coefficient for this path indicates this is a moderate negative relationship, suggesting that higher perceptions of risk caused lower intentions to engage in physical activity. This path was not significant

in Experiment 1, although there was a small negative relationship. This finding is somewhat puzzling, as it counters what prior health risk research would suggest, that is, that individuals who feel more at risk show greater intentions to reduce their risk.

Discussion

Experiment 2 had two primary goals. First, I sought to correct a methodological issue raised in Experiment 1 by altering a potential confound in the baseline perceived efficacy condition. Second, I sought determine whether the results of Experiment 1 could be replicated in Experiment 2.

Experiment 2 provided a number of interesting findings. First, the analyses showed that once again, I successfully manipulated perceived efficacy, and this was demonstrated in both the ANOVAs and the SEM analyses. As previously noted, given the nature of my self-affirmation manipulation, there is no established manipulation check for self-affirmation. Experiment 1 provided some evidence that I successfully manipulated self-affirmation, as it replicated the self-affirmation effect on risk perception found in previous studies. However, I found few other effects of self-affirmation on the dependent variables. In Experiment 2, I found no evidence that self-affirmation significantly affected any of the dependent variables. Thus, even though my self-affirmation manipulation is commonly used, I cannot be certain that this variable was successfully manipulated.

Next, Experiment 2 provided additional evidence that risk-reducing behavioural intentions are not one-dimensional, and instead can fall into one of several distinct domains of health-promoting behavioural intentions. Experiment 2 replicated the results of Experiment 1: my study showed three distinct categories of behavioural intentions:

behavioural intentions associated with maintaining an active lifestyle; seeking medical advice and assessment; and maintaining a healthy diet. The ANOVAs in Experiment 1 provided some evidence that my manipulation of perceived efficacy affected the three categories in different ways, with the strongest effects found for intentions related to maintaining an active lifestyle. However, in Experiment 2, the ANOVA did not show any significant effects for the manipulation of perceived efficacy, with the exception of a marginally significant effect for intentions related to maintaining a healthy diet. In fact, the results of the ANOVAs in Experiment 2 were somewhat opposite to those of Experiment 1, which was puzzling. When I examined the causal relationships among the variables using SEM in Experiment 2, I found that perceptions of risk significantly influenced behavioural intentions related to maintaining an active lifestyle; however, this relationship was negative, which was unexpected. This suggests that individuals who perceived themselves as being at greater risk showed decreased behavioural intentions to engage in physical activity, which is counter to what prior research would suggest.

There were several interesting findings related to perceived risk in Experiment 1. First, recall there was an unexpected interaction between self-affirmation and manipulated efficacy in Experiment 1, which was thought to be due to a confounded baseline condition. In Experiment 2, I sought to correct this methodological issue, which would allow me to determine whether the unexpected interaction in Experiment 1 was due to actual effects, or whether it was due to the potential confound. My hypothesis was correct; after correcting the baseline manipulated efficacy condition in Experiment 2, I found no interaction between self-affirmation and manipulated efficacy, which suggests

that the significant interaction found in Experiment 1 was likely driven by the confounded baseline condition.

Second, Experiment 1 suggested the presence of a suppression effect, that is, I found that the manipulation designed to increase perceived efficacy had direct positive effects on perceptions risk, but also, that this manipulation had indirect *negative* effects on perceptions of risk, with measured efficacy acting as a mediator. In Experiment 2, I did replicate the strong negative relationship between measured efficacy and perceived risk; however, I did not find a significant direct effect of high manipulated efficacy on perceived risk. This relationship was small and positive, and in the expected direction, but nonsignificant. In considering the differences between Experiment 1 and Experiment 2, this nonsignificant finding could perhaps be explained by the change in the baseline manipulated efficacy condition in Experiment 2.The discussion of heart attack and its risk factors in the baseline condition (which did not occur in Experiment 1) may have attenuated the effect found in Experiment 1.

Overall, Experiment 2 provided a number of useful results. First, I found additional evidence that risk-reducing behavioural intentions can fall into several distinct categories of health promoting behaviour. Second, I found additional support for a negative relationship between perceived control and perceptions of risk. Finally, the results of Experiment 2 once again highlight the value of exploring the dynamics of such variables in one coherent model. Previous research has not examined these variables in such a manner, and as with Experiment 1, my results suggest that simple manipulations can have complex effects on several variables. I next wondered whether the results of Experiments 1 and 2 could be generalized to other health risks. Thus, in Experiment 3, I

sought to examine the same variables and the same relationships using a different health issue: risk of developing colorectal cancer.

Chapter 4: Experiment 3

Method

Participants

Participants were 253 undergraduate students (75 men, 178 women). All participants were between the ages of 17 and 27, with a mean age of 18.69 years (*SD* = 1.31). Because the nature of my study involved future health outcomes (i.e., risk of colorectal cancer prior to the age of 50) two participants ages 31 and 33 were removed from the data set. Participants received either course credit or $5 for their participation.

Experimental Design and Procedure

Experiment 3 used the same design as Experiments 1 and 2: the study consisted of a 2 (self-affirmation condition: self-affirmation versus no self-affirmation) X 3 (manipulated perceived efficacy: high versus low versus baseline) between-subjects factorial design. However, Experiment 3 set out to examine whether the effects found in Experiments 1 and 2 could be generalized to another health risk paradigm: risk of colorectal cancer prior to the age of 50. As in the first two experiments, in Experiment 3 all participants received the same order of manipulations and dependent measures: the self-affirmation manipulation, followed by the perceived efficacy manipulation, followed by negative health risk information, and finally the dependent measures.

At the outset of the study, participants were told that the purpose of the study was ostensibly to determine the predictors of colorectal cancer and to validate a risk

assessment questionnaire (See Appendix F for all materials used in Experiment 3) They were shown the following message:

> Our lab been measuring university-aged students on certain behavioural characteristics and then following them over time to track who develops colorectal cancer. By using ordinary least-squares multiple regression we've developed a mathematical formula that can predict whether or not an individual is at an increased risk of developing colorectal cancer, and how high an individual's risk is, based on these behavioural characteristics. Using these results, we have developed a risk assessment questionnaire that can estimate an individual's risk of developing colorectal cancer prior to the age of 50. Today, you are going to be taking part in a study further validating this risk assessment questionnaire.

As with Experiments 1 and 2, following the study introduction, participants were told that prior to the main risk assessment study, they would be asked to complete two brief studies for other researchers associated with the lab, which again, were the two experimental manipulations: participants were told that the self-affirmation manipulation was an attitudes-related study examining values, and that the perceived efficacy manipulation was a memory study related to the lab's research examining variables associated with colorectal cancer.

Self-affirmation manipulation. I used the same self-affirmation manipulation used in Experiments 1 and 2 (See Appendix A).

Perceived efficacy manipulation. As with Experiments 1 and 2, the perceived efficacy manipulation had three levels, high, low, and baseline manipulated efficacy. Participants in the *high manipulated efficacy* condition read a passage about colorectal

cancer risk emphasizing that the prevention of colorectal cancer is easier than one might think. To increase response efficacy, the passage emphasized that behaviour and lifestyle were significant predictors of colorectal cancer risk, with diet, physical activity, smoking status, and weight being the most important factors in the prevention of a colorectal cancer. Participants in this condition were also told that background factors such as genetics and familial history of colorectal cancer and bowel disease were only minor predictors of colorectal cancer. To increase self-efficacy, the passage emphasized that colorectal cancer prevention was well within their control, and that they could substantially reduce their risk with simple and effective behavioural changes.

Participants in the *low manipulated efficacy* condition read a passage about colorectal cancer risk emphasizing that the prevention of colorectal cancer is difficult. To decrease response efficacy, the passage emphasized that behaviour and lifestyle were not as important as (uncontrollable) factors such as genetics and familial history of colorectal cancer and bowel disease, as well as other common cancers (e.g., breast cancer, prostate cancer, and uterine cancer). This passage described research showing that genetics and uncontrollable situational factors such stress were the most important predictors of colorectal cancer risk. To decrease self-efficacy, participants were told that individuals may not have as much control over their risk of developing colorectal cancer as they have been led to believe.

Participants in the *baseline manipulated efficacy* condition served as a control group for the perceived efficacy manipulation. As with Experiment 2, the baseline condition avoided a potential confound by discussing colorectal cancer. Participants read a paragraph about the signs and symptoms of colorectal cancer, as well as a description of

the different methods used to screen and test for colorectal cancer. In designing the passage, I was careful not to include any information about controllability and risk factors.

As with Experiments 1 and 2, following each of the perceived efficacy manipulations, all participants completed multiple choice questions testing their memory for the paragraph they had just read. Within these filler questions was a manipulation check designed to ensure that perceptions of perceived efficacy were properly manipulated.

Negative Risk Feedback. Just as in Experiments 1 and 2, following the two experimental manipulations the participants were exposed to negative health risk information through the use an ostensible computerized risk assessment questionnaire (RAQ; see Appendix F). The RAQ comprised 31 questions regarding participants' health behaviours such as their diet, physical activity, and their familial history of colorectal cancer, bowel disease, and cancer.

Participants were given risk feedback regarding their risk of developing colorectal cancer prior to the age of 50. However, once again, risk feedback was *not* based on participants' responses to the RAQ questions; instead, all participants received the same, high-risk feedback. Each participant was told that they were likely to develop colorectal cancer prior to the age of 50, and that the percentage likelihood of this occurring was 45%. The percentage likelihood value was determined through pretesting; 20 undergraduate students were asked to estimate their likelihood of developing colorectal cancer prior to the age of 50, and I chose a value that was extreme (i.e., double) relative

to this mean. After participants were given their risk feedback, they completed the dependent measures: risk perception and risk-reducing behavioural intentions.

Measures

Perceived Efficacy Manipulation Check: This manipulation check consisted of the same four questions used in Experiments 1 and 2, using colorectal cancer risk instead of heart attack risk: two questions assessed participants' general perceptions of control over their risk of colorectal cancer and two questions assessed more specific aspects of control (the effects of background factors and situational factors on risk; see Appendix G). Reliability analyses conducted on the four items showed adequate reliability (alpha = .72), and item-total statistics indicated that reliability would be improved if item 3 was removed (alpha = .80). Additional analyses indicated that reliability could be further improved (albeit minimally) if item 1 was removed. To maintain consistency with the measured efficacy scale used in Experiments 1 and 2, item 1 was removed as well. The final scale comprised items 2 and 4 and had good reliability (alpha = .81). Responses to the two items were summed to produce a measured efficacy scale, with possible scores ranging from 2 to 14.

Self Risk Perception. Participants' estimates of their own risk were assessed using one question: "Prior to the age of 50, what is the likelihood that you will develop colorectal cancer?". Responses were given on an 11-point Likert scale ranging from 1 (*extremely unlikely*) to 11 (*extremely likely*). An additional self risk item was also included in Experiment 3 "Considering everything you know about yourself, what is the likelihood that YOU will develop colorectal cancer prior to the age of 50? Please enter a

number from 0 to 100", however, for the same reasons indicated in Experiment 2, this item was not included in subsequent analyses.

Others Risk Perception. Others risk perception was assessed using two questions. The first question asked "Prior to the age of 50, what is the likelihood that the average Queen's University student of your age and sex will develop colorectal cancer?", and responses were given on an 11-point Likert scale ranging from 1 (*extremely unlikely*) to 11 (*extremely likely*). The second question asked "Considering everything you know about Queen's students, what is the likelihood that the average Queen's University student of your age and sex will develop colorectal cancer prior to the age of 50? Please enter a number from 0 to 100", however, for the same reasons indicated in Experiment 2, this item was not included in subsequent analyses.

Risk-Reducing Behavioural Intentions. Behavioural intentions to reduce the risk of colorectal cancer were similar to those used in the heart attack paradigm. However, a number of items were altered to account for the differences in the two health conditions (see Appendix G for behavioural intentions items). For example, a number of additional items related to diet were added (e.g., intentions related to eating more fiber and less processed meat). There were 21 behavioural intentions items, including questions related to diet, physical activity, and colorectal health behaviour (e.g., speaking with physician, knowledge of familial risk). Responses were made on an 11-point Likert scale ranging from 1 (*strongly disagree*) to 11 (*strongly agree*). See Appendix C for the correlation matrix for the dependent variables.

Following the completion of the dependent measures, all participants were thoroughly debriefed and informed of the true nature of the study (See Appendix H for

information sheet, consent form, and debriefing materials). Participants were given both an oral debriefing, as well as a written debriefing sheet. To dispel any concerns participants may have had about their risk of colorectal cancer, they were told that the risk feedback they received was fabricated and that all participants in the study received the same high-risk feedback. In addition, to address any misconceptions about colorectal cancer risk presented in the study, all participants received a colorectal cancer fact sheet.

Results

Factor Structure of Behavioural Intentions Measure

The first set of analyses sought to examine the factor structure of the behavioural intentions items. The results of Experiment 1 and 2 both showed that there were three distinct factors underlying the risk-reducing behavioural intentions measure: behavioural intentions related to maintaining an active lifestyle, seeking medical advice and assessment, and maintaining a healthy diet. The behavioural intentions items used in Experiment 3 were slightly different than the items used in Experiments 1 and 2, with the addition and deletion of several items. However, the items remained similar enough such that I did expect to find a similar factor structure underlying them.

Factor Analysis Method

Experiment 3 used the same EFA methods as those used in Experiments 1 and 2 to identify the dimensions underlying the 21 behavioural intentions items.

Scree plot. The scree plot of eigenvalues from the reduced correlation matrix for the behavioural intentions items is presented in Figure 6. With this plot, the substantial drops in the curve following the first, second, and third eigenvalues provide support for a three-factor solution.

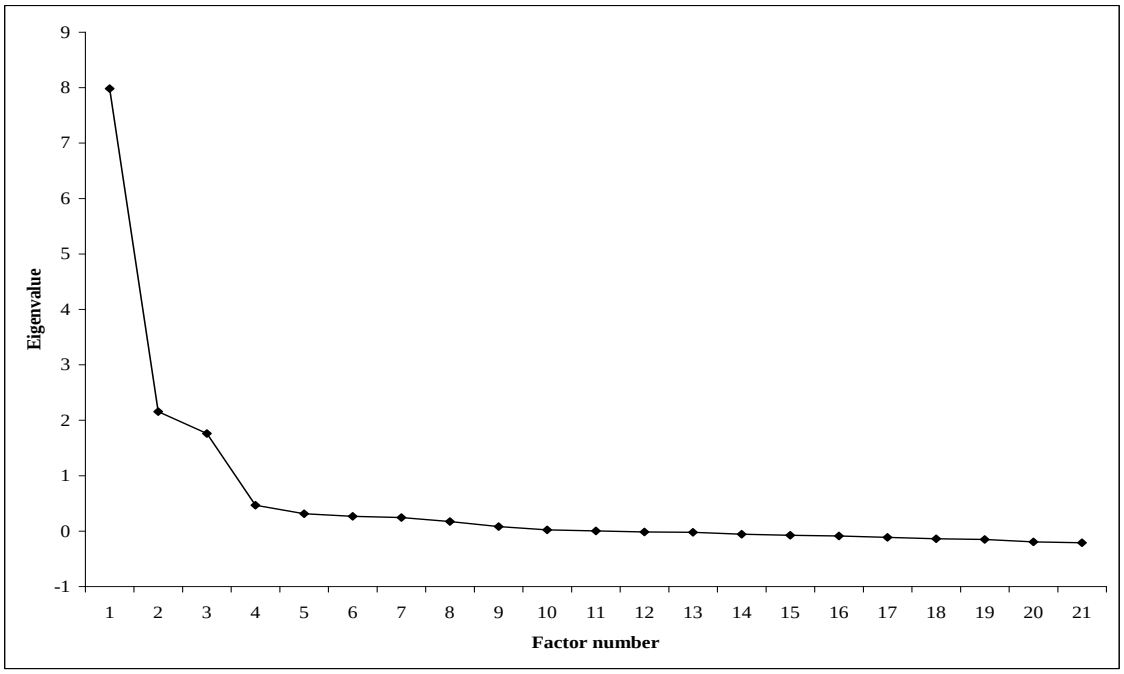


Figure 6. Experiment 3 behavioural intentions scree plot of eigenvalues.

Parallel analysis. The observed eigenvalues, the mean eigenvalues, and the 95th percentile eigenvalues are presented in Table 12. For both the mean and the 95th percentile values, the observed eigenvalue was greater than the expected value until the five-factor solution. Thus, both sets of values support a four-factor solution.

Table 12

Experiment 3 Behavioural Intentions Observed Eigenvalues, Mean Eigenvalues, and 95th Percentile Eigenvalues

Factor	Observed	Mean	95th Percentile
1	7.983	0.641	0.748
2	2.155	0.533	0.608
3	1.760	0.454	0.516

4	0.465	0.398	0.447
5	0.313	0.336	0.391
6	0.265	0.284	0.333
7	0.245	0.230	0.271
8	0.172	0.181	0.222
9	0.082	0.138	0.179
10	0.022	0.090	0.122
11	0.002	0.043	0.081
12	-0.017	0.009	0.047
13	-0.023	-0.033	-0.003
14	-0.057	-0.073	-0.039
15	-0.076	-0.109	-0.082
16	-0.088	-0.145	-0.113
17	-0.114	-0.184	-0.158
18	-0.139	-0.220	-0.194
19	-0.152	-0.260	-0.222
20	-0.196	-0.301	-0.270
21	-0.211	-0.357	-0.328

Model fit. RMSEA values and corresponding confidence intervals for the behavioural intentions items were compared for a series of models, from one to four factors (the factor analysis for the five-factor model could not be computed, even after

100 iterations). The magnitude of improvement in RMSEA between models was also considered. RMSEA values and 90% confidence intervals are presented in Table 13.

The one-factor model demonstrated poor model fit. The two-factor model decreased in RMSEA by 0.034 and represented a substantial improvement, but the fit of the model remained in the poor range. The three-factor model decreased in RMSEA by 0.072 and represented a substantial improvement as well, and the fit of the model moved to the good range. Finally, the four-factor model decreased in RMSEA minimally (0.006) and model fit remained in the good range. Based on this model fit assessment, a three- or four-factor solution is supported.

Table 13

Experiment 3 RMSEA Values and Corresponding 90% Confidence Intervals for the Behavioural Intentions Items

Number of factors	RMSEA	90% CI
1-factor	0.176	0.168-0.184
2-factor	0.142	0.125-0.142
3-factor	0.070	0.059-0.080
4-factor	0.064	0.053-0.075

Interpretability and stability of solutions. Thus far, the factor-number procedures used in examining the factor structure of the behavioural intentions items supported both three- and four-factor solutions. To further investigate the utility of these models, I examined their interpretability.

The rotated solution for the three-factor solution was examined first. Recall that in both Experiments 1 and 2, three-factor solutions were identified as best representing the behavioural intentions data. Factor loadings and communalities are presented in Table 14. Factor loadings that are bolded represent substantial loadings. The first factor appeared to represent behavioural intentions related to seeking medical advice. Three items had substantial loadings on this first factor, with low loadings on the second and third factors. The second factor appeared to represent behavioural intentions related to maintaining an active lifestyle, with five items loading substantially. Finally, the third factor appeared to best represent intentions related to maintaining a healthy diet, with eleven items loading substantially. Two items loaded substantially one factor and marginally on another.

