

Exploring Distorted Thinking about Food and Dietary Misinformation in Nonclinical Samples:
Instrument and Intervention Development and Validation

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Abstract

Distorted thinking about food is common in both clinical and nonclinical populations from dieters to diabetics and eating disorder patients. This type of thinking is triggered by exposure to dieting culture (including related social media), familial dieting, and internalization of the thin ideal, among other factors. The consequences of distorted thinking about food include disordered eating, weight management problems, body dissatisfaction and many others. Despite these findings, distorted thinking about food in nonclinical samples remains poorly understood. Thus, the current research sought to investigate distorted thinking about food within a broad theoretical model that includes societal antecedents important to understanding the nature of this construct in the general population. This model, which is based on Cognitive Behaviour Therapy (CBT) and the Theory of Planned Behaviour (TPB; Ajzen, 1991), was investigated in two studies. The first study is a psychometric validation study of a tool designed to measure distorted thinking about food, the Calorie Catastrophizing Scale (CCS; Monaghan & Santor, 2017) as well as the tool designed to measure dietary misinformation, the Dietary Misinformation Questionnaire (DMQ; Monaghan & Santor, 2017). Results lend support for defining and measuring distorted thinking about food as a distinct construct in nonclinical individuals and suggest that dietary misinformation is an important antecedent to this type of thinking and unhealthy eating behaviours. The second study is a randomized controlled trial of a CBT- and TPB-based workshop intervention aimed at reducing these antecedents. Results show that this intervention led to a significant reduction in distorted thinking about food, dietary misinformation, and other important clinical variables such as symptoms of eating pathology. These findings have a number of important implications for how subclinical eating problems are conceptualized, measured, and prevented.

General Introduction

Distorted Thinking About Food

A common symptom among individuals with eating disorders is distorted thinking (Garner & Bemis, 1982; Fairburn, Cooper, & Cooper, 1986). This distortion can occur in many distinct forms, from body-image perceptual problems (e.g., believing the body is larger than it is) and disparaging beliefs about the self (e.g., “I am only good if I can control my food intake”; Garner, Olmsted, & Bohr, 1982; Lake, Staiger, & Glowinski, 2000), to the overvaluation of weight and shape (e.g. the “thin ideal”; Fairburn, Cooper, & Shafran, 2003; Polivy & Herman, 2002), and exaggerated and unrealistic beliefs about food (e.g., one cookie will cause weight gain), also known as “distorted thinking about food”. The consequence of these beliefs for people with eating disorders is clear: they contribute to the onset and maintenance of pathological symptoms, such as bingeing and purging and caloric restriction (Stice, 2002). Not surprisingly, an explicit goal of many eating disorder treatments is to challenge and reduce this distorted thinking in order to address the cognitive barriers to recovery and so that the uptake of healthful behaviours can occur (e.g., regular, healthy eating, appropriate exercise, finding other sources of self-worth, etc.; Garner & Bemis, 1982; Fairburn, Cooper, & Cooper, 1986). Given the importance and significance of this construct, researchers have developed a number of different tools to measure this type of thinking.

Measuring Distorted Thinking About Food. Many clinical measures have been developed to assess an eating disorder patient’s level of preoccupation with food (e.g., Eating Attitudes Test; Garner & Garfinkel, 1979; Bulimia Cognitive Distortion Scale; Schulman, Kinder, Powers, Prange, & Gleghorn, 1986; Three Factor Eating Questionnaire; Stunkard & Messick, 1985, and many others). The use of these measures on the general population suggests

that cognitive antecedents (e.g., negative attitudes and beliefs about food) are predictive of eating patterns in individuals in the general population as well (de Lauzon et al., 2004); though the incidence of distorted thinking about food in the general population is currently unknown. This is consistent with previous research that has shown that individuals with and without eating disorders can have similar attitudes about food (Carter & Moss, 1984).

Studies have suggested that, just as with eating disorder patients, specific thoughts or cognitions about food and dieting have a stronger influence over food choice and intake than do physiological factors like hunger in the general population (Polivy & Herman, 1985). Therefore, an understanding of how distorted thinking about food impacts nonclinical individuals could illuminate how eating problems develop in nonclinical samples. The utility of clinical measures, however, is limited by the fact that food-related cognitions are usually captured by 3 or 4 items which have not been developed with nonclinical samples and therefore likely do not capture the construct accurately in this population (Garner, Olmstead, Bohr, & Garfinkel, 1982; Stunkard & Messick, 1985). In fact, the use of these measures in nonclinical samples often leads to the false positive identification of eating disorders (Mintz & O'Halloran, 2000), which supports the notion that these tools may not be appropriate for capturing symptoms in the general population. Thus, the development of a tool to measure food-related cognitive distortion for subclinical populations is warranted, and it could enhance the current understanding of how dieting and disordered eating develop, which are problems on the rise in North America.

Consequences of Distorted Thinking about Food

As mentioned above, in clinical populations, the main consequence of distorted thinking about food is the onset and maintenance of pathological symptoms of eating disorders (Stice, 2002). Thus, formal intervention models are designed around the careful measurement and

reduction of this type of thinking and are relatively successful (Fairburn, 2009). Given that research has shown that distorted thinking about food occurs in nonclinical samples and has an impact on eating, it likely follows that the consequences of this thinking in the general population include subclinical eating problems such as dieting and disordered eating. In fact, Polivy and Herman (1985) suggested that food-related cognitive distortions eventually lead to a cycle of dieting (i.e., unhealthy weight-control behaviors such as skipping meals and eliminating food groups; Ackard, Croll, & Kearney-Cooke, 2002; Brownell & Rodin, 1994) and disordered eating behaviours (such as bingeing).