Table 14

Experiment 3 Pattern Matrix of Rotated Three-Factor Solution of the Behavioural Intentions Items

Item	Pattern Coefficients			Communalities
	1	2	3	
Speak with physician to increase gastrointestinal health	**0.95**	0.05	0.03	0.95
Speak with physician to reduce colorectal risk	**0.94**	0.05	0.01	0.91
Speak to family about risk	**0.82**	0.05	0.11	0.78
Engage in regular physical activity	-0.14	**0.88**	0.10	0.80
Go to the gym	0.001	**0.84**	-0.06	0.67
Engage in cardiovascular activity	0.07	**0.80**	-0.001	0.67
Weight training	0.17	**0.75**	-0.12	0.57

Physical activity with friends	0.09	**0.67**	0.11	0.56
Eat more insoluble fiber	-0.12	0.05	**0.89**	0.76
Eat more soluble fiber	0.06	-0.01	**0.82**	0.70
Decrease saturated fat	0.13	-0.02	**0.69**	0.56
Decrease fast food	0.003	0.10	**0.66**	0.50
Eat more salads	0.01	0.20	**0.63**	0.54
Decrease processed meats	0.009	0.05	**0.59**	0.39
Eat more plant-based protein	0.18	-0.09	**0.56**	0.38
Eat 5-10 servings of fruits & vegetables	0.007	0.23	**0.54**	0.45
Decrease fat	0.09	0.19	**0.51**	0.42
Decrease red meat	0.02	-0.10	**0.50**	0.22
Alcohol in moderation	0.09	-0.09	**0.41**	0.15
Maintain a healthy diet	-0.13	0.36	0.47	0.43
Walk/bike/rollerblade when possible	0.08	0.46	0.28	0.43

Factors 1 and 3 were moderately correlated (r = .40), as were Factors 2 and 3 (r = .41). There was a small correlation between Factors 1 and 2 (r = .24). This three-factor solution is very similar to the three-factor solutions shown in Experiments 1 and 2. Overall, from both a stability and an interpretability standpoint, the three-factor solution appears to provide a good representation of the data.

The rotated solution for the four-factor solution was examined next. This solution was almost identical to the three-factor solution; however, it appeared the diet behavioural intentions factor split into two factors. The first factor appeared to represent

behavioural intentions related to seeking medical assessment, with three items loading substantially. A second factor appeared to represent behavioural intentions related to maintaining an active lifestyle, with five items loading substantially. A third factor appeared to represent behavioural intentions related to maintaining a diet high in fiber, fruits and vegetables, with five items loading substantially. Finally, the fourth factor appeared to represent behavioural intentions related to maintaining a diet low in saturated fat, processed meat and fast food, with three items loading substantially. Three items loaded substantially on one factor and marginally on another; and one item failed to load significantly on any of the factors. This four-factor solution did not emerge in either Experiments 1 or 2. Thus, from an interpretability standpoint, the four-factor solution does represent the data well. However, from a stability standpoint, the four-factor solution does not.

Conclusion. Three of the four factor-number procedures (scree plot examination, parallel analysis, and model fit) supported both three- and four-factor models. Interpretability of both models was examined, and both the three- and four-factor solutions were readily interpretable and made sense conceptually. However, the three-factor model in Experiment 3 was consistent with the three-factor models identified in both Experiments 1 and 2. Given the stability of the three-factor solution across multiple studies, I ultimately decided that a three-factor model best represented the data. Further, the use of a four-factor behavioural intentions scale produced similar findings to the three-factor scale in subsequent analyses.

The results of these factor analyses were important. Once again, I replicated the results of Experiment 1 and 2, finding three factors underlying the behavioural intentions

items. Moreover, I found additional support for the premise that risk-reducing behavioural intentions are multidimensional and comprise several distinct categories.

Based on these results, three behavioural intentions scales were created by summing the items: medical behavioural intentions (items 4, 11, and 16; scores could range from 3 to 33; See Appendix G for items and corresponding item numbers), activity behavioural intentions (items 5, 6, 7, 8, and 9; scores could range from 5 to 55); and dietary behavioural intentions (items 2, 3, 12, 13, 14, 15, 17, 18, 19, 20, and 21); scores could range from 5 to 55). Reliability analyses showed the medical behavioural intentions scale to have excellent reliability (alpha = .94); and both the activity and the dietary behavioural intentions scale to have good reliability (alphas = .89 and .88, respectively). The medical and dietary behavioural intentions scales were moderately correlated (r = .46), as were the medical and activity scales (r = .33), and the dietary and activity scales (r = .42).

Univariate ANOVAs

Manipulation Check

Prior to the main analyses, I conducted a manipulation check to ensure that the perceived efficacy manipulation was effective in altering measured efficacy related to colorectal cancer. To examine these effects, a 2 (self-affirmation condition: self-affirmation versus no self-affirmation) X 3 (manipulated perceived efficacy: high versus low versus baseline) ANOVA was conducted, with measured efficacy as the dependent variable. The ANOVA showed a significant main effect for manipulated efficacy, $F(2, 247) = 127.55$, $p < .001$. Significant differences among the three conditions were evaluated using the 95% confidence intervals. Participants in the high manipulated

efficacy condition reported significantly higher measured efficacy (M = 12.03, CI = 11.56-12.51) compared to participants in the baseline condition (M = 8.30, CI = 7.85-8.75), and participants in the baseline condition reported significantly higher measured efficacy compared to those in the low condition (M = 6.76, CI = 6.29-7.23). These significant findings once again suggested that the manipulation of perceived efficacy was successful in influencing participants' measured efficacy related to risk of colorectal cancer.

The ANOVA indicated no significant main effect for self-affirmation $F(1, 247) = 0.02$, $p = .88$. In Experiments 1 and 2, there was a marginally significant interaction between self-affirmation and manipulated efficacy, such that the effects of the perceived efficacy manipulation were somewhat stronger in self-affirmed participants. However, there was no such interaction between in Experiment 3, $F(2, 247) = 0.64$, $p = .53$.

Unrealistic Optimism

As with Experiments 1 and 2, prior to examining the effects of the independent variables on risk perception, I wanted to confirm that I did in fact, replicate the classic unrealistic optimism effect. I found this effect in the first two experiments, such that participants viewed themselves as being significantly less at risk relative to similar others. Thus, I expected a similar effect for participants in Experiment 3. A paired sample t test was conducted, and as predicted, the results showed that individuals' perceptions of others risk (M = 5.25, SD = 1.80) were significantly greater than perceptions of self risk (M = 4.71, SD = 1.82), $t(252) = 4.44$, $p < .001$, again replicating the unrealistic optimism effect.

Self Risk Perception

The next set of analyses tested the study's core hypotheses. First, I examined the effects of the independent variables on self risk perception, and conducted a 2 (self-affirmation condition: self-affirmation versus no self-affirmation) X 3 (manipulated perceived efficacy: high versus low versus baseline) ANOVA, with the self risk score as the dependent variable.

Experiments 1 and 2 showed no main effect of self-affirmation on risk perception, thus I did not expect a significant main effect for this manipulation in Experiment 3. My hypothesis was confirmed; the ANOVA showed no significant main effect for self-affirmation, $F(1, 247) = 2.31$, $p = .13$, indicating there were no differences in risk perception for participants who self-affirmed versus those who did not self-affirm.

Experiments 1 and 2 also showed no significant main effect of manipulated efficacy on risk perception, thus again, I did not expect a significant main effect in Experiment 3. My hypothesis was confirmed; the ANOVA showed no significant main effect, $F(2, 247) = 0.10$, $p = .91$, indicating there were no significant differences in risk perception among participants in the three manipulated efficacy conditions.

Finally, Experiment 1 did show a significant interaction between self-affirmation and manipulated efficacy, however, when the potential confound in the baseline condition was corrected in Experiment 2, a significant interaction did not emerge. This null effect was replicated in Experiment 3, and there was no significant interaction between self-affirmation and manipulated efficacy, $F(2, 247) = 0.70$, $p = .50$. This provides more support for the supposition that the significant interaction in Experiment 1 was due to a confounded baseline condition.

Behavioural Intentions

The next set of analyses examined the effects of the independent variables on risk-reducing behavioural intentions. As with the first two studies, I conducted a series of three 2 (self-affirmation condition: self-affirmation versus no self-affirmation) X 3 (manipulated perceived efficacy: high versus low versus baseline) ANOVAs, with each of the three behavioural intentions scales as the dependent variable. Experiments 1 and 2 showed no effect for self-affirmation on any of the three behavioural intentions variables, thus I did not expect to find a significant effect in Experiment 3. For the most part, my hypothesis was confirmed: the ANOVA showed no significant self-affirmation main effect for activity behavioural intentions, $F(1, 247) = 1.42$, $p = .24$ or medical behavioural intentions, $F(1, 247) = 1.87$, $p = .18$. However, the effect did approach significance for dietary behavioural intentions, $F(1, 247) = 2.68$, $p = .11$. Estimated marginal means and 95% confidence intervals for each of the three behavioural intentions scales are presented in Table 15. Examination of the means suggests that self-affirmed participants showed

Table 15

Experiment 3 Estimated Marginal Means and 95% Confidence Intervals for Behavioural Intentions

	Activity Behavioural Intentions (95% CI)	Medical Behavioural Intentions (95%CI)	Dietary Behavioural Intentions (95%CI)
No Self-Affirmation	42.07 (40.33-43.81)	17.10 (15.60-18.61)	88.59 (85.14-92.04)
Self-Affirmation	43.59 (41.77-45.41)	15.59 (14.02-17.16)	84.45 (80.85-88.05)

slightly decreased dietary intentions relative to non-affirmed participants, and the means for medical behavioural intentions trended in the manner as well.

I next considered the manipulated efficacy main effect. In Experiment 1, there was a significant main effect for activity behavioural intentions, and a marginally significant main effect for medical behavioural intentions. In Experiment 2, there was a marginally significant main effect for dietary behavioural intentions. Although different effects were found in Experiments 1 and 2, the means trended in the same way, such that participants in the high manipulated efficacy condition showed the greatest intentions to reduce their risk, followed by participants in the baseline, then low conditions. In Experiment 3, the ANOVAs showed no significant manipulated efficacy main effect for activity behavioural intentions, $F(2, 247) = 0.41$, $p = .67$; medical behavioural intentions, $F(2, 247) = 0.59$, $p = .56$; or dietary behavioural intentions, $F(2, 247) = 0.02$, $p = .98$. This result was surprising, as it suggested that manipulated efficacy had no effect on any of the three categories of behavioural intentions.

Finally, I examined the interactive effects of self-affirmation and manipulated efficacy on behavioural intentions. There was no significant interaction between the two independent variables in either Experiment 1 or Experiment 2. This result was replicated in Experiment 3: results of the ANOVAs showed no significant interaction between self-affirmation and manipulated efficacy for activity behavioural intentions, $F(2, 247) = 0.44$, $p = .64$; medical behavioural intentions, $F(2, 247) = 0.13$, $p = .88$; or dietary behavioural intentions, $F(2, 247) = 0.39$, $p = .68$.

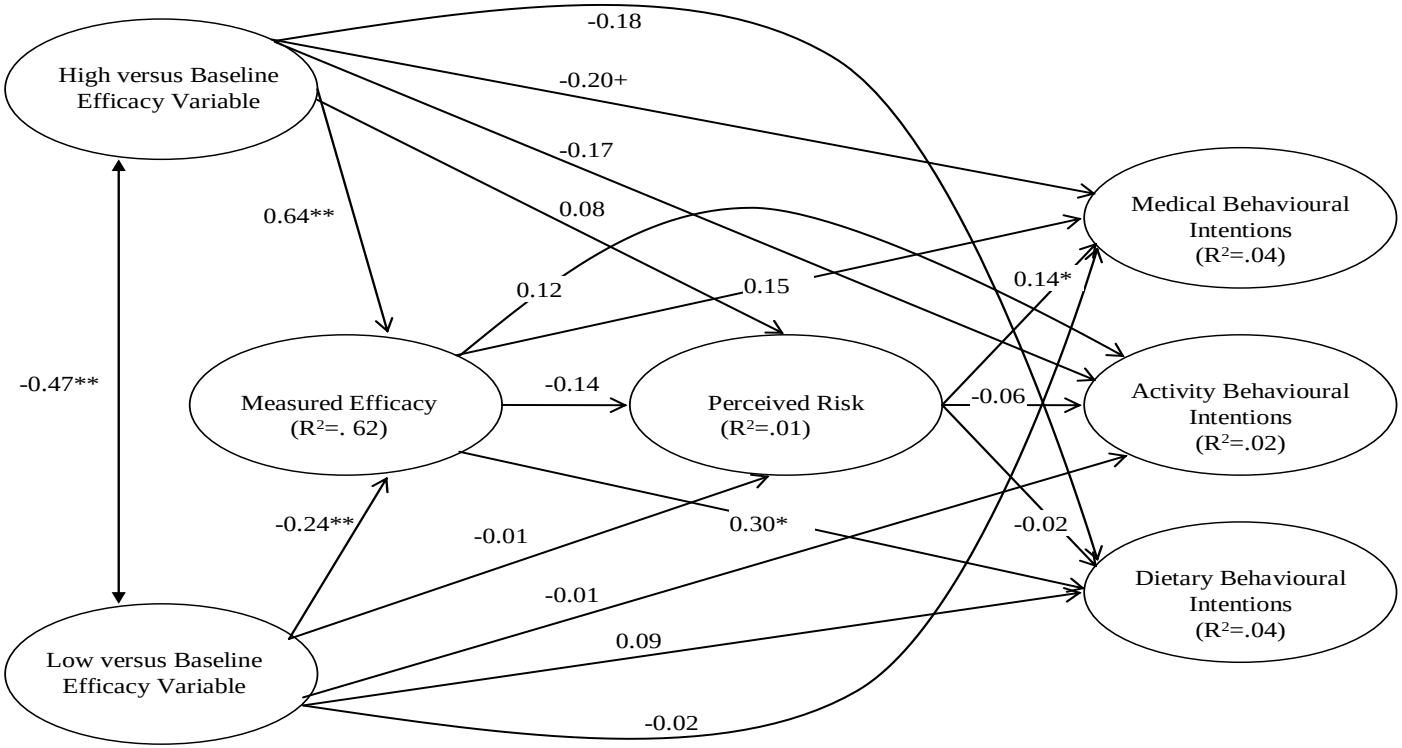
Structural Equation Modeling

Model Specification and Model Fitting

The final set of analyses in Experiment 3 sought to examine the causal relationships among the independent and dependent variables. Once again, I sought to examine the dynamics among the dependent variables, as well as the effects of the manipulated variables on these dependent variables. Moreover, I wanted to determine whether the model of interrelationships proposed and evaluated in Experiments 1 and 2 would produce similar results. I evaluated the same model and using the same SEM procedures as used in the first two studies. The model was fit to a covariance matrix of the 24 measured variables

Model fit. Model fit was evaluated using the same four goodness-of-fit indices used in Experiments 1 and 2. For the proposed model, RMSEA = 0.064 (90% CI = 0.055-0.072, indicating acceptable model fit. In addition, SRMR and NNFI indicated good model fit (SRMR = 0.056; NNFI = 0.95). The χ^2 Test of Goodness-of-Fit was significant χ^2 (234, N = 253)= 500.85, $p < .001$, indicating that the model was not a perfect representation of the data.

Path Estimates. The path estimates for the model are presented in Figure 7. All of the 24 measured variables loaded significantly (ps < .05) on their hypothesized latent variables. For the sake of simplicity, only the structural paths are presented in the figures. First, these results show that assignment to perceived efficacy condition had a significant effect on measured efficacy: both the high efficacy dummy variable and the low efficacy dummy variable paths were significant, which is consistent with Experiments 1 and 2. There was a strong positive relationship between the high efficacy dummy variable and



+ $p \leq .10$, * $p \leq .05$, ** $p \leq .01$

Figure 7. Experiment 3 path diagram

measured efficacy, which indicates that participants in the high manipulated efficacy condition had higher measured efficacy relative to participants in the baseline. Conversely, there was a strong negative relationship between the low efficacy dummy variable and measured efficacy, which indicates that participants in the low manipulated efficacy condition had lower measured efficacy relative to participants in the baseline condition. This result replicates those of my two previous studies, and supports earlier ANOVAs which indicated that my manipulation of perceived efficacy worked quite well in affecting participants' measured efficacy related to colorectal cancer risk.

Next, Experiments 1 and 2 showed a strong negative relationship between participants' measured efficacy and perceptions of risk, which suggested that increased measured efficacy related to heart attack risk caused decreased perceptions of being at risk. Given the two previous studies and the prior correlational literature, I did expect to find a strong negative relationship between these two variables in Experiment 3. This path was not significant, but there was a small relationship in the expected direction. This result was somewhat surprising; however, there could be several reasons for this result. First, the self risk variable used in Experiment 3 comprised only one item, whereas the self risk variable used in Experiments 1 and 2 comprised three items. It may be that the effect was attenuated because the measure of risk was not as powerful. In addition, Experiment 3 used a different health risk paradigm – risk of colorectal cancer – than the first two studies, which examined risk of heart attack. It may be also be that the causal relationship is simply stronger in heart attack risk.

The results of Experiment 1 suggested the presence of a suppression effect for perceived risk: the high efficacy dummy variable had a direct positive effect on perceived

risk, as well as an indirect *negative* effect on perceived risk though a mediator, measured efficacy. The strong negative relationship between measured efficacy and perceived risk was replicated in Experiment 2, but there was not a significant direct effect of the high efficacy dummy variable on perceived risk. I supposed that changes in the baseline manipulated condition in Experiment 2 attenuated the effect found in Experiment 1. In Experiment 3, there was no evidence of a suppression effect. As noted above, there was no significant relationship between measured efficacy and perceived risk, and no evidence of a direct relationship between the high efficacy dummy variable and perceived risk.

With respect to behavioural intentions, there were a number of significant paths in Experiment 3 that did not emerge in either of the first two studies. First, participants' measured efficacy significantly influenced dietary behavioural intentions. Examination of the standardized regression coefficient for this path indicates this is a strong positive relationship, suggesting that higher measured efficacy caused greater intentions to maintain a healthy diet. Although nonsignificant in either of Experiments 1 and 2, this path did trend in the expected direction. Second, perceptions of risk significantly influenced medical behavioural intentions. Examination of the standardized regression coefficient for this path indicates this is a strong positive relationship, suggesting that higher perceptions of risk caused increased intentions to seek medical assessment and advice. Experiment 1 also showed positive relationship between these two variables, however, this path only approached significance ($p = .13$). In Experiment 2, this path did not approach significance, but it did trend in the expected direction.

Discussion

In Experiment 3, I sought to replicate and extend the results of Experiments 1 and 2 to a second health risk paradigm: I examined the same variables and the same relationships using a different health issue: risk of developing colorectal cancer.

With respect to the independent variables, analyses once again indicated that I successfully manipulated perceived efficacy, and this was demonstrated in both the ANOVAs and the SEM analyses. The results of Experiment 1 provided some evidence that I successfully manipulated self-affirmation, but there as no evidence of this in either Experiment 2 or in Experiment 3. Once again, I cannot be certain that self-affirmation was manipulated successfully.

Next, Experiment 3 provided additional support for the premise that risk-reducing behavioural intentions are not one-dimensional, and instead can fall into one of several distinct categories of health-promoting behavioural intentions. Experiment 3 replicated the results of Experiments 1 and 2: my study showed the same three distinct categories of behavioural intentions: behavioural intentions associated with maintaining an active lifestyle; seeking medical advice and assessment; and maintaining a healthy diet. This result is exciting, as I replicated the factor structure in an entirely different health risk paradigm. Overall, I found very clear support for a multidimensional model of risk-reducing behavioural intentions. However, the results of ANOVAs examining the effects of the independent variables on behavioural were somewhat surprising. Experiments 1 and 2 provided some evidence that the manipulation of perceived efficacy significantly affected behavioural intentions. Although differing effects were found in Experiments 1 and 2, the means trended in the same way, such that participants in the high manipulated

efficacy condition showed the greatest intentions to reduce their risk, followed by participants in the baseline, then low condition. In Experiment 3, however, there was no evidence that manipulated efficacy had an effect on any of the three behavioural intentions scales. There was some evidence that self-affirmation affected intentions to maintain a healthy diet, but this effect only approached significance.

Even though the behavioural intentions ANOVAs were somewhat disappointing, when I examined the causal relationships among the variables using SEM, I found several significant paths. First, my results showed a positive relationship between perceptions of efficacy and intentions to maintain a healthy diet. I also found a positive relationship between perceptions of risk and intentions to seek medical advice and assessment. Both findings are consistent with previous literature.

With respect to perceived risk, the ANOVAs in Experiment 3 showed no significant effects of self-affirmation or manipulated efficacy, either in isolation or in conjunction with one another. These results are consistent with Experiments 1 and 2, and provide additional support for the premise that the significant interaction found in Experiment 1 was driven a confounded baseline condition. The SEM results did not provide any support for the suppression effect highlighted in Experiment 1: I did not find a significant relationship between a) high manipulated efficacy and perceived risk, or b) measured efficacy and perceived risk. However, these paths trended in the expected direction, which suggests that while the effect may not be as strong in this third study, the effects may still be meaningful.

Overall, Experiment 3 provided a number of useful results. First, I found further support for the premise that risk-reducing behavioural intentions can fall into several

distinct categories of health promoting behaviour. In addition, the results of the SEM analyses in Experiment 3 once again highlighted value of exploring the dynamics of such variables in one coherent model.

Chapter 5: Meta-Analytic Study

Up to this point, I have examined a number of effects in Experiments 1, 2, and 3. Across each of these studies, I have found some consistent effects, some inconsistent effects, and some consistent trends. This raises an important issue: given the vast number of analyses and effects, how many of these effects are due to chance, and how many effects show enough consistency that they would merit attention? Meta-analysis allowed me to examine this issue.

In conducting my meta-analyses, the initial step was to determine which effects to meta-analyze. I first considered the behavioural intentions factor analyses. With factor analysis, there is no test of significance *per se*, and moreover, I found reasonably consistent results across each of the three studies. Thus, I did not feel that meta-analysis would provide me with much additional information. Next, I considered the results of the ANOVAs and tests of the unrealistic optimism effect. A summary of these results across the three studies is presented in Table 16, and both significant effects and effects that approached significance are included. As shown in the table, I demonstrated the unrealistic optimism effect across the three studies, and this effect was consistent and robust. Thus, I did not feel that meta-analysis was necessary. Next, I considered the perceived efficacy manipulation check, that is, the effect of manipulated efficacy on measured efficacy. Once again, at the individual study level, this effect was consistent and robust, and my previous analyses indicated that I can be certain that perceived

efficacy was manipulated successfully. Thus, I did not feel that meta-analysis was warranted.

Table 16

Summary of Tests of the Unrealistic Optimism Effect and ANOVAs Across Experiments

	Experiment 1	Experiment 2	Experiment 3
Unrealistic Optimism	✓✓	✓✓	✓✓
Measured Efficacy			
SA main effect			
MPE main effect	✓✓	✓✓	✓✓
SA x MPE interaction	✓	✓	
Risk Perception			
SA main effect			
MPE main effect			
SA x MPE interaction	✓✓		
Medical BI			
SA main effect			
MPE main effect	✓		
SA x MPE interaction			
Activity BI			
SA main effect			
MPE main effect	✓✓		
SA x MPE interaction			

Dietary BI	
SA main effect	
MPE main effect	✓
SA x MPE interaction	

Note. SA = self-affirmation; MPE = manipulated perceived efficacy; BI = Behavioural intentions

✓✓ = $p < .05$; ✓ = $p < .10$

I also considered the effects of the two independent variables on the primary dependent variables (i.e., risk perception and behavioural intentions). At the individual study level, the results of the ANOVAs showed some effects, but a) there were no consistent significant effects and b) there was no consistency in the direction of the effects. Meta-analysis is most useful when there is consistency at the directional level; if effects fluctuate in different directions across studies, it is unlikely that meta-analysis would produce a significant effect. Therefore, I did not think it was useful to conduct meta-analyses of these effects.

Finally, I considered the SEM analyses. At the individual study level, I found a number of significant effects, some of which were consistent and significant across each of the three studies. However, I also found a number of effects that were significant in only one or two of the studies, but that trended in the same direction in the other studies. These results appeared to be good candidates for meta-analysis, as aggregating the effects across the three studies would allow me to determine whether the effects represented meaningful effects, or whether they were simply due to chance. Thus, after reviewing the results of each of the analyses across the three studies, I decided that the meta-analytic study would only focus on the SEM results.

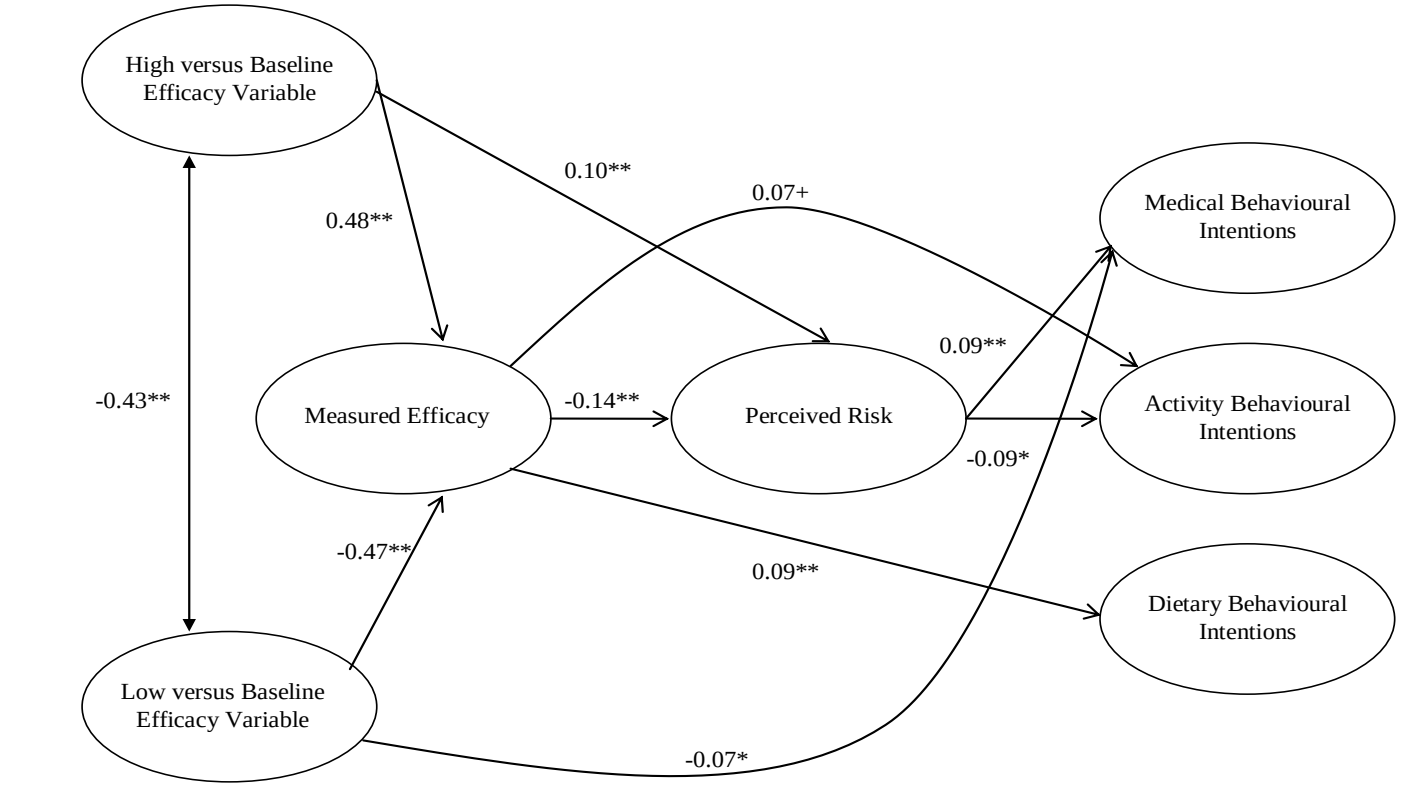
The next step was to determine which paths to meta-analyze. I examined a number of paths in the structural equation model, but because of the sheer number of paths, from an efficiency standpoint I did not want to meta-analyze all possible paths. Instead, I chose to meta-analyze only those paths that showed some promise in producing significant meta-analytic results, that is, paths that occurred with sufficient regularity that any significant result would likely not be due to chance. Therefore, in choosing paths to meta-analyze, I set two criteria. To be considered for meta-analysis I required that a) it reach conventional levels of significance in at least one of the three studies; or that b) if there was no significant path, the path showed consistency in direction among all three studies. Ultimately, eleven paths met at least one of these criteria: nine paths met this first criterion, and two paths met the second.

Meta-analysis was performed using Advance BASIC Meta-Analysis software (Mullen, 1989). To calculate each effect, the significance test for each parameter (a *t* value, which approximates a *z* score distribution in SEM) with its corresponding sample size from each of the three studies was used. Results of the meta-analyses were interpreted using three statistics. First, I computed the overall *z* value across the three studies, which reflects statistical significance at the meta-analytic level. This would allow me to determine whether the overall parameter was significant. Second, I computed average effect size across the three studies, which was indicated by *r*. Third, I considered the chi square test of effect size, which would allow me to determine whether an effect size fluctuated significantly among the three studies, or whether it was homogenous. The paths chosen for meta-analysis, along with the corresponding *z* tests, *r*s, and chi-square tests of effect size, are presented in Table 17. Figure 8 presents a summary of these paths,

Table 17

Meta-Analytic Significance Tests, Effect Sizes, and Tests of Homogeneity for Structural Equation Modeling Path Coefficients

Path	Z, *p*	*r*	Chi Square, *p*
High versus Baseline Efficacy Variable ↔ Low versus Baseline Efficacy Variable	-12.05, $p < .01$	-0.43	0.13, $p = .93$
High versus Baseline Efficacy Variable → Measured Efficacy	12.48, $p < .01$	0.48	61.78, $p < .01$
Low versus Baseline Efficacy Variable → Measured Efficacy	-12.45, $p < .01$	-0.47	39.86, $p < .01$
Measured Efficacy → Perceived Risk	-3.95, $p < .01$	-0.14	2.77, $p = .25$
High versus Baseline Efficacy Variable → Perceived Risk	2.79, $p < .01$	0.10	1.99, $p = .37$
Low versus Baseline Efficacy Variable → Perceived Risk	-0.92, $p = .36$	<0.01	0.27, $p = .87$
Low versus Baseline Efficacy Variable → Medical Behavioural Intentions	-2.03, $p = .04$	-0.07	1.48, $p =$. 48
Measured Efficacy → Dietary Behavioural Intentions	2.61, $p < .01$	0.09	0.83, $p = .66$
Measured Efficacy → Activity Behavioural Intentions	1.79, $p = .07$	0.07	0.04, $p = .98$
Perceived Risk → Activity Behavioural Intentions	-2.38, $p = .02$	-0.09	1.23, $p = .54$
Perceived Risk → Medical Behavioural Intentions	2.58, , $p < .01$	0.09	1.14, $p = .57$



+ $p \leq .10$, * $p \leq .05$, ** $p \leq .01$

Figure 8. Summary of significant meta-analytic paths

along with the associated effect sizes. The path model presents only those paths that reached meta-analytic significance.

The first path chosen for analysis was the correlation between the two dummy coded variables, the high efficacy dummy variable and the low efficacy dummy variable. These variables represented the manipulation of efficacy. Because of the nature of the variables, they were guaranteed to be correlated with one another. Moreover, this path was significant in each of the three individual studies. Thus, I did expect to find significant meta-analytic effect. This was confirmed; I found a significant effect, with a strong negative effect size. In addition, the chi square test of effect size indicated that I could not reject the null hypothesis that the effect sizes were homogenous across the three studies.

Next, I examined the effect of the perceived efficacy manipulations on measured efficacy. I first considered the effect of the high efficacy dummy variable. At the individual study level, there was a strong positive effect of the high efficacy dummy variable on measured efficacy across each of the studies, thus I did expect a significant meta-analytic effect. This was confirmed; I found a significant effect, with a strong positive effect size. Interestingly, the chi square test of effect size was significant, which suggests that there was significant fluctuation in the magnitude of the effect among the three studies. This result is not surprising if one considers the individual coefficients from the SEM analyses; there is a strong positive effect in all three studies, however, it appears that the effect of the high efficacy dummy variable was strongest in Experiment 3. I next considered the effect of the low efficacy dummy variable on measured efficacy. Once again, I expected a significant meta-analytic effect, given the strong negative effect

shown in each of the three individual studies. This was confirmed: results of the meta-analysis showed that there was a significant effect, with a strong negative effect size. The chi square test of effect size was significant, which once again suggests that there was significant fluctuation of effects among the three studies. As with the high efficacy dummy variable, the individual coefficients indicated that there was a strong effect in all three studies. However, it appears the effect of the low efficacy dummy variable was strongest in Experiment 2.

Up to this point, the meta-analyses have confirmed the significance of effects of which I was already certain. With the remaining meta-analyses, I examined paths that were significant in only one or two of the studies, thus there was much more ambiguity as to whether these paths represented meaningful effects, or whether they were due to chance fluctuations in the data. Therefore meta-analysis provided more substantive information, as it allowed me to clarify my effects.

The first of these paths was the effect of measured efficacy on perceived risk. This path was negative and significant in Experiments 1 and 2, but not in Experiment 3, although it did trend in the expected direction. When meta-analyzed, the effect easily reached significance. Examination of the *r* indicates that this was a modest, negative effect size. The chi square test of effect size was not significant, indicating that I could not reject the null hypothesis that the effect sizes were homogenous across the three studies. This result provides good support for the negative influence of perceived efficacy on risk perception. This relationship has been documented in previous correlational studies, but rarely has it been examined in the context of a larger SEM study, where the effects of other variables can be controlled.

Next, I considered the effect of the high efficacy dummy variable on perceived risk. This represented a novel effect, one that has not been addressed in previous literature. In Experiment 1, I found a significant and positive relationship between the two variables. In Experiments 2 and 3, this path was not significant; however, the coefficients trended in the same direction. Results of the meta-analysis showed a significant effect, with a small positive effect size. The chi square test of effect size was not significant, indicating that I could not reject the null hypothesis that the effect sizes were homogenous. This finding provides further support for the suppression effect noted previously in the results of the individual SEM analyses. The meta-analytic results confirmed that at one level, attempts at increasing efficacy directly caused increased perceptions of risk. However, these attempts also appear to have offset this effect, because as indicated above, they also caused increased measured efficacy, which in turn, led to *decreased* perceptions of risk. This finding suggests that the manipulation of perceived efficacy can have much more complex effects, particularly on risk perception, than simply affecting measured efficacy.

I next considered the effect of the low efficacy dummy variable on perceived risk. This path was not significant in any of the three studies, but all paths trended in the negative direction. Results of the meta-analysis were not significant. Thus, while the manipulation to increase perceived efficacy did have a direct and significant relationship with risk perception, the manipulation to decrease perceived efficacy did not.

Next I considered the effect of the low efficacy dummy variable on medical behavioural intentions. This path was significant and negative in Experiment 1, but was not significant in Experiments 2 and 3, although the coefficients did trend in the same

direction. Results of the meta-analysis showed a significant effect, with a small negative effect size, and the chi square test of effect size indicated that I could not reject the null hypothesis that the effect sizes were homogenous. Thus, this result suggests that attempts at decreasing manipulated efficacy can cause decreased intentions to seek medical advice and assessment. This result is somewhat sensible, as it would suggest that an individual who is made to feel as though they cannot control their health risk would not bother getting further information about their risk because they are not able to control it.

I next considered the effect of measured efficacy on dietary behavioural intentions. This path was significant and positive in Experiment 3, but was not significant in Experiments 1 and 2, although once again the coefficients trended in the same direction. Results of the meta-analysis showed a significant effect, with a small positive effect size, and the chi square test of effect size indicated that I could not reject the null hypothesis that the effect sizes were homogenous. Overall, this finding suggests that increased perceptions of efficacy can cause increased intentions to maintain a healthful diet. This result is in line with previous literature that has shown that increased perceived efficacy is related increased risk-reducing behavioural intentions and behaviours.

The effect of measured efficacy on activity behavioural intentions was considered next. This path was not significant in any of the three studies, however, the path consistently trended the positive direction. Results of the meta-analysis showed a marginally significant effect (p = .07), with a small positive effect size. Once again, the chi square test of effect size was nonsignificant, which indicated that I could not reject the null hypothesis that the effect sizes were homogenous. This finding suggests that, while not quite significant, increased perceptions of efficacy can cause increased

intentions to maintain an active lifestyle. Once again, this result is in line with previous literature.

The next meta-analysis examined the effect of perceived risk on activity behavioural intentions. This path was significant and negative in Experiment 2, and but was not significant in either Experiments 1 or 3, although the coefficients trended in the same direction. Results of the meta-analysis showed a significant effect, with a small negative effect size, and the chi square test of effect size indicated that I could not reject the null hypothesis that the effect sizes were homogenous. Overall, this finding suggests that increased risk perception can cause decreased intentions to maintain an active lifestyle. This was an interesting result, as it somewhat contradicts the common assumption that previous research makes, that is, that feeling more at risk for a given health issue causes individuals to be more likely to want to engage in behaviour that would reduce their risk. Thus, this result highlights the potential problem with the previous literature, which typically does not differentiate between different types of risk-reducing behavioural intentions.

The final meta-analysis examined the effect of perceived risk on medical behavioural intentions. This path was significant and positive in Experiment 3, but was not significant in Experiments 1 or 2, although the coefficients trended in the expected direction. Results of the meta-analysis showed a significant effect, with a small positive effect size, and the chi square test of effect size indicated that I could not reject the null hypothesis that the effect sizes were homogenous. Thus, this result suggests that increased risk perception can cause increased intentions to seek medical assessment and advice. Again, this result makes sense and is in line with previous literature, which has

demonstrated that individuals who feel more at risk are more likely to take steps to reduce their risk.

The results of the meta-analyses provided a number of significant paths, and overall, the results of the meta-analytic study were quite useful. At the individual study level, when I compared many of these paths across the three studies, there was inconsistency in the extent to which the path reached conventional levels of significance. In many cases, the path was significant in only one of the three studies, but trended in the expected direction in the remaining studies. Thus, it was unclear as to whether the effects were meaningful, or due to sampling error in the data. The meta-analyses showed that in most cases, these were, in fact, meaningful and significant effects. In every case where there was at least one path among the three studies that reached significance, the path met significance at the meta-analytic level. The results of the meta-analyses were also useful in that they demonstrated that in general, the significant meta-analytic effects were not heterogeneous across the three studies. Ironically, the only paths where heterogeneity of the effect emerged were two paths that were the most consistent in the individual SEM analyses.

Overall, the results of the meta-analytic study showed that there was far more consistency among the SEM analyses than one might think, and they allowed me to determine those paths that I can speak about with some confidence. Although many of the significant paths showed somewhat modest effect sizes, they have a number of theoretically interesting implications.

Chapter 6: General Discussion

Summary of Results

First, factor analyses of the risk-reducing behavioural intentions items indicated three distinct categories of intentions: behavioural intentions associated with maintaining an active lifestyle, seeking medical advice and assessment, and maintaining a healthy diet. I found support for this three-factor model of intentions in the context of two distinct health risk paradigms (heart attack and colorectal cancer) across each of the three studies. Overall, these results provide strong evidence for a multidimensional model of risk-reducing behavioural intentions.

The effects of my two experimental manipulations were somewhat mixed. First, there was clear evidence, across each of the studies, that I successfully manipulated perceived efficacy. This finding was demonstrated in both the ANOVAs and SEM analyses, and I am confident in the manipulation of this independent variable. On the other hand, there was little evidence that I successfully manipulated self-affirmation across the three studies. I did use a self-affirmation manipulation commonly used by self-affirmation researchers, but because there is no established manipulation check, it is difficult to determine whether this manipulation was successful. Across each of the three studies there was virtually no effect of self-affirmation on any of the dependent variables, thus I cannot be certain that I successfully manipulated this independent variable.

Next, my analyses indicated that I replicated the classic unrealistic optimism effect: participants consistently viewed themselves as being significantly less at risk relative to similar others. This effect emerged in each of the three studies, within the context of both health risk paradigms.

The results of the ANOVAs examining the effects of my manipulations on the primary dependent variables were generally disappointing. After correcting for a confounded baseline manipulated efficacy condition, I found virtually no effect of manipulated perceived efficacy or self-affirmation on risk, either in conjunction with, or independent of one another. However, as subsequent SEM analyses indicated, my null findings regarding perceived efficacy's effect on risk was likely due to a suppression effect. With respect to behavioural intentions, I did find some significant effects of manipulated efficacy in expected directions; however, these findings were not consistent across any of the studies.

Even though the ANOVAs provided somewhat disappointing results, the results of the SEM analyses highlighted a number of interesting relationships among the independent and dependent variables. In addition to confirming the effectiveness of my perceived efficacy manipulations on participants' perceptions of efficacy, these analyses also suggested the presence of a suppression effect on risk perception. I found that my manipulation designed to increase perceived efficacy had direct positive effects on perceptions of risk, but also, that this manipulation had indirect *negative* effects on perceptions of risk, with measured efficacy acting as a mediator. Now, these effects were not consistently significant across the three studies, making it difficult to conclude that this was a meaningful result. However, the relationships among the variables all trended in the same direction in the nonsignificant paths, which suggested that the effects were due to more than chance fluctuations among the data. In addition to the effects of the variables on risk, the SEM analyses also highlighted a number of significant paths within

individual studies. But once again, these paths were not consistently significant across studies, even though there were consistent trends.

My final set of analyses, the meta-analytic study, allowed me to clarify the relationships suggested by the SEM analyses. In fact, the meta-analyses indicated that there was far more consistency among the SEM analyses than initially thought. First, these analyses once again confirmed the effectiveness of my perceived efficacy manipulations. Second, the results confirmed the existence of the perceived risk suppression effect suggested at the individual study level. Third, even though the effect sizes were modest, the results also showed a number of significant and interesting relationships among the independent and dependent variables. The manipulation designed to decrease perceived efficacy had a direct negative effect on behavioural intentions related to seeking medical advice and assessment. In addition, participants' perceptions of efficacy were shown to affect two types of behavioural intentions: there was a positive relationship with both intentions related to maintaining a healthy diet, and with intentions to maintaining an active lifestyle. Interestingly, perceptions of risk were shown to affect behavioural intentions differently: there was a positive relationship with medical behavioural intentions, but a negative relationship with activity behavioural intentions.

Implications

Dimensionality of Risk-Reducing Behavioural Intentions

The results of my series of three studies advanced the health psychology literature in several important ways. First, I found strong evidence for a multidimensional model of risk-reducing behavioural intentions: the results of the factor analyses clearly identified separate and distinct factors underlying the intentions items. As highlighted in the

introduction, previous studies examining the effects of self-affirmation on risk-reducing behavioural intentions have generally treated intentions as a single factor, by either using only one item to measure behavioural intentions (thereby implicitly assuming one dimension), or, by using several items and aggregating them into one total score. Thus, any comparison of results among these previous studies is problematic, as in a sense, one might be comparing apples and oranges. Moreover, interventions designed to increase behavioural intentions may affect different domains of intentions differently. The results of the SEM analyses provided strong support for this notion, as each of the three categories of behavioural intentions had very distinctive predictors. For example, perceptions of risk had a positive relationship with medical behavioural intentions, a negative relationship with activity behavioural intentions, and no significant relationship with dietary behavioural intentions. Given the very different effects of risk perception on these three categories of intentions, had they been aggregated and treated as a single behavioural intentions factor, I likely would have seen no effect of perceived risk on intentions whatsoever.

The finding that the behavioural intentions items were multidimensional may also help explain some of the discrepancies highlighted in the introduction. Previous self-affirmation studies have shown inconsistent results in regards to the effect of self-affirmation on both risk-reducing behavioural intentions and behaviours. These inconsistencies may be due to the fact across studies, the effects of self-affirmation on different domains of health-promoting behaviour are being examined. Thus, the studies' manipulations may have simply affected different domains of risk-reducing behavioural intentions, which would explain the inconsistent results.

Overall, the results of the factor analyses and the SEM analyses have important implications for studies examining the effects of interventions targeted at altering risk-reducing behavioural intentions (and potentially risk-reducing behaviours). Researchers must consider that their intervention may affect different domains of health promoting behaviour, as the intervention may have varied effects depending on the domain of intention being assessed. In addition, my results highlight the importance of assessing multiple domains of behavioural change. Many studies only assess a single intention (e.g., intention to quit smoking) or behaviour (e.g., number of cigarettes), and this is not unreasonable, as health interventions often target the reduction of a very specific risk behaviour, such as cigarette smoking. Thus, in cases such as this where the context is quite narrow, my findings may not be as relevant. However, other health interventions attempt to target broader health actions, for example, behaviour changes following a heart attack. Healthcare providers will often promote multiple behavioural changes, including dietary changes and increases in physical activity following such an event. Thus, when targeting broader health actions such as these, it is important recognize that there are multiple categories of behaviours, perhaps with different antecedents and consequences.

For example, if one were to devise an intervention designed to increase risk perception and acceptance in an at-risk population (e.g., individuals who have high blood pressure and are at risk for having a heart attack or stroke), based on my results, one would have to consider that if the intervention is effective, increased risk perception may result in increased risk-reducing behaviour such as seeking medical assessment and advice, but, that individuals may also decrease other aspects of risk-reducing behaviour such as their activity level. Thus, in designing such an intervention, researchers would

have to considering adding a component to their intervention to address any potential decreases in risk-reducing behaviour.

Even though my studies examined the dimensionality of behavioural intentions and not behaviours, it is not unreasonable to assume that a multidimensional model of risk-reducing behaviours also exists. My results imply that in considering only one specific aspect of health promoting behaviour, researchers might also be losing important information regarding the effects of their interventions on other health promoting behaviours.

Interrelationships Among Efficacy, Risk Perception, and Behavioural Intentions

The results provided by both the SEM and meta-analyses represent a significant contribution to the health psychology literature. Variables such as perceived risk and perceived efficacy are well-studied within this literature, and are included in several theories of behaviour change (e.g., Janz & Becker, 1984; Rogers, 1975). Manipulations of perceived efficacy and self-affirmation have also received attention within the literature (e.g., McMath & Prentice-Dunn, 2005; Sherman et al., 2000). However, for the most part, these variables have been studied in relative isolation of one another; to my knowledge, research has not analyzed these variables together in a single model. Previous studies have not assessed the dynamics among the dependent variables of risk, measured perceived efficacy, and risk-reducing behavioural intentions, nor have they considered the effects of the manipulated independent variables on these dependent variables in such a manner. By examining my variables in this way, I found a number of theoretically important relationships.

Perceived Risk. First, the meta-analyses of paths affecting perceived risk showed the presence of a suppression effect. I found that at one level, attempts at increasing efficacy directly caused increased perceptions of risk (relative to a control condition), but at another level, these attempts indirectly caused *decreased* perceptions of risk via a mediating variable, perceptions of efficacy. Attempts at increasing efficacy also caused increased measured efficacy, which in turn, led to decreased perceptions of risk. The fact that attempts at increasing efficacy directly increased perceptions of efficacy is not surprising given that it was the targeted variable. Further, given the extensive literature supporting a negative relationship between perceived efficacy and risk, my replication of this relationship is once again, not surprising. However, the fact that the manipulations to increase perceived efficacy directly caused increased risk perception was somewhat unexpected. Given the correlational research, one would predict that these attempts to increase efficacy would cause decreased risk perceptions. So the question remains, why did this manipulation cause an increase in risk perception?

It may not have been the emphasis of controllability of risk that caused the increase, but perhaps it was the discussion of *risk factors* that influenced individuals' risk perception. Previous research has highlighted the influence of thinking about one's standing on risk factors in increasing risk perception (Weinstein, 1982; Weinstein & Klein, 1995). In the baseline condition, participants read about the health issue, including symptoms and diagnosis, but there was no mention of any risk factors. In the high manipulated efficacy condition, participants read about both controllability of risk and controllability of risk factors for the health conditions (e.g., diet, weight, activity). It could be that participants in the high manipulated efficacy condition evaluated

themselves on these risk factors, and perhaps concluded that they fell short, which in turn, increased their perception of risk. This effect would not necessarily have occurred in the low manipulated efficacy condition; even though there was a discussion of risk factors in this condition, uncontrollable risk factors were emphasized. Moreover, these risk factors were variables that participants would not necessarily be able to evaluate as easily factors such as diet and activity. The low condition discussed risk factors such as genetics, and specific amino acids and proteins, which would be difficult for participants to assess.

Overall, my results examining perceived risk suggest that manipulations designed to target perceived efficacy can have much more complex effects than simply affecting measured efficacy. Had it not been for the SEM analyses, I might have concluded that manipulated efficacy did not affect risk perception at all, given the null results of the ANOVAs. This finding has implications for researchers examining risk perception, as it highlights the importance of considering the effects of multiple variables and their interrelationships on perceived risk.

Risk-Reducing Behavioural Intentions. As noted above, the SEM and related meta-analytic results highlighted the importance of considering multiple domains of risk-reducing behaviours: I found that both independent and dependent variables affected the three categories of risk-reducing behavioural intentions, and these results differed for each category of behavioural intentions. Even though the effects sizes for these relationships were modest, they do have theoretically interesting implications.

I found that my manipulation designed to decrease perceived efficacy made individuals less likely to intend to seek medical advice and assessment. This finding

makes some sense conceptually; if individuals are made to feel as though their health risks are difficult to control, they may not feel it necessary to assesses this risk or speak with their physician because they do not feel that it would affect their overall risk status.

Next, I found that perceptions of efficacy affected two of the three types of behavioural intentions: I found that higher perceptions of efficacy were associated with both increased intentions to maintain a healthy diet, and increased intentions to maintain an active lifestyle. Again, this result makes sense conceptually, and is in line with previous literature. As described in the introduction, studies have shown that in general, individuals who feel a greater sense of control over their health risk are more likely to engage in risk-reducing behaviours. What is most interesting about these results is that perceptions of efficacy did not affect all three categories of risk-reducing behavioural intentions in the same manner; intentions to seek medical advice and assessment were not significantly affected by perceptions of efficacy. Once again, this underscores the importance of considering the multidimensional nature of risk reducing behaviour. One might wonder why only dietary and activity intentions were affected by perceptions of efficacy. It may be that individuals who feel as though they can control their health risk will intend to engage in those behaviours they know will reduce their risk (i.e., diet and exercise), and because of these intentions, do not feel that seeking medical advice and assessment is necessary because they already intend to take steps to reduce their risk.

Finally, I found several effects for perceptions of risk and behavioural intentions. First, I found that higher perceptions of risk were associated with increased intentions to seek medical advice and assessment. This makes sense conceptually, as it would imply that individuals who feel more at risk would seek out medical attention in order to reduce

their risk. Conversely, I found that higher perceptions of risk were associated with *decreased* intentions to maintain an active lifestyle. This finding was somewhat puzzling, as it is not in line with what previous literature would suggest, that is, that increased perceptions of risk would motivate an individual to reduce their risk. This effect could be due to the nature of my sample: young undergraduate university students, which is a population that generally tends to be more active than other others. This finding may have occurred because after perceiving a high risk, participants might have concluded that perhaps their active lifestyles were not as beneficial as they might have initially thought in promoting health. In a sense, the effect of increased risk perception may have backfired and these participants may have given up and decreased their intentions to maintain their already high level of activity. The fact that dietary intentions were not affected in this manner may not be surprising, as even though activity is generally higher in university students, their diets are generally not as healthful.

Overall, the results of my analyses examining the dynamics and interrelationships among manipulated efficacy, perceptions of efficacy and risk, and the different types of risk-reducing intentions have important clinical implications. First, as noted above, the fact that the manipulation of efficacy had more complex effects than anticipated is important. In developing interventions to target health risk behaviour, researchers and clinicians must consider the potentially complex interplay among variables affecting risk perception and behaviours. As seen in my results, manipulations can have both direct and indirect effects on targeted variables, which ultimately can lead to unexpected effects. Second, the fact that I found differing effects of targeted variables on the three categories of risk-reducing behaviours has implications for interventions as well. Researchers and

clinicians targeting risk-reducing behaviour need to be aware that their intervention may affect different types of health promoting behaviours differently. Therefore, any intervention designed to promote health risk-reduction needs to consider a) the possibility of multiple domains of health promoting behaviour, and b) the possibility that interventions may have differing effects on these domains of behaviour.

Self-Affirmation

As described above, my program of research offers several important contributions to the health psychology literature. However, my studies also fell short in several ways. One major issue not clarified by my study was the effect of self-affirmation. There was essentially no effect of the self-affirmation manipulation on any of the dependent variables. This was surprising given the previous literature examining the use of self-affirmation in promoting risk acceptance and behavioural change, and caused me to question whether self-affirmation was successfully manipulated in any of my three studies.

When one considers the previous literature examining self-affirmation's use in promoting risk acceptance, it may not be as consistent as one might think. First, I could only find three studies that examined risk perception directly (Harris & Napper, 2005; Harris et al., 2007; Sherman et al., 2000), and these results were not consistent: only two of the three studies found that self-affirmed participants reported higher perceptions of risk. Other self-affirmation studies have examined its effect on message acceptance (e.g., "it is important to quit smoking"; "there is a relationship between breast cancer and caffeine"; Armitage et al., 2008; Sherman et al., 2000), message derogation (van Koningsbruggen & Das, 2009), and biased message processing (Reed & Aspinwall,

1998), and these studies do provide evidence that self-affirmation increases message acceptance and decreases message derogation and biased message processing. While these variables are important to consider, they are not necessarily indicative of accepting oneself as being at risk. Thus, the null findings for the effect of self-affirmation on risk perception may not be reflective of unsuccessful manipulation, and instead could be due to a lack of effect of self-affirmation on risk perception in general. At this point, there are so few studies examining this relationship that it is difficult to determine whether this is the case, but it may at least partially explain the null results.

There is a larger body of research examining the use of self-affirmation in increasing risk-reducing behavioural intentions and behaviours. As noted above, there have been some inconsistencies within these studies, but there have been a number of significant effects, such that more often than not, self-affirmed individuals report greater intentions to engage in a behaviour to reduce their risk (e.g., Armitage & Conner, 2000; Harris & Napper, 2005; Jessop, Simmons, & Sparks, 2009; Sherman et al., 2000), with several other studies showing decreased intentions, or no differences at all (e.g., Epton & Harris, 2008; Reed & Aspinwall, 1998). However, each of these studies assessed very specific, often single-item behavioural intentions (e.g., intention to quit smoking), which makes it difficult to compare the results of my studies with these results, given that my measure of behavioural intentions reflects an aggregate of multiple behavioural intentions items, assessing a number of different behaviours.

Methodological Limitations

One limitation of the three studies is the nature of the sample. This research was conducted on undergraduate university students, a population that is predominantly

young, female, healthy, intelligent, of upper socioeconomic status, and English speaking. Thus, the generalizability of my results is unclear. With respect to methodology, there are several considerations and perhaps areas of improvement. First, the materials used in the three studies (e.g., manipulated efficacy passages and the RAQ) were quite lengthy. In designing the materials this was done purposefully, in order to ensure that perceived efficacy was manipulated properly, and in order to increase the believability of the study. However, in retrospect, the lengthy nature of my materials could have hindered how much participants attended to all of the information. Next, the order of the dependent measures could have decreased the power of the threat for subsequent measures. That is, having participants consider the risk of others (and be unrealistically optimistic by rating themselves as at lower risk compared to others) prior to completing the measures of intentions could have decreased the power of the threatening risk information for the subsequent dependent measures (i.e., behavioural intentions items).

Directions for Future Research

The present study represents a significant contribution to advancing the health psychology literature, and provides valuable information regarding the nature of risk-reducing behavioural intentions, as well as the interrelationships between constructs commonly used in health behaviour research. However, I do feel that there is a need for more research.

Dimensionality of Risk-Reducing Behavioural Intentions

My study provided strong evidence for the multidimensional nature of risk-reducing behavioural intentions, and highlighted the importance of considering multiple domains of behaviour change in health risk research. I found three distinct domains of

risk-reducing behavioural intentions – seeking medical advice and assessment, maintaining a healthy diet, and maintaining an active lifestyle – in two health risk paradigms, risk of heart attack and colorectal cancer. My participants were all young undergraduate university students, which is a population that is generally healthy and at lower risk for developing serious and chronic health problems. Moreover, temporally, their risk feedback was future-oriented (i.e., risk prior to the age of 50). Therefore, it would be important to examine whether a similar pattern of risk-reducing behavioural intentions would emerge using health risk paradigms that are more immediate for a university-age population, for example, risk of alcohol poisoning or risk of contracting a sexually transmitted disease. In addition, the dimensionality of risk-reducing intentions for chronic disease (e.g., cancer, diabetes, heart disease) should be examined in other populations, for example, in individuals who are closer to an age where such illnesses are more frequent.

Future studies should also examine the potential dimensionality of risk-reducing behaviours. Research has demonstrated that behavioural intentions have strong correlations with behaviour, and that experimental changes in behavioural intentions frequently lead to changes in behaviour (see Webb & Sheeran, 2006, for a review). Thus, it is reasonable to assume that there are distinct categories of risk-reducing behaviour as well. It would be worthwhile to examine whether multiple categories of health promoting behaviour exist for behaviours, and if so, whether this structure is similar to that of behavioural intentions. As with intentions, such a finding would have important clinical implications: interventions designed to increase risk-reducing behaviours may affect different domains in different ways.

Interrelationships Among Efficacy, Risk Perception, and Behavioural Intention

First and foremost, research needs to continue to investigate the interrelationships among perceived efficacy, risk, and intention. To my knowledge, my series of studies represents the first time that the complex interplay among such variables has been examined, and is a foundation from which other investigations can begin. Future studies should examine whether this model of relationships exists in other chronic health risk paradigms (e.g., type 2 diabetes, other types of cancer), and in different populations (e.g., in older participants, or in participants who have already developed the disease).

Future studies might also consider the inclusion of additional variables in their investigations of these interrelationships. Researchers have noted the importance of other variables involved in health risk and health behaviour, including severity of the threat, personality factors (e.g., Floyd, Prentice-Dunn, & Rogers, 2000), affective attitudes towards performing a health behaviour (e.g., Lawton, Conner, & McEachan, 2009), and whether an individual perceives that important others think the behaviour should be performed (e.g., Albarracin, Johnson, Fishbein, & Muellerleile, 2001), and such variables have been emphasized in several models of health behaviour (e.g., protection motivation theory, theory of reasoned action). Thus, the results of my study should be replicated and extended by including such variables in subsequent models.

Self-Affirmation

The use of self-affirmation in promoting health behaviour shows a great deal of promise, but more research is necessary. Primarily, the effect of self-affirmation on risk perception needs to be clarified. Only a handful of studies have examined the use of self-affirmation in increasing perceptions of risk, and while several studies have shown that

self-affirmation can increase an individual's perception of their risk, other studies (including ours) found null effects. Given the positive relationship between perceived risk and risk-reducing behaviour, a clear understanding of self-affirmation's effect on risk perceptions is essential. More studies are needed to clarify the nature of the relationship.

Future studies should continue to examine the effect of self-affirmation on risk-reducing behavioural intentions and risk-reducing behaviours. My study highlighted the importance of considering multiple domains of health promoting behaviour when examining the effects of an intervention targeted to increase behavioural intentions. Therefore, future studies examining the effects of self-affirmation on intentions need to assess multiple domains of risk-reducing behavioural intentions. Moreover, subsequent studies should also extend the range of risk-reducing *behaviours* examined. My study only considered behavioural intentions. Despite promising evidence that self-affirming can promote greater message acceptance and positive changes in intentions, there is much more inconsistency in its effects on behaviours than intentions. In fact, Epton and Harris (2008) proposed that the reasons for the failure to demonstrate behavioural effects are technical, rather than theoretical. They noted that in many self-affirmation studies, the focus has been on encouraging people to reduce or terminate health compromising behaviours (e.g., eliminating alcohol or cigarette consumption), as opposed to health *promoting* behaviours, such as improving diet or exercise regimes. Thus, in order to clarify the effect of self-affirmation on behaviour, future research must include multiple domains of risk-reducing behaviour.

In summary, the present program of research made a number of advancements in the health psychology literature. First, I demonstrated the multidimensional nature of

risk-reducing behavioural intentions, which has important implications for any study examining the effects of interventions targeted to change such intentions. Second, my program of research was the first of its kind to examine the dynamics and interrelationships among manipulated efficacy, perceptions of efficacy and risk, and risk-reducing intentions. My findings have important implications, and highlighted the complexity of variables in health risk behaviour. Overall, my results provide a strong foundation for future research in the area of health risk behaviour.

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Appendix A

Experiment 1 Materials

Study Intro

Heart disease is the number one killer in Canada. It is also the most costly, putting the greatest burden on our national health care system. A 2005 Statistics Canada study estimates that at present, 1,286,000 Canadians have heart disease and 537,000 of those individuals have had a heart attack. By 50 years of age, at least one in four men and one in five women report having heart disease. Researchers in Canada want to know why these numbers are so high. More specifically, what factors predict an individual's risk of having a heart attack? Is it their weight? Eating habits? Genetics?

In partnership with the Heart and Stroke Foundation of Canada, our lab has been investigating risk of heart attack. In particular, we're interested in finding early behavioural predictors that will tell us a) who is at an increased risk of having a heart attack and b) how high an individuals' risk is.

Our lab been measuring university-aged students on certain behavioural characteristics and then following them over time to track who develops heart disease and/or suffers a heart attack. By using ordinary least-squares multiple regression we've developed a mathematical formula that can predict whether or not an individual is at an increased risk of having a heart attack, and how high an individual's risk is, based on these behavioural characteristics. Using these results, we have developed a risk assessment questionnaire that can estimate an individual's risk of having a heart attack prior to the age of 50. Today, you are going to be taking part in a study further validating this risk assessment questionnaire.

Before you begin the main study, you will be asked to complete two brief studies for other researchers associated with the lab.

Self-Affirmation Manipulation

Self-Affirmation

The first study is an attitudes-related study examining values.
Below is a list of values. Please select the value that is MOST important to you personally.

Please write a short essay (a few paragraphs) describing why this value is important to you. In your essay, indicate how you use this value in everyday life, if possible describing specific occasions on which this value determined what you did.

1) business
2) economics
3) art
4) music
5) theatre
6) social life
7) relationships
8) science
9) pursuit of knowledge

No Self-Affirmation

The first study is an attitudes-related study examining values.
Below is a list of values. Please select the value that is LEAST important to you personally.

Please write a short essay (a few paragraphs) describing why this value might be important to another student.

1) business
2) economics
3) art
4) music
5) theatre
6) social life
7) relationships
8) science
9) pursuit of knowledge

Perceived Efficacy Manipulation

The second study is related to our research examining variables associated with heart attacks.

This particular study examines participants' MEMORY for information related to the heart.

Please read the following passage.

High Perceived Efficacy

Preventing a heart attack is easier than you think. With proper lifestyle changes, one can substantially decrease their risk of having a heart attack. Many individuals think that heart attacks occur as a part of aging, with genetics and family history of heart disease being major predictors of risk. However, recent research has demonstrated lifestyle and situational factors are the most important factors in preventing heart attacks.

Your diet impacts many risk factors related to cardiovascular disease and heart attacks. For example, cholesterol, high blood pressure, diabetes, and obesity -- all associated with heart attack risk -- can be improved through dietary means. Simple changes such as increasing fruits and vegetables and decreasing saturated fat can substantially reduce your risk.

Physical activity is another important situational factor that can reduce your heart attack risk. In today's fast-paced society, physical activity is at an all-time low and Canadians are some of the most sedentary individuals in the developed world. And this is bad news for the heart. If our heart does not get enough exercise, it gets weak and cannot pump blood as efficiently as it should. However, if we exercise regularly our heart gets stronger, the level of fat in our bloodstream drops, and our arteries stay clear of dangerous deposits that cause blockages. A simple 20 minutes per day can do wonders for your heart muscle.

Smokers' risk of developing coronary heart disease is 2-4 times that of nonsmokers. Cigarette smoke contains over 850 chemical components which cause serious damage to the human body. They damage blood vessels, increase fat-levels, encourage blood-clotting, and damage the walls of our arteries. Quitting smoking (or not starting to smoke in the first place) can substantially reduce your heart attack risk.

Being overweight can also increase your risk of heart attack. Even individuals who are moderately overweight (i.e., 10-15lbs) have a much higher risk of a heart attack. A 2006 Dutch study (Bogers) found that individuals who were moderately overweight had a 32% increased risk of heart disease compared those who were not overweight. Being obese increased risk by 81%.

In 2007 the American Heart Association released a ground-breaking study demonstrating that lifestyle is the most important predictor of heart attacks. This study followed a group of 18 year-olds over four decades. They found that diet, physical activity, smoking, and weight were the most important factors in preventing a heart attack. When combined, these factors accounted for about 80% of the variance in predicting one's risk of a heart attack prior to the age of 50.

The AHA study also investigated background factors such as genetics, family history of heart disease, as well as homocysteine (an amino acid thought to be related to cardiac risk), c-reactive protein (a protein produced as part of the inflammatory process), and lipoprotein-A (another protein thought to be related to risk). AHA's results indicated that these background factors contributed only minimally (less than 10%) to an individual's heart attack risk. This was an important finding, as many individuals thought that these factors were some of the best predictors of heart attack.

Based on their research of risk, Heart and Stroke Foundation of Canada has listed five important changes that can substantially decrease your risk of having a heart attack:

1) Eat your fruits and veggies. The Harvard-based Nurses' Health Study found that the higher the average daily intake of fruits and vegetables, the lower the chances of developing cardiovascular disease.

2) Watch the saturated fat. Saturated fat is bad fat, and diets high in this type of fat are associated with increased risk of heart disease and heart attack. It's found in animal products, such as in red meat, butter, and cheese. So start replacing that butter and steak with fish and olive oil!

3) Stop smoking!!

4) Get moving! Just 20 minutes of moderate activity every day can decrease your risk by 65%.

5) Watch your waistline! Keeping your weight in the normal range is one of the most important things you can do to drop your risk.

Together, these findings suggest by that you can control your risk of having a heart attack. YOU HAVE CONTROL OVER YOUR RISK! YOUR behaviour and YOUR lifestyle are the most important factors in reducing your risk of a heart attack. The 5 changes are simple to implement and may well save your life.

Please complete the following questions.

What was the name of the agency that released the groundbreaking risk study?
1 Statistics Canada

2 American Heart Association
3 American Diabetes Association
4 Heart and Stroke Foundation of Canada

What was the major finding of the American Heart Association?

1 Lifestyle is the most important predictor of heart attacks.
2 Smoking is the most important predictor of heart attacks.
3 Weight is the most important predictor of heart attacks.
4 Diet is the most important predictor of heart attacks.

When combined, diet, physical activity, smoking, and weight account for __% of the variance in the prediction of a heart attack prior to the age of 50.

1 80%
2 81%
3 82%
4 83%

Individuals who were moderately overweight had a __ % increased risk of having a heart attack compared to those who were normal weight.

1 32%
2 42%
3 52%
4 62%

What type of fat is primarily associated with increased cardiovascular risk?

1 Unsaturated
2 Saturated
3 All fats

Cigarette smoke increases heart attack risk by damaging blood vessels.

1 True
2 False

Thank you. You have completed this experiment.

Low Perceived Efficacy

Preventing a heart attack prior to the age of 50 might be more difficult than you expect. Many individuals think that the strongest predictors of heart attacks are lifestyle factors such as poor diet, inactivity, and smoking. While these factors do influence one's risk to some degree, recent research suggests that such lifestyle factors may not be as important as genetics and family history in determining an individual's risk of a heart attack, especially before the age of 50.

It is common knowledge that cardiovascular disease and heart attacks run in families, and there is an immense amount of conclusive research that has shown that familial history is the greatest predictor of cardiac events. Rates of high blood pressure, heart attacks, and strokes are highly correlated among first degree relatives, with familial history accounting for 76% of the variance! This means that people who are closely related tend to have similar rates of heart attacks and other cardiovascular events. So if your parents and/or grandparents have cardiovascular problems, there is a good chance you will as well.

Investigations of identical twins raised in different homes show that the genetic contribution to heart attack risk might be as high as 70%. This means that people with certain genes tend to have similar rates of heart attacks. Moreover, your family may have a genetic condition that raises unwanted blood cholesterol levels, and this can significantly increase your heart attack risk. Another contributor to heart attack risk - high blood pressure - is also largely genetic. This means that much of your heart attack risk (and risk of other cardiac events) is determined the day you are born. A recent study released by the American Heart Association showed that after controlling for lifestyle factors, individuals with one or more first-degree relatives with a history of heart attack, stroke, or high blood pressure had a 78% risk of heart attack.

Certain proteins and amino acids have also caught the attention of researchers. Homocysteine is an amino acid in the blood, and epidemiological studies have shown that too much homocysteine in the blood is related to a higher risk of heart attack. Other studies have shown that high homocysteine can influence heart attack risk by damaging the inner lining of arteries and promoting blood clots. C-reactive protein (CRP) is a protein that increases during systemic inflammation, and blood levels of CRP levels are used assess cardiovascular and heart attack risk. High levels of both homocysteine and CRP consistently predict heart attacks and other cardiovascular events above and beyond lifestyle factors, and researchers now think that these chemicals are very important factors determining risk. Both markers are controlled largely by genetics and their levels are very hard to decrease.

Lipoprotein-A (LpA) is a type of "bad" cholesterol found in your blood, and its use in predicting heart attacks has recently gained attention. Researchers at found that Oxford University (Danesh, 2008) tracked 5200 people over 10 years, and found that those with the highest LpA levels had 70% more heart attacks than those with the lowest LpA levels.

LpA levels are determined by your genes, and to date, there are no known strategies for reducing those levels.

Situational factors also affect people's risk of heart attack, especially before the age of 50. Today's fast-paced society has increased the stress levels of Canadians. Stress itself is a risk factor for heart attack, and unless you live in a bubble, stress is unavoidable. Stressors can be minor hassles, major lifestyle changes or a combination of both. Four of the most common stressors include academic, occupational, monetary, and relationship stress. Stress exposes your body to unhealthy, persistently elevated levels of stress hormones like adrenaline and cortisol, and studies have also linked stress to changes in the way blood clots, which increases the risk of heart attacks.

This research implies that individuals do not have as much control over their risk of having a heart attack as they have been led to believe. According to leading cardiac research W.A. Cornish, "Genetics may be the most important factor in determining who has a heart attack and who does not".

Please complete the following questions.

What agency examined the effect of family history on heart attack prevalence?

1 Statistics Canada
2 American Heart Association
3 American Diabetes Association
4 Heart and Stroke Foundation of Canada

What is the genetic contribution to heart attack risk?

1 70%
2 80%
3 90%
4 100%

Individuals with one or more first-degree relatives with a history of heart attack, stroke, or high blood pressure have a ___% risk of heart attack.

1 78%
2 79%
3 81%
4 82%

The Oxford study showed that individuals the with the highest LpA levels had ___% more heart attacks than those with the lowest LpA levels.

1 68%
2 69%
3 70%
4 80%

According to W.A. Cornish, what is the most important factor in determining who has a heart attack?

1 Smoking
2 Genetics
3 Stress
4 C-reactive protein levels

Homocysteine can influence heart attack risk by damaging the inner lining of arteries and promoting blood clots.

1 True
2 False

Thank you. You have completed this experiment.

Baseline Perceived Efficacy

History of Heart-Related Discoveries:
The valves of the heart were discovered by a physician of the Hippocratean school around the 4th century BC. However, at that time their function was not properly understood. Because blood pools in the veins after death, arteries look empty. Ancient anatomists assumed they were filled with air and that they were for transport of air.

Herophilos distinguished veins from arteries, but thought that the pulse was a property of arteries themselves. Erasistratos observed that arteries that were cut during life bleed. He ascribed the fact to the phenomenon that air escaping from an artery is replaced with blood that entered by very small vessels between veins and arteries. Thus, Erasistratos apparently postulated capillaries but with reversed flow of blood.

The 2nd century AD, Greek physician Galen knew that blood vessels carried blood and identified venous (dark red) and arterial (brighter and thinner) blood, each with distinct and separate functions. Growth and energy were derived from venous blood created in the liver from chyle, while arterial blood gave vitality by containing pneuma (air), and originated in the heart. Blood flowed from both creating organs to all parts of the body where it was consumed and there was no return of blood to the heart or liver. The heart

did not pump blood around. Instead, the heart's motion sucked blood in during diastole and the blood moved by the pulsation of the arteries themselves. Galen believed that the arterial blood was created by venous blood passing from the left ventricle to the right by passing through "pores" in the interventricular septum, air passed from the lungs via the pulmonary artery to the left side of the heart. As the arterial blood was created, "sooty" vapors were created and passed to the lungs also via the pulmonary artery to be exhaled.

Heart Structure:
The heart is a muscular organ in all vertebrates responsible for pumping blood through the blood vessels by repeated, rhythmic contractions, or a similar structure in annelids, mollusks, and arthropods. The term cardiac (as in cardiology) means "related to the heart" and comes from the Greek kardia, for "heart."

The heart of a vertebrate is composed of cardiac muscle, an involuntary muscle tissue which is found only within this organ. The average human heart beating at 72 BPM, will beat approximately 2.5 billion times during a lifetime spanning 66 years.

In the human body, the heart is usually situated in the middle of the thorax with the largest part of the heart slightly offset to the left. The heart is usually felt to be on the left side because the left heart (left ventricle) is stronger (it pumps to all body parts). The left lung is smaller than the right lung because the heart occupies more of the left hemithorax. The heart is enclosed by a sac known as the pericardium and is surrounded by the lungs. The mediastinum, a subdivision of the thoracic cavity, is the name of the heart cavity.

In humans, the function of the right side of the heart is to collect de-oxygenated blood in the right atrium, and pump it, via the right ventricle, into the lungs so that carbon dioxide can be dropped off and oxygen picked up. This happens through the passive process of diffusion. The left side collects oxygenated blood from the lungs into the left atrium. From the left atrium the blood moves to the left ventricle which pumps it out to the body. On both sides, the lower ventricles are thicker and stronger than the upper atria. The muscle wall surrounding the left ventricle is thicker than the wall surrounding the right ventricle due to the higher force needed to pump the blood through the systemic circulation.

Please complete the following questions.

What did ancient anatomists assume arteries were for?

1 The transport of blood
2 The transport of air
3 The transport of lymph
4 The transport of urine

Who distinguished veins from arteries?

1 Herophilos
2 Erasistratos
3 Galen
4 Weisman

Galen was:

1 A Greek physician
2 An Egyptian physician
3 A Greek philosopher
4 An Egyptian philosopher

How many times will the average heart beat (at 72 BPM) during a lifetime spanning 66 years?

1 1.5 billion times
2 2.5 billion times
3 3.5 billion times
4 4.5 billion times

Where is the heart usually felt?

1 On the right side of the chest
2 On the left side of the chest
3 In the middle of the chest
4 In your shoulder

The function of the right side of the heart is to collect de-oxygenated blood.

1 True
2 False

Thank you. You have completed this experiment.

Risk Assessment Questionnaire

You have reached main study on heart attack prediction, which was described at the start of today's session. You will now be asked to complete the RAQ to determine your risk of having a heart attack prior to the age of 50.

Please complete the following questions regarding you and your behaviour. These items comprise the demographic and behavioural characteristics that have been demonstrated to be useful in predicting the likelihood of having a heart attack prior to the age of 50.

1) What is your current age?
2) Are you male or female?
3) How tall are you (in feet and inches – e.g., 5'7")?
4) What is your current weight (in pounds)?
5) Have you smoked a cigarette in the past 3 years?
6) Do you eat the recommended 5-10 servings of fruits and vegetables every day?
7) How many times per week do you eat soy-based products (e.g., tofu, miso, soymilk)?
8) Do you engage in the recommended 20 minutes of physical activity every day?
9) In a given month, how many days do you miss planned exercise?
10) Do you weight train at least three times per week?
11) Have any of your immediate family members (parents, siblings) ever been or are they currently overweight by more than 10lbs.?
12) Do any of your first-degree relatives (sibling, parent, aunt/uncle, grandparent, etc.) have high blood pressure?
13) Do any of your first-degree relatives (sibling, parent, aunt/uncle, grandparent, etc.) have high cholesterol?
14) Have any of your first-degree relatives (sibling, parent, aunt/uncle, grandparent, etc.) had a heart attack or stroke?
15) Do you eat fast food at least once per month?
16) Do you eat red meat at least four times per month?
17) Do you eat hard cheeses (e.g., cheddar, blue cheese, etc.)?
18) Do you drink alcohol at least three times per month?
19) Do you eat bran?
20) How many times per week do you eat salmon?
21) How many cans of pop (including diet and non-diet versions) do you drink per week?
22) Do you eat chocolate (milk or dark) at least once per week?
23) How many times per month do you eat chips and/or french fries?
24) If you are female, are you on the birth control pill?
25) Have you ever had your cholesterol levels checked?
26) Have you been stressed (academic, occupational, monetary, and/or relationship) in the past 6 months?
27) Have you had your blood pressure checked in the past year?
28) Do you know what your blood pressure is?

29) Do you take a multi-vitamin every day?
30) Do you drink coffee?
31) How many times per month do you do yoga (either a class or on your own)?

You have now entered all of the required information. By clicking below you will submit your responses and our computer alogirithm will compute a probability for the likelihood of a heart attack prior to the age of 50.

Please be patient. The calculation of your risk may take a few seconds.

The computer algorithm has computed a probability for the likelihood of having a heart attack prior to the age of 50.

This feedback is based on data collected from Queen's University students. Our data indicate that these predictions are highly reliable.

Please click below to view your results.

Based on your responses to the Risk Assessment Questionnaire, YOU ARE LIKELY TO HAVE A HEART ATTACK prior to the age of 50.

Calculations indicate that your % likelihood of having a heart attack prior to the age of 50 is: 35%

Thank you for completing the Risk Assessment Questionnaire.

Appendix B

Experiment 1 Measures

Perceived Efficacy Manipulation Check

1) To what extent can background factors beyond your control (e.g., genetics, family history of heart disease, homocysteine, c-reactive protein, lipoprotein-A etc.) affect your risk of having a heart attack prior to the age of 50?

1 not at all
2
3
4
5
6
7 to a great degree

2) How much control you do feel you have over your risk of having a heart attack prior to the age of 50?

1 no control
2
3
4
5
6
7 a great deal of control

3) To what extent can situational factors beyond your control (e.g., stress, etc.) affect your risk of having a heart attack prior to the age of 50?

1 not at all
2
3
4
5
6
7 to a great degree

4) I can control my risk of having a heart attack prior to the age of 50.

1 strongly disagree
2
3
4
5
6
7 strongly agree

Risk-Reducing Behavioural Intentions

Please indicate the extent to which you agree or disagree with the following statements.

Over the next year…

1) I intend to maintain a healthy diet.
2) I intend to eat more salads.
3) I intend to eat plant-based protein, such as soy at least 3 times per week.
4) I intend to get my cholesterol levels assessed by a physician.
5) I intend to maintain a regular schedule of physical activity.
6) I intend to engage in weight-bearing exercise (e.g., free weights, weight machines) at least twice per week.
7) I intend to spend more time with my friends doing physical activities (e.g., play Frisbee, basketball, running etc.).
8) I intend to go to the gym at least three times per week.
9) I intend to engage in cardiovascular activity five times per week.
10) I intend to increase activity by walking/biking/rollerblading whenever possible (instead of cabbing, driving, or taking the bus).
11) I intend to speak with my physician about ways of reducing my risk of heart disease.
12) I intend to eat red meat no more than four times per month.
13) I intend to eat the recommended 5-10 servings of fruit and vegetables per day.
14) I intend to get no more than 30% of my calories from fat.
15) I intend to drink alcohol only in moderation.
16) I intend to avoid smoking cigarettes, even socially.
17) I intend to eat fast food no more than once every two weeks.
18) I intend to get my blood pressure checked at least once.
19) I intend to get no more than 5% of my calories from saturated fat (saturated fat is fat from animal products such as cheese and steak).

Risk-Reducing Behaviours

Thank you for you participation in the current study. For your information, we have included information regarding opportunities intended to help reduce your risk of having a heart attack prior to the age of 50. Please read over the information. If you are interested in participating in any of the events, please indicate so, and provide us with your email so we can contact you with more details.

The joint partnership between the Heart and Stroke Foundation of Canada and Queen's University is offering a number of opportunities, including nutritional counseling and one-on-one heart history assessment through the School of Medicine, as well as a workshop entitled "Preventing Heart Attacks: Early Intervention is the Best Protection". These services are provided free of charge, but space is limited.

Please read over the information. If you are interested in participating in any of the events, please indicate so, and provide us with your email. Doing so will reserve you a spot at any events you are interested in.

Should you agree to attend any of the available opportunities, you will be sent an information sheet via email regarding the schedule of the event(s) within 3-5 business days. This email will provide you with information regarding the dates, times, and locations of the event(s) (if applicable). The schedule of the events is flexible, and the opportunities are offered at a variety of times. Should you be unable to attend your selection(s), you can indicate that you wish to remain on the list to be contacted for future sessions.

If ultimately you are unable to attend your selected event(s), please reply to the organizers. This is important, as they will need to provide estimates of numbers attending.

1) Nutritional Counseling

The Queen's University School of Medicine is offering nutritional counseling designed to provide you with the information you need to follow a heart healthy diet, either now or in the future. Counseling will be provided by fourth-year medical students as part of their course requirements. This program begins with a one-hour meeting with your counselor. This session will consist of an introduction to the program, information about basic nutrition and its relationship to heart disease, followed by an initial assessment of your dietary habits. Following this, you will be required to keep a detailed food diary for one week. Once this diary is completed and returned to your counselor, you will receive formal feedback as well as a detailed eating plan.

Yes! Please sign me up for nutritional counseling.
Email:
No, I am not interested in nutritional counseling.

2) Heart Function and Heart History Assessment

The Queen's School of Medicine is also offering a heart history assessment, designed to examine your heart health right now, as well as provide a more thorough assessment of heart disease history in your family. This service will be provided by upper-year students in the program who will receive course credit. The consultation will begin with a two-hour non-invasive heart function assessment, as well as an interview regarding your family history of cardiovascular health. In consultation with a supervising physician, your counselor will then be able to provide you with feedback regarding a) you current heart health and b) your familial risk of cardiovascular disease.

Yes! Please sign me up for the heart assessment.
Email:
No, I am not interested in the heart assessment.

3) Preventing Heart Attacks: Early Intervention is the Best Protection

This workshop is designed for university-aged students and is provided by the AMS and the Heart and Stroke Foundation of Canada. It is a two-hour intensive workshop that will provide you with information and tips to help prevent you from having a heart attack. To help the organizers know more about you and your habits, you will be required to complete a 200-item computer-based questionnaire prior to attendance. This workshop will be held in Dunning Hall, and will be offered four times over two weeks.

Yes! Please sign me up for the workshop.
Email:
No, I am not interested in the workshop.

Appendix C

Dependent Variable Correlation Matrices for Experiments 1, 2, and 3

Experiment 1: Dependent Variable Correlation Matrix

	Perceived Control	Self Risk	MBI	ABI	DBI	Others' Risk
Perceived Control	1.00					
Self Risk	-.21**	1.00				
MBI	.05	.13*	1.00			
ABI	.16*	-.08	.49**	1.00		
DBI	.09	.02	.51**	.45**	1.00	
Others' Risk	.06	.35**	.14*	.13*	.09	1.00

Note: MBI = Medical Behavioural Intentions; ABI = Activity Behavioural Intentions; DBI = Diet Behavioural Intentions

$p < .05$. ** $p < .001$

Experiment 2: Dependent Variable Correlation Matrix

	Perceived Control	Self Risk	MBI	ABI	DBI	Others' Risk
Perceived Control	1.00					
Self Risk	-.15*	1.00				
MBI	.03	.02	1.00			
ABI	.11	.17**	.39**	1.00		
DBI	.16*	.03	.56**	.42**	1.00	
Others' Risk	.01	.51**	.15**	-.02	.11	1.00

Note: MBI = Medical Behavioural Intentions; ABI = Activity Behavioural Intentions; DBI = Diet Behavioural Intentions

$p < .05$. ** $p < .001$

Experiment 3: Dependent Variable Correlation Matrix

	Perceived Control	Self Risk	MBI	ABI	DBI	Others' Risk
Perceived Control	1.00					
Self Risk	-.06	1.00				
MBI	.03	.13*	1.00			
ABI	.01	-.05	.33**	1.00		
DBI	.10	-.04	.46**	.42**	1.00	
Others' Risk	.01	.42**	.10	.04	.14*	1.00

Note: MBI = Medical Behavioural Intentions; ABI = Activity Behavioural Intentions; DBI = Diet Behavioural Intentions

$p < .05$. ** $p < .001$

Appendix D

Experiments 1 and 2 Ethics Materials

DESTINY: INFORMATION SHEET FOR PARTICIPANTS

This study is being conducted by Megan Davidson, a Ph.D. student in the Department of Psychology at Queen's University, under the supervision of Dr. Leandre Fabrigar, a professor in the Department of Psychology at Queen's University.

DESTINY comprises several studies. You will be asked to complete a brief writing assignment. You will be provided with information regarding the heart. You will be asked to complete several questionnaires regarding your behaviours and you will be given feedback regarding your risk of a heart attack prior to the age of 50.

You will be seated in front of a personal computer and then the experimenter will verbally describe the context of the experiment. The study will be completed on the computer. The entire session should last approximately 60 minutes.

There are no known physical, psychological, economic, or social risks associated with this study. Your participation in this procedure is completely voluntary and you may withdraw from this study at any time without any consequences on your academic standing at Queen's University. You are not obliged to answer any questions that you find objectionable or which make you feel uncomfortable.

You will be awarded 1.0 course credit for your participation in this study whether you complete it or not.

You may be asked to provide an email address; however, your name and email address will be kept separately from your questionnaire responses that you provide during participation in this study.

Please also be assured that the data will be kept in a secure location, and that all of your responses will be coded to conceal your identity. Your responses will remain confidential and anonymous; only authorized researchers will have access to the data. The information provided will be used only for this research project. Your confidentiality is guaranteed, with no identification of individuals in publications resulting from this study. The raw data provided in this study will be kept in a locked room for which only the experimenters will have access. When the data is no longer needed, it will be destroyed.

While individual results will not be published, and will not be made available to you, publications will consist of the collected data, and you are entitled to a copy of the grouped findings and results.

If you would like further information about the study, or have additional questions or concerns, please feel free to contact the researchers, Megan Davidson (613 533-6000, ext. 75677), or Leandre Fabrigar (613 533-6492). You may also contact the Head of the Department of Psychology at Queen's University, Dr. Ronald Holden (613 533-2879), or the Chair of the Queen's University General Research Ethics Board, Dr. Joan Stevenson, (613 533-6000 ext. 74579, email stevensj@post.queensu.ca).

CONSENT FORM

I, _____, have volunteered to participate in the study titled, DESTINY.

I consent to the above information and understand what is required for participation in the study. I understand that I will be asked to complete a brief writing assignment. I understand that I will be provided with information regarding the heart, that I will be asked to complete a questionnaire regarding my behaviours, and that I will be given feedback regarding my risk of a heart attack prior to the age of 50.

I understand that my participation in the study is completely voluntary and that I am free to withdraw at any time. I also understand that my confidentiality will be protected throughout the study, and that the information I provide will be available only to authorized researchers.

Should I have further questions I understand that I can contact any of the following individuals: the researchers Megan Davidson (613 533-6000, ext. 75677), or Leandre Fabrigar (613 533-6492), the Head of the Department of Psychology at Queen's University, Dr. Ronald Holden (613 533-2879), or the Chair of the Queen's University General Research Ethics Board, Dr. Joan Stevenson, (613 533-6000 ext. 74579, email stevensj@post.queensu.ca).

Signature: _____

Date: _____

Guide to Physical Activity

Exercise has a benefit of reducing risks of cardiovascular disease and diabetes. Start exercising slowly, and gradually increase the intensity. Trying too hard at first can lead to injury.

Examples of moderate amounts of physical activity

Common Chores
Washing and waxing a car for 45-60 minutes
Washing windows or floors for 45-60 minutes
Gardening for 30-45 minutes
Wheeling self in wheelchair 30-40 minutes
Pushing a stroller 11/2 miles in 30 minutes
Raking leaves for 30 minutes
Stairwalking for 15 minutes
Shoveling snow for 15 minutes
Jumping rope for 15 minutes

Sporting Activities
Playing volleyball for 45-60 minutes
Playing touch football for 45 minutes
Walking 13/4 miles in 35 minute (20min/mile)
Basketball (shooting baskets) 30 minutes
Bicycling 5 miles in 30 minutes
Dancing fast (social) for 30 minutes
Walking 2 miles in 30 minutes (15min/mile)
Water aerobics for 30 minutes
Basketball (playing game) for 15-20 minutes
Bicycling 4 miles in 15 minutes
Swimming Laps for 20 minutes
Running 11/2 miles in 15 min. (10min/mile)

Your exercise can be done all at one time, or intermittently over the day. Initial activities may be walking or swimming at a slow pace. You can start out by walking 30 minutes for three days a week and can build to 45 minutes of more intense walking, at least five days a week. With this regimen, you can burn 100 to 200 calories more per day. All adults should set a long-term goal to accumulate at least 30 minutes or more of moderate-intensity physical activity on most, and preferably all, days of the week. This regimen can be adapted to other forms of physical activity, but walking is particularly attractive because of its safety and accessibility. Also, try to increase "every day" activity such as taking the stairs instead of the elevator. Reducing sedentary time is a good strategy to increase activity by undertaking frequent, less strenuous activities. With time, you may be able to engage in more strenuous activities. Competitive sports, such as tennis and volleyball, can provide an enjoyable form of exercise for many, but care must be taken to avoid injury.

Activity Progression

For the beginner, activity level can begin at very light and would include an increase in standing activities, special chores like room painting, pushing a wheelchair, yard work, ironing, cooking, and playing a musical instrument. The next level would be light activity such as slow walking of 24 min/mile, garage work, carpentry, house cleaning, child care, golf, sailing, and recreational table tennis. The next level would be moderate activity such as walking 15 minute/mile, weeding and hoeing a garden, carrying a load, cycling, skiing, tennis, and dancing. High activity would include walking 10 minute/mile or walking with load uphill, tree felling, heavy manual digging, basketball, climbing, or soccer/kick ball. You may also want to try: flexibility exercise to attain full range of joint motion, strength or resistance exercise, and/or aerobic conditioning.

Source: National Heart Lung and Blood Institute (NBLHI) Obesity Education Initiative
http://www.nhlbi.nih.gov/health/public/heart/obesity/lose_wt/phy_act.htm

Debriefing Form: DESTINY

You have just participated in a study examining **unrealistic optimism.** The purpose of this study is to examine the effectiveness of two strategies hypothesized to reduce this bias. In this particular study, we were examining individuals' acceptance of risk of academic mark decrease. **THE FEEDBACK YOU RECEIVED REGARDING YOUR RISK OF A HEART ATTACK WAS FALSE. All participants received the same, high risk feedback from the risk assessment questionnaire.**

The programs and all related materials described during this session are fictitious. Proper nutrition and physical activity are important for both heart and general health. You will not be expected to attend workshops or engage in any other tasks. All information provided by you during this session will remain confidential and will not be used for any purpose other than the present study.

Please do not discuss this study with anyone outside of the laboratory. By speaking to others, you may disclose the nature of the study and negatively affect the results. Your cooperation is appreciated.

Perceived susceptibility, one's belief about the likelihood of personal harm, is a key concept in considering behaviour. When individuals perceive their risk of some adverse outcomes to be high, they are more likely to take preventative action to reduce their risk. However, numerous studies show that persons tend to underestimate their risk of negative outcomes. This underestimation of perceived risk has been termed as an **optimism bias**, or **unrealistic optimism**.

In our lab we are examining the effects of two strategies in reducing unrealistic optimism. We needed to give you false (and high-risk) feedback about your risk of a heart attack in order to examine how these strategies affect unrealistic optimism. One of these strategies we are examining is known as self-affirmation, which involves thinking about one's affirming and sustaining valued self-images. In the study you just completed, self-affirmation was manipulated by having you write an essay regarding your values. In the high self-affirmation condition, participants wrote about a highly-important value. In the low self-affirmation condition, participants wrote about an unimportant value. Self-affirmation was manipulated to see if high self-affirmation would increase risk acceptance and increase risk-reducing behaviours.

The second strategy is known as perceived efficacy. Perceived efficacy is composed of two dimensions, self-efficacy (one's beliefs about his or her ability to perform the recommended response) and response efficacy (one's beliefs about whether the recommended response is effective in averting the threat). In the study you just completed, perceived efficacy was manipulated by having you read over information regarding the effectiveness of different behaviours in preventing heart attacks. In the high perceived efficacy condition, participants read information stating that proper eating and fitness habits are the most important factors in preventing heart attacks. This condition emphasized that preventing these events was within your control. In the low perceived efficacy condition participants read information stating that these practices were largely ineffective in controlling your risk. This condition emphasized that these events were out of your control. Participants in the control condition read about the history of the heart. Perceived efficacy was manipulated to see if high perceived efficacy would increase risk acceptance and increase risk-reducing behaviours.

Should you want help in keeping a healthy heart there are resources available to Queen's students. You can contact Health Services at Queen's to speak with a nurse or a physician.

Information regarding living a heart-healthy lifestyle can be found on the American Heart Association's website, at http://www.americanheart.org/presenter.jhtml?identifier=1200009. You can also read information about proper nutrition on Health Canada's website, http://www.hc-sc.gc.ca/fn-an/nutrition/index_e.html.

If you are uncomfortable with the deception, please note that you are free to withdraw your data from the study. Remember that your results are confidential to me and my supervisor, and that all results are published anonymously as group data. If participating in this experiment has caused sufficient distress you that you wish to speak to a counselor, please contact Queen's Counseling at 613 533-2506, or TALK Distress and Information Line at 613 533 1771.

For more information regarding unrealistic optimism and strategies designed to reduce it, please refer to:

Harris, P. R. & Napper, L. (2005). Self-affirmation and the biased processing of health-risk information. Personality and Social Psychology Bulletin, 31, 1250-1263.

Weinstein, N. D. (1996). Unrealistic optimism: Present and future. Journal of Social and Clinical Psychology, 15, 1-8.

Witte, K. & Allen, M. (2000). A meta-analysis of fear appeals: Implications for effective public health campaigns. Health Education and Behavior, 27, 591-615.

In the event that you have any complaints, concerns, or questions about this research, please feel free to contact my supervisor, Dr. Leandre Fabrigar (613 533-6492). Should this approach not remove your dissatisfaction, you may also contact the Head of the Department of Psychology at Queen's University, Dr. Ronald Holden (613 533-2879), or the Chair of the Queen's University General Research Ethics Board, Dr. Joan Stevenson, (613 533-6000 ext. 74579, email stevensj@post.queensu.ca).

If you would like any information about the results of the study once it is completed, feel free to contact me, Megan Davidson, at 613 533-6000, ext. 75677.

Thank you very much for participating!

Appendix E

Experiment 2 Modified Materials and Measures

Baseline Perceived Efficacy

Myocardial infarction (MI or AMI for acute myocardial infarction), commonly known as a heart attack, occurs when the blood supply to part of the heart is interrupted causing some heart cells to die. Heart attacks occur most often as a result of a condition called coronary artery disease (CAD). In CAD, a fatty material called plaque builds up over many years on the inside walls of the coronary arteries (the arteries that supply blood and oxygen to your heart). Eventually, an area of plaque can rupture, causing a blood clot to form on the surface of the plaque. If the clot becomes large enough, it can mostly or completely block the flow of oxygen-rich blood to the part of the heart muscle fed by the artery. This resulting ischemia (restriction in blood supply) and oxygen shortage, if left untreated for a sufficient period of time, can cause damage and/or death (infarction) of heart muscle tissue (myocardium).

The onset of symptoms in heart attacks is usually gradual, over several minutes, and rarely instantaneous. Chest pain is the most common symptom of acute myocardial infarction and is often described as a sensation of tightness, pressure, or squeezing. Chest pain due to ischemia (a lack of blood and hence oxygen supply) of the heart muscle is termed angina pectoris. Pain radiates most often to the left arm, but may also radiate to the lower jaw, neck, right arm, back, and epigastrium, where it may mimic heartburn. Levine's sign, in which the patient localizes the chest pain by clenching their fist over the sternum, has classically been thought to be predictive of cardiac chest pain.

Shortness of breath (dyspnea) occurs when the damage to the heart limits the output of the left ventricle, causing left ventricular failure and consequent pulmonary edema. Other symptoms include excessive sweating, weakness, light-headedness, nausea, vomiting, and palpitations. These symptoms are likely induced by a massive surge of catecholamines from the sympathetic nervous system, which occurs in response to pain and the hemodynamic abnormalities that result from cardiac dysfunction. Loss of consciousness and sudden death can occur in heart attacks.

Women and older patients experience atypical symptoms more frequently than their male and younger counterparts. Women also have more symptoms compared to men (2.6 on average versus 1.8 symptoms in men). The most common symptoms of heart attack in women include dyspnea (shortness of breath), weakness, and fatigue. In women, chest pain may be less predictive of coronary ischemia than in men. Fatigue, sleep disturbances, and dyspnea have been reported as frequently occurring symptoms which may manifest as long as one month before the actual clinically manifested ischemic event. Approximately half of all heart attack patients have experienced warning symptoms such as chest pain prior to the infarction.

Approximately one fourth of all heart attacks are silent, without chest pain or other symptoms. These cases can be discovered later on electrocardiograms or at autopsy without a prior history of related complaints. A silent course is more common in the elderly, in patients with diabetes mellitus, and after heart transplantation, probably because the donor heart is not connected to nerves of the host. In diabetics, differences in pain threshold, autonomic neuropathy, and psychological factors have been cited as possible explanations for the lack of symptoms.
The diagnosis of a heart attack is made by integrating the history of the presenting symptoms and physical examination with electrocardiogram findings and cardiac markers (blood tests for heart muscle cell damage). A coronary angiogram allows visualization of narrowings or obstructions on the heart vessels, and therapeutic measures can follow immediately. At autopsy, a pathologist can diagnose a heart attack based on anatomopathological findings.

Please complete the following questions.

What is ischemia?

1 Restriction in venous blood
2 Restriction in oxygen
3 Restriction in blood supply
4 Restriction in arterial blood

The onset of heart attack symptoms is instantaneous.

1 True
2 False

Levine’s sign is:

1 A clenched fist during a heart attack
2 A clenched fist held over the heart
3 A clenched fist held over the stomach
4 A clenched fist held over the sternum

How many heart attack symptoms do men have, on average?

1 1.8
2 2.6
3 2.8
4 3.6

What are the most common symptoms of heart attacks in women?

1 Dyspnea
2 Fatigue

3 Weakness
4 All of the above

How many heart attacks are silent (i.e., without chest pains or other symptoms)?

1 1/2
2 1/3
3 1/4
4 1/5

Risk-Reducing Behavioural Intentions

Please indicate the extent to which you agree or disagree with the following statements.

Over the next year…

1) I intend to maintain a healthy diet.
2) I intend to eat more salads.
3) I intend to eat more plant-based protein, such as soy.
4) I intend to get my cholesterol levels assessed by a physician.
5) I intend to maintain a regular schedule of physical activity.
6) I intend to engage in weight-bearing exercise (e.g., free weights, weight machines) at least twice per week.
7) I intend to spend more time with my friends doing physical activities (e.g., play Frisbee, basketball, running etc.).
8) I intend to go to the gym at least three times per week.
9) I intend to engage in cardiovascular activity five times per week.
10) I intend to increase activity by walking/biking/rollerblading whenever possible (instead of cabbing, driving, or taking the bus).
11) I intend to speak with my physician about ways of reducing my risk of heart disease.
12) I intend to eat red meat no more than four times per month.
13) I intend to eat the recommended 5-10 servings of fruit and vegetables per day.
14) I intend to get no more than 30% of my calories from fat.
15) I intend to drink alcohol only in moderation.
16) I intend to avoid smoking cigarettes, even socially.
17) I intend to reduce my consumption of fast food.
18) I intend to get my blood pressure checked at least once.
19) I intend to get no more than 5% of my calories from saturated fat (saturated fat is fat from animal products such as cheese and steak).
20) I intend to speak with my family about my familial risk of heart disease and heart attacks.

Appendix F

Experiment 3 Materials

Study Intro

Colorectal cancer is cancer of the large intestine (colon), the lower part of your digestive system. Rectal cancer is cancer of the last 6 inches of the colon. Together, they're often referred to as colorectal cancers. This disease has a higher prevalence and a higher mortality rate than most would estimate. On average, 423 Canadians will be diagnosed with colorectal cancer every week and 171 will die from the disease weekly. In fact, colorectal cancer is the second leading cause of cancer-related deaths in Canada.

Canadian cancer researchers want to know why these numbers are so high. More specifically, what factors predict an individual's risk of developing colorectal cancer? Is it lifestyle? Eating habits? Genetics?

In partnership with Colon Cancer Canada, our lab has been investigating risk of colorectal cancer. In particular, we're interested in finding early behavioural predictors that will tell us a) who is at an increased risk of developing colorectal cancer and b) how high an individuals' risk is.

Our lab been measuring university-aged students on certain behavioural characteristics and then following them over time to track who develops colorectal cancer. By using ordinary least-squares multiple regression we've developed a mathematical formula that can predict whether or not an individual is at an increased risk of developing colorectal cancer, and how high an individual's risk is, based on these behavioural characteristics. Using these results, we have developed a risk assessment questionnaire that can estimate an individual's risk of developing colorectal cancer prior to the age of 50. Today, you are going to be taking part in a study further validating this risk assessment questionnaire.

Before you begin the main study, you will be asked to complete two brief studies for other researchers associated with the lab.

Perceived Efficacy Manipulation

The second study is related to our research examining variables associated with cancer.

This particular study examines participants' MEMORY for information related to colorectal cancer.

Please read the following passage.

High Perceived Efficacy

Preventing colorectal cancer is easier than you think. In fact, it is one of the most preventable forms of cancer. The most effective prevention of colorectal cancer is early detection and removal of precancerous colon polyps (small clumps of cells that form on the colon lining) before they turn cancerous. Moreover, with proper lifestyle changes, one can substantially decrease their risk of developing colorectal cancer. Many individuals think that cancer is a natural part of aging, with genetics and family history being major predictors of risk. However, recent research has demonstrated lifestyle and situational factors are the most important factors in preventing colorectal cancer.

Proper diet is one of the most important factors in the prevention of colorectal cancer. Studies show that a diet high in red meat and processed meat, and low in fresh fruits, vegetables, poultry, and fish increases the risk of colorectal cancer. Other studies have also suggested that diets low in fiber are also associated with an increased risk of colorectal cancer. There are two types of dietary fiber, both of which can help reduce your colorectal cancer risk. Soluble fiber (e.g., beans and legumes, oats and barley) forms a gel when mixed with liquid, while insoluble fiber (e.g., nuts and seeds, vegetables such as celery and spinach) passes through our intestines largely intact. Simple dietary changes such as increasing fruits and vegetables and decreasing red and processed meats can substantially reduce your risk.

In addition to diet, alcohol consumption is another important factor in the prevention of colorectal cancer. Numerous studies have shown a relationship between alcohol consumption and colorectal cancer risk. In fact, one study found that individuals who drank a high amount of alcohol each week had at least a 1 in 5 chance of having a colorectal tumour. Research suggests that to minimize your risk of developing colorectal cancer, it's best to drink in moderation.

Physical activity is another important factor that can reduce your risk of colorectal cancer. Canadians are some of the most sedentary individuals in the developed world. A sedentary lifestyle has been associated with an increased risk of developing colorectal cancer, and in contrast, research has consistently shown that people who are physically active are at lower risk of developing colorectal cancer.

Evidence from epidemiologic studies suggests that cigarette smoking increases the risk of colorectal cancer. Smokers' risk of developing colorectal cancer is 2-4 times that of nonsmokers. Quitting smoking (or not starting to smoke in the first place) can substantially reduce your colorectal cancer risk.

Being overweight can also increase your risk of colorectal cancer. Even individuals who are moderately overweight (i.e., 10-15lbs) have a much higher risk of a developing the disease. A 2008 Dutch study found that individuals who were moderately overweight had a 32% increased risk of colorectal cancer compared those who were not overweight. Being obese increased risk by 81%.

In 2007 the American Cancer Society released a ground-breaking study demonstrating that lifestyle is the most important predictor of colorectal cancer. This study followed a group of 18 year-olds over four decades. They found that diet, physical activity, smoking, and weight were the most important factors in preventing colorectal cancer. When combined, these factors accounted for about 80% of the variance in predicting one's risk of developing colorectal cancer prior to the age of 50.

The ACA study also investigated background factors such as genetics, and family history of colorectal cancer and bowel disease. Results indicated that these background factors contributed only minimally (less than 10%) to an individual's risk of developing colorectal cancer. This was an important finding, as many individuals thought that these factors were some of the best predictors of developing the disease.

Based on their research of risk, Colon Cancer Canada has listed five important changes that can substantially decrease your risk of developing colorectal cancer:

1) Eat your fruits, veggies, and whole grains. Fruits, vegetables and whole grains contain fiber, vitamins, minerals, and antioxidants which can significantly reduce your risk. Try to eat five or more servings of fruits and vegetables every day.

2) Decrease consumption of red meat and processed meat. This includes steak, hamburgers, luncheon meats, and hotdogs. Try replacing these foods with healthier options, such as chicken, pork, and fish.

3) Drink alcohol only in moderation, and stop smoking.

4) Get moving! Physical activity every day can substantially decrease your risk.

5) Watch your waistline! Keeping your weight in the normal range is an important way to reduce your risk.

Together, these findings suggest by that you can control your risk of developing colorectal cancer. YOU HAVE CONTROL OVER YOUR RISK! YOUR behaviour and YOUR lifestyle are the most important factors in reducing your risk of colorectal cancer. The 5 changes are simple to implement and may well save your life.

Please complete the following questions.

What was the name of the agency that released the groundbreaking risk study?

1 Statistics Canada
2 American Cancer Society
3 American Diabetes Association
4 Canadian Cancer Society

What was the major finding of the American Cancer Society?

1 Lifestyle is the most important predictor of colorectal cancer.
2 Smoking is the most important predictor of colorectal cancer.
3 Weight is the most important predictor of colorectal cancer.
4 Diet is the most important predictor of colorectal cancer.

When combined, diet, physical activity, smoking, and weight account for __% of the variance in the prediction of developing colorectal cancer prior to the age of 50.

1 80%
2 81%
3 82%
4 83%

Individuals who were moderately overweight had a __ % increased risk of developing colorectal cancer compared to those who were normal weight.

1 32%
2 42%
3 52%
4 62%

What type of meat(s) is/are primarily associated with increased colorectal cancer risk?

1 Red meat and processed meats
2 Red meat and pork
3 Pork and processed meats
4 Red meat

Smokers' risk of developing cancer is __ times that of non-smokers.

1 1-3
2 2-4
3 3-5
4 4-6

Low Perceived Efficacy

Preventing colorectal cancer prior to the age of 50 might be more difficult than you expect. Many individuals think that the strongest predictors of colorectal cancer are lifestyle factors such as poor diet, alcohol consumption, inactivity, and smoking. While these factors do influence one's risk to some degree, recent research suggests that such lifestyle factors may not be as important as genetics and family history in determining an individual's risk of developing colorectal cancer, especially before the age of 50.

It is common knowledge that colorectal cancer (and cancer in general) runs in families, and there is an immense amount of conclusive research that has shown that familial history is the greatest predictor of developing colorectal cancer. Studies have shown that individuals with a familial history of colorectal cancer or any inflammatory bowel disease (e.g., ulcerative colitis, Crohn's disease) are 10 times more likely to develop colorectal cancer than those without such a history. Further, individuals with a familial history of other common cancers – including breast cancer, prostate cancer, and uterine cancer – are 5 times more likely to develop colorectal cancer than those without such a history. Family history accounts for 76% of the variance in colorectal cancer risk. This means that if your parents, grandparents, or other extended family members have been diagnosed with these diseases, your risk of developing colorectal cancer is high.

Investigations of identical twins raised apart show that the genetic contribution to colorectal cancer risk might be as high as 70%. This means that people with certain genes tend to have similar rates of colorectal cancer. Moreover, your family may have a genetic condition that raises the risk of developing polyps of the colon, such as familial adenomatous polyposis (FAP) or hereditary nonpolyposis colorectal cancer (HNPCC). And such a predisposition can significantly increase your risk of developing colorectal cancer. Polyps are small clumps of cells that form on the colon lining, and as many as 30% of adults have one or more colon polyps. Some polyps are harmless, but others can become cancerous over time.

Certain proteins and hormones have also caught the attention of colorectal cancer researchers. C-reactive protein (CRP) is a protein that increases during systemic inflammation, and blood levels of CRP levels can be used to predict colorectal cancer risk: numerous studies have shown that elevated CRP levels predict development of colorectal cancer. Guanylyl cyclase C (GCC) is a protein receptor on the surface of intestinal epithelial cells for two hormones that regulate the growth of intestinal epithelial cells. GCC is known to suppress tumour formation. Low levels of GCC have been shown to be associated with an increased risk of colon tumours. Both CRP and GCC consistently predict colorectal cancer above and beyond lifestyle factors, and researchers now think that these chemicals are very important factors determining risk. Both markers are controlled largely by genetics and their levels are very hard to control.

Situational factors also affect people's risk of colorectal cancer, especially before the age of 50. Stressors can be minor hassles, major lifestyle changes or a combination of both. Four of the most common stressors include academic, occupational, monetary, and

relationship stress. Stress exposes your body to unhealthy, persistently elevated levels of stress hormones like adrenaline and cortisol, which are associated with colorectal cancer risk.

This research implies that individuals do not have as much control over their risk of developing colorectal cancer as they have been led to believe. According to leading colorectal cancer researcher, W.A. Waldman, "Family history and genetics may be the most important factor in determining who develops colorectal cancer and who does not".

Please complete the following questions.

CRP is a protein that increases during systemic inflammation.

1 True
2 False

What is the genetic contribution to colorectal risk?

1 70%
2 80%
3 90%
4 100%

Individuals with one or more first-degree relatives with a history of colorectal cancer are ___times more likely to develop colorectal cancer themselves.

1 5
2 6
3 7
4 8

What were the two genetic conditions associated with colorectal cancer risk?

1 HNPCC and CRP
2 CRP and cortisol
3 FAP and CRP
4 FAP and HNPCC

What is the most important factor in determining who has develops colorectal cancer?

1 Smoking
2 Genetics
3 Stress
4 C-reactive protein levels

What is the name of the protein receptor known to suppress tumour formation?

1 CCC
2 CRP
3 GCC
4 Cortisol

Thank you. You have completed this experiment. Please click below to begin the next experiment.

Baseline Perceived Efficacy

Colorectal cancer is cancer of the large intestine (colon), the lower part of your digestive system. Rectal cancer is cancer of the last 6 inches of the colon. Together, they're often referred to as colorectal cancers. Most cases of colorectal cancer begin as small, noncancerous (benign) clumps of cells called polyps. When certain types of polyps grow large enough, they can become cancerous. The polyps that become cancerous are called adenomatous polyps or adenomas. Adenomas account for approximately 75% of all colon polyps. There are several subtypes of adenoma that differ primarily in the way the cells of the polyp are assembled when they are examined under the microscope. Thus, there are tubular, villous, or tubulo-villous adenomas. Villous adenomas are the most likely to become cancerous, and tubular adenomas are the least likely.

Symptoms of colorectal cancer are numerous and nonspecific. They include fatigue, weakness, shortness of breath, change in bowel habits, narrow stools, diarrhea or constipation, red or dark blood in stool, weight loss, abdominal discomfort (e.g., cramps, gas, bloating, or pain), abdominal pain with a bowel movement, or unexplained weight loss.

Symptoms vary according to where the tumor is located. The right colon is spacious, and cancers of the right colon can grow to large sizes before they cause any abdominal symptoms. Typically, right-sided cancers cause iron deficiency anemia due to the slow loss of blood over a long period of time. Iron deficiency anemia causes fatigue, weakness, and shortness of breath. The left colon is narrower than the right colon. Therefore, cancers of the left colon are more likely to cause partial or complete bowel obstruction. Cancers causing partial bowel obstruction can cause symptoms of constipation, narrowed stool, diarrhea, abdominal pains, cramps, and bloating. Bright red blood in the stool may also indicate a growth near the end of the left colon or rectum.

There are a number of tests used to screen and test for colorectal cancer. The fecal occult blood test (FOBT) test checks for hidden blood in fecal material (stool). Currently, two types of FOBT are available. One type, called guaiac FOBT, uses the chemical guaiac to

detect heme in stool. Heme is the iron-containing component of the blood protein hemoglobin. The other type of FOBT, called immunochemical FOBT, uses antibodies to detect human hemoglobin protein in stool (13–15).

Sigmoidoscopy is a procedure where the rectum and lower colon are examined using a lighted instrument called a sigmoidoscope. During this procedure, precancerous and cancerous growths in the rectum and lower colon can be found and either removed or biopsied.

Colonoscopy is a procedure where the rectum and entire colon are examined using a lighted instrument called a colonoscope. During colonoscopy, precancerous and cancerous growths throughout the colon can be found and either removed or biopsied, including growths in the upper part of the colon, where they would be missed by sigmoidoscopy.

During a virtual colonoscopy (also called computerized tomographic colonography) special x-ray equipment is used to produce pictures of the colon and rectum. A computer then assembles these pictures into detailed images that can show polyps and other abnormalities. Because it is less invasive than standard colonoscopy and sedation is not needed, virtual colonoscopy may cause less discomfort and take less time to perform.

A double contrast barium enema (DCBE) involves taking x-rays of the colon and the rectum after the patient is given an enema with a white, chalky liquid containing barium. The barium outlines the large intestines on the x-rays. Tumors and other abnormalities appear as dark shadows on the x-rays.

Finally, a digital rectal exam (DRE) can also be used to screen for colon cancer. In this test, a health care provider inserts a lubricated, gloved finger into the rectum to feel for abnormal areas. DRE allows examination of only the lower part of the rectum.

Please complete the following questions.

What types of polyps cause the majority of colorectal cancers?

1 Adenomatous
2 Villous
3 Tubular
4 Tubulo-villous

In which area of the gastrointestinal tract is colorectal cancer most likely to cause iron deficiency anemia?

1 Left side of the colon
2 Middle of the colon
3 Right side of the colon
4 Rectum

In which area of the gastrointestinal tract is colorectal cancer most likely to cause partial or complete bowel obstruction?

1 Left side of the colon
2 Middle of the colon
3 Right side of the colon
4 Rectum

Sigmoidoscopy is a procedure where the rectum and lower colon are examined.

1 True
2 False

What are the two types of fecal occult blood tests?

1 guaiac FOBT and hemoglobin FOBT
2 hemoglobin FOTB and tubular FOBT
3 immunochemical FOBT and hemoglobin FOBT
4 guaiac FOBT and immunochemical FOBT

What white, chalky chemical is given to patients during the double contrast enema?

Thank you. You have completed this experiment. Please click below to begin the next experiment.

Risk Assessment Questionnaire

You have reached main study on colorectal cancer prediction, which was described at the start of today's session. You will now be asked to complete the RAQ to determine your risk of developing colorectal cancer prior to the age of 50.

Please complete the following questions regarding you and your behaviour. These items comprise the demographic and behavioural characteristics that have been demonstrated to be useful in predicting the likelihood of developing colorectal cancer prior to the age of 50.

1) What is your current age?
2) Are you male or female?
3) How tall are you (in feet and inches – e.g., 5'7")?
4) What is your current weight (in pounds)?
5) Do you eat the recommended 5-10 servings of fruits and vegetables every day?
6) How many times per week do you eat soy-based products (e.g., tofu, miso, soymilk)?
7) Do you engage in the recommended 60 minutes of physical activity every day?
8) In a given month, how many days do you miss planned exercise?
9) Do you weight train at least three times per week?
10) Have any of your immediate family members (parents, siblings) ever been or are they currently overweight by more than 10lbs.?
11) Have any of your first-degree relatives (sibling, parent, aunt/uncle, grandparent, etc.) developed colorectal cancer?
12) Have any of your first-degree relatives (sibling, parent, aunt/uncle, grandparent, etc.) developed cancer of any kind?
13) Do any of your first-degree relatives (sibling, parent, aunt/uncle, grandparent, etc.) have a history of bowel disease (e.g., irritable bowel syndrome, Crohn's disease)?
14) Do you eat fast food at least once per month?
15) Do you eat red meat at least four times per month?
16) Do you eat hard cheeses (e.g., cheddar, blue cheese, etc.)?
17) Do you drink alcohol at least three times per month?
18) Do you eat bran at least five days per week?
19) How many times per week do you eat salmon?
20) How many cans of pop (including diet and non-diet versions) do you drink per week?
21) Do you eat chocolate (milk or dark) at least once per week?
22) How many times per month do you eat chips and/or french fries?
23) If you are female, are you on the birth control pill?
24) Have you been stressed (academic, occupational, monetary, and/or relationship) in the past 6 months?
25) Do you take a multi-vitamin every day?
26) Do you drink coffee?
27) How many times per month do you do yoga (either a class or on your own)?
28) How many times per week do you eat spinach and/or kale?

29) Do you eat processed meat (luncheon meats such as smoked turkey and ham, hotdogs, and sausages)?
30) What percentage of your diet consists of organic food?

You have now entered all of the required information. By clicking below you will submit your responses and our computer alogirithm will compute a probability for the likelihood of developing colorectal cancer prior to the age of 50.

Please be patient. The calculation of your risk may take a few seconds.

The computer algorithm has computed a probability for the likelihood of developing colorectal cancer prior to the age of 50.

This feedback is based on data collected from Queen's University students. Our data indicate that these predictions are highly reliable.

Please click below to view your results.

Based on your responses to the Risk Assessment Questionnaire, YOU ARE LIKELY TO DEVELOP COLORECTAL CANCER prior to the age of 50.

Calculations indicate that your % likelihood of developing colorectal cancer prior to the age of 50 is: 45%

Thank you for completing the Risk Assessment Questionnaire.

In addition to finding predictors of colorectal cancer, we are also interested in individuals' own expectations regarding their likelihood of developing colorectal cancer, as well as their plans over the next year.

Appendix G

Experiment 3 Measures

Perceived Efficacy Manipulation Check

1) To what extent can background factors beyond your control (e.g., genetics, family history of cancer, homocysteine, guanylyl cyclase C) affect your risk of developing colorectal cancer prior to the age of 50?

1 not at all
2
3
4
5
6
7 to a great degree

2) How much control you do feel you have over your risk of developing colorectal cancer prior to the age of 50?

1 no control
2
3
4
5
6
7 a great deal of control

3) To what extent can situational factors beyond your control (e.g., stress, etc.) affect your risk of developing colorectal cancer prior to the age of 50?

1 not at all
2
3
4
5
6
7 to a great degree

4) I can control my risk of developing colorectal cancer prior to the age of 50.

1 strongly disagree
2
3
4
5
6
7 strongly agree

Risk-Reducing Behavioural Intentions

Please indicate the extent to which you agree or disagree with the following statements.

Over the next year…

1) I intend to maintain a healthy diet.
2) I intend to eat more salads.
3) I intend to eat more plant-based protein, such as soy.
4) I intend to speak with my physician about ways of increasing gastrointestinal health.
5) I intend to maintain a regular schedule of physical activity.
6) I intend to engage in more weight-bearing exercise.
7) I intend to spend more time with my friends doing physical activities (e.g., play Frisbee, basketball, running etc.).
8) I intend to go to the gym at least three times per week.
9) I intend to engage in cardiovascular activity five times per week.
10) I intend to increase activity by walking/biking/rollerblading whenever possible (instead of cabbing, driving, or taking the bus).
11) I intend to speak with my physician about ways of reducing my risk of colorectal cancer.
12) I intend to eat red meat no more than four times per month.
13) I intend to eat the recommended 5-10 servings of fruit and vegetables per day.
14) I intend to get no more than 30% of my calories from fat.
15) I intend to drink alcohol only in moderation.
16) I intend to speak with my family about my familial risk of colorectal cancer and inflammatory bowel disease.
17) I intend to reduce my consumption of fast food.
18) I intend to decrease my consumption of processed meats (e.g., deli meats such as smoked turkey and ham, hotdogs, sausages).
19) I intend to reduce my intake of saturated fat (saturated fat is fat from animal products such as cheese and steak).
20) I intend to eat more insoluble fibre (e.g., nuts and seeds, vegetables such as celery and spinach).
21) I intend to eat more soluble fibre (e.g., beans and legumes, oats and barley).

Appendix H

Experiment 3 Ethics Materials

DESTINY: INFORMATION SHEET FOR PARTICIPANTS

This study is being conducted by Megan Davidson, a Ph.D. student in the Department of Psychology at Queen's University, under the supervision of Dr. Leandre Fabrigar, a professor in the Department of Psychology at Queen's University.

DESTINY comprises several studies. You will be asked to complete a brief writing assignment. You will be provided with information regarding the colon. You will be asked to complete several questionnaires regarding your behaviours and you will be given feedback regarding your risk of developing colorectal cancer prior to the age of 50.

You will be seated in front of a personal computer and then the experimenter will verbally describe the context of the experiment. The study will be completed on the computer. The entire session should last approximately 30 minutes.

There are no known physical, psychological, economic, or social risks associated with this study. Your participation in this procedure is completely voluntary and you may withdraw from this study at any time without any consequences on your academic standing at Queen’s University. You are not obliged to answer any questions that you find objectionable or which make you feel uncomfortable.

You will be awarded 0.5 course credit for your participation in this study whether you complete it or not.

You may be asked to provide an email address; however, your name and email address will be kept separately from your questionnaire responses that you provide during participation in this study.

Please also be assured that the data will be kept in a secure location, and that all of your responses will be coded to conceal your identity. Your responses will remain confidential and anonymous; only authorized researchers will have access to the data. The information provided will be used only for this research project. Your confidentiality is guaranteed, with no identification of individuals in publications resulting from this study. The raw data provided in this study will be kept in a locked room for which only the experimenters will have access. When the data is no longer needed, it will be destroyed.

While individual results will not be published, and will not be made available to you, publications will consist of the collected data, and you are entitled to a copy of the grouped findings and results.

If you would like further information about the study, or have additional questions or concerns, please feel free to contact the researchers, Megan Davidson (613 533-6000, ext. 75677), or Leandre Fabrigar (613 533-6492). You may also contact the Head of the Department of Psychology at Queen’s University, Dr. Richard Beninger (613 533-2486), or the Chair of the Queen's University General Research Ethics Board, Dr. Joan Stevenson, (613 533-6000 ext. 74579, email stevensj@post.queensu.ca).

CONSENT FORM

I, _____, have volunteered to participate in the study titled, DESTINY.

I consent to the above information and understand what is required for participation in the study. I understand that I will be asked to complete a brief writing assignment. I understand that I will be provided with information regarding the colon, that I will be asked to complete a questionnaire regarding my behaviours, and that I will be given feedback regarding my risk of developing colorectal cancer prior to the age of 50.

I understand that my participation in the study is completely voluntary and that I am free to withdraw at any time. I also understand that my confidentiality will be protected throughout the study, and that the information I provide will be available only to authorized researchers.

Should I have further questions I understand that I can contact any of the following individuals: the researchers Megan Davidson (613 533-6000, ext. 75677), or Leandre Fabrigar (613 533-6492), the Head of the Department of Psychology at Queen's University, Dr. Richard Beninger (613 533-2486), or the Chair of the Queen's University General Research Ethics Board, Dr. Joan Stevenson, (613 533-6000 ext. 74579, email stevensj@post.queensu.ca).

Signature: _____

Date: _____

Guide to Physical Activity

Exercise has a benefit of reducing risks of cardiovascular disease and diabetes. Start exercising slowly, and gradually increase the intensity. Trying too hard at first can lead to injury.

Examples of moderate amounts of physical activity

Common Chores
Washing and waxing a car for 45-60 minutes
Washing windows or floors for 45-60 minutes
Gardening for 30-45 minutes
Wheeling self in wheelchair 30-40 minutes
Pushing a stroller 11/2 miles in 30 minutes
Raking leaves for 30 minutes
Stairwalking for 15 minutes
Shoveling snow for 15 minutes
Jumping rope for 15 minutes

Sporting Activities
Playing volleyball for 45-60 minutes
Playing touch football for 45 minutes
Walking 13/4 miles in 35 minute (20min/mile)
Basketball (shooting baskets) 30 minutes
Bicycling 5 miles in 30 minutes
Dancing fast (social) for 30 minutes
Walking 2 miles in 30 minutes (15min/mile)
Water aerobics for 30 minutes
Basketball (playing game) for 15-20 minutes
Bicycling 4 miles in 15 minutes
Swimming Laps for 20 minutes
Running 11/2 miles in 15 min. (10min/mile)

Your exercise can be done all at one time, or intermittently over the day. Initial activities may be walking or swimming at a slow pace. You can start out by walking 30 minutes for three days a week and can build to 45 minutes of more intense walking, at least five days a week. With this regimen, you can burn 100 to 200 calories more per day. All adults should set a long-term goal to accumulate at least 30 minutes or more of moderate-intensity physical activity on most, and preferably all, days of the week. This regimen can be adapted to other forms of physical activity, but walking is particularly attractive because of its safety and accessibility. Also, try to increase "every day" activity such as taking the stairs instead of the elevator. Reducing sedentary time is a good strategy to increase activity by undertaking frequent, less strenuous activities. With time, you may be able to engage in more strenuous activities. Competitive sports, such as tennis and volleyball, can provide an enjoyable form of exercise for many, but care must be taken to avoid injury.

Activity Progression

For the beginner, activity level can begin at very light and would include an increase in standing activities, special chores like room painting, pushing a wheelchair, yard work, ironing, cooking, and playing a musical instrument. The next level would be light activity such as slow walking of 24 min/mile, garage work, carpentry, house cleaning, child care, golf, sailing, and recreational table tennis. The next level would be moderate activity such as walking 15 minute/mile, weeding and hoeing a garden, carrying a load, cycling, skiing, tennis, and dancing. High activity would include walking 10 minute/mile or walking with load uphill, tree felling, heavy manual digging, basketball, climbing, or soccer/kick ball. You may also want to try: flexibility exercise to attain full range of joint motion, strength or resistance exercise, and/or aerobic conditioning.

Source: National Heart Lung and Blood Institute (NBLHI) Obesity Education Initiative http://www.nhlbi.nih.gov/health/public/heart/obesity/lose_wt/phy_act.htm

Debriefing Form: DESTINY

You have just participated in a study examining **unrealistic optimism.** The purpose of this study is to examine the effectiveness of two strategies hypothesized to reduce this bias. In this particular study, we were examining individuals' acceptance of risk of academic mark decrease. **THE FEEDBACK YOU RECEIVED REGARDING YOUR RISK OF DEVELOPING COLORECTAL CANCER WAS FALSE. All participants received the same, high risk feedback from the risk assessment questionnaire.**

The programs and all related materials described during this session are fictitious. Proper nutrition and physical activity are important for both colorectal and general health. You will not be expected to attend workshops or engage in any other tasks. All information provided by you during this session will remain confidential and will not be used for any purpose other than the present study.

Please do not discuss this study with anyone outside of the laboratory. By speaking to others, you may disclose the nature of the study and negatively affect the results. Your cooperation is appreciated.

Perceived susceptibility, one's belief about the likelihood of personal harm, is a key concept in considering behaviour. When individuals perceive their risk of some adverse outcomes to be high, they are more likely to take preventative action to reduce their risk. However, numerous studies show that persons tend to underestimate their risk of negative outcomes. This underestimation of perceived risk has been termed as an **optimism bias**, or **unrealistic optimism**.

In our lab we are examining the effects of two strategies in reducing unrealistic optimism. We needed to give you false (and high-risk) feedback about your risk of colorectal cancer in order to examine how these strategies affect unrealistic optimism. One of these strategies we are examining is known as self-affirmation, which involves thinking about one's affirming and sustaining valued self-images. In the study you just completed, self-affirmation was manipulated by having you write an essay regarding your values. In the high self-affirmation condition, participants wrote about a highly-important value. In the low self-affirmation condition, participants wrote about an unimportant value. Self-affirmation was manipulated to see if high self-affirmation would increase risk acceptance and increase risk-reducing behaviours.

The second strategy is known as perceived efficacy. Perceived efficacy is composed of two dimensions, self-efficacy (one's beliefs about his or her ability to perform the recommended response) and response efficacy (one's beliefs about whether the recommended response is effective in averting the threat). In the study you just completed, perceived efficacy was manipulated by having you read over information regarding the effectiveness of different behaviours in preventing colorectal cancer. In the high perceived efficacy condition, participants read information stating that proper eating and fitness habits are the most important factors in preventing colorectal cancer. This condition emphasized that preventing this event was within your control. In the low perceived efficacy condition participants read information stating that these practices were largely ineffective in controlling your risk. This condition emphasized that this event was out of your control. Participants in the control condition read about colorectal cancer, with no information about its controllability. Perceived efficacy was manipulated to see if high perceived efficacy would increase risk acceptance and increase risk-reducing behaviours.

Should you want help in keeping a healthy colon there are resources available to Queen's students. You can contact Health Services at Queen's to speak with a nurse or a physician.

Information regarding living a heart-healthy lifestyle can be found on Colon Cancer Canada's website, at http://www.coloncancercanada.ca/. You can also read information about proper nutrition on Health Canada's website, http://www.hc-sc.gc.ca/fn-an/nutrition/index_e.html.

If you are uncomfortable with the deception, please note that you are free to withdraw your data from the study. Remember that your results are confidential to me and my supervisor, and that all results are published anonymously as group data. If participating in this experiment has caused sufficient distress you that you wish to speak to a counselor, please contact Queen's Counseling at 613 533-2506, or TALK Distress and Information Line at 613 533 1771.

For more information regarding unrealistic optimism and strategies designed to reduce it, please refer to:

Harris, P. R. & Napper, L. (2005). Self-affirmation and the biased processing of health-risk information. Personality and Social Psychology Bulletin, 31, 1250-1263.

Weinstein, N. D. (1996). Unrealistic optimism: Present and future. Journal of Social and Clinical Psychology, 15, 1-8.

Witte, K. & Allen, M. (2000). A meta-analysis of fear appeals: Implications for effective public health campaigns. Health Education and Behavior, 27, 591-615.

In the event that you have any complaints, concerns, or questions about this research, please feel free to contact my supervisor, Dr. Leandre Fabrigar (613 533-6492). Should this approach not remove your dissatisfaction, you may also contact the Head of the Department of Psychology at Queen's University, Dr. Richard Beninger (613 533-2486), or the Chair of the Queen's University General Research Ethics Board, Dr. Joan Stevenson, (613 533-6000 ext. 74579, email stevensj@post.queensu.ca).

If you would like any information about the results of the study once it is completed, feel free to contact me, Megan Davidson, at 613 533-6000, ext. 75677.

Thank you very much for participating!