Dieting and disordered eating have become increasingly common among males and females across the lifespan in North America over the past decade (Haines & Neumark-Sztainer, 2006). At least 50% of girls and 30% boys are dissatisfied with their bodies (Keel, Fulkerson, & Leon 1997; Neumark-Sztainer et al., 2002; Ricciardelli, McCabe, 2001), which is one of the most robust risk factors for dieting and eating disorders (Stice, 2002). McVey, Tweed and Blackmore (2004) showed that almost 30% of adolescents are trying to lose weight, while 3.9% report binge eating and 1.5% report self-induced vomiting. The 2003 Youth Risk Behavioural Surveillance System found that 60% of females and 29% of males are dieting for weight-loss (Grunbaum et al., 2004). For children between the ages of 6-11, it is estimated that between 20-56% of girls and 31-39% of boys are currently dieting (Grunbaum et al., 2004). Another study of over 80 000 high school students showed that 56% of girls and 28% of boys in grade 9 engage in one or more of the following behaviours: fasting, skipping meals, taking diet pills or laxatives, self-induced vomiting, smoking cigarettes to lose weight, and binge eating. Among grade 12 students, these figures rose to 57% for females and 31% for males (Croll, Neumark-Sztainer, Story, & Ireland, 2002). Similar rates have been found in other studies (Field, Colditz, &

Peterson, 1997; Grunbaum et al., 2004). These figures are concerning, since dieting has been shown to be ineffective and disordered eating is associated with negative outcomes and health problems.

Dieting has been causally linked to obesity (Field et al., 2003) and eating disorders (Patton et al., 1990). In adolescents, self-reported dieting predicts an increased risk of disordered eating behaviours (Stice, 2001; Stice & Agras, 1998) and subclinical eating disorders (Leon et al., 1999; Patton, Johnson-Sabine, & Wood, 1990; Santonastaso, Friederici, & Favaro, 1999). In fact, the majority of individuals with eating disorders report that dieting preceded the initiation of eating disorder behaviours (Bulik et al., 1997; Fairburn, Cooper, Doll, & Welch, 1999). Furthermore, dieting is not effective in preventing weight-gain (French et al., 1994; Heatherton, Polivy, & Herman, 1991; Klesges, Klerm, Epkins, & Klesges, 1991) and, in fact, cross-sectional data has shown BMI to be correlated with dieting in children (Candy & Fee, 1998; Thelen & Cromier, 1996) and adolescents (Boutelle et al., 2002; Neumark-Sztainer et al., 2002). Finally, dieting actually predicts weight-gain in adolescents and is associated with a number of psychological and physiological consequences (Field et al., 2003; Neumark Sztainer et al., 2006; Stice et al., 1999).

Unhealthy dieting and disordered eating behaviours are associated with body dissatisfaction (Klemchuck, Hutchinson, & Frank, 1990), low self-esteem (Hesse-Biber, 1989; Mintz & Betz, 1988) and mood disturbance (Rotenberg & Flood, 2000). These behaviours are also linked to health problems, such as cardiac complications (Arik, Dessser, & Benchimol, 1985), oral and dental complications (Touyz et al., 1993), and weight-management problems (Kayman, Bruvold, & Stern, 1990). Finally, as mentioned above, the use of unhealthy eating behaviours is associated with the development and maintenance of clinical eating disorders

(Heatherton & Polivy, 1992; Stice & Agras, 1998).

It is clear that subclinical eating problems are on the rise; however, few effective prevention or intervention strategies exist to combat them (see study 2). In fact, despite the similarities between clinical and subclinical populations, no prevention or intervention models for dieting or disordered eating explicitly target food-related cognitions, which could potentially account for their poor results (discussed in detail in study 2). Thus, the current line of research sought to investigate the link between distorted thinking about food and unhealthy eating behaviours. As a first step, the goal of the first study was to develop a psychometric tool to specifically measure distorted thinking about food in nonclinical samples, and to understand its association with other pathological variables, such as poor body image and disordered eating. Then, the goal of the second study was to develop an evidence-based intervention to target distorted thinking about food and other variables. In order to gain a thorough understanding of the nature of this construct, a number of antecedents were also considered in the conceptualization of distorted thinking about food.

Antecedents of Distorted Thinking about Food

Extensive research has been conducted with clinical populations to determine causes, antecedents, and predictors of eating disorder symptoms. This has mostly focused on determining predictors of dieting and body dissatisfaction. Despite the importance of distorted thinking about food, little is known about how it initially develops; however, cognitive behavioural models of eating disorders have suggested that dysfunctional beliefs about eating could emerge from early experiences and messages about dieting and body shape (such as having family members or role models who diet), which are later activated and maintained by critical events such as media exposure and the pressure to be thin (Fairburn, Cooper, & Shafran, 2003).

This is consistent with research that has shown that, among the most robust predictors of dieting are familial attitudes towards weight and dieting, early childhood experiences including media use, and youth experiences including social media use (Fairburn, Cooper, Doll, & Welch, 1999; Ferguson, Muñoz, Garza, & Galindo, 2014; Leung, Schwartzman, & Steiger, 1996).

Family values (e.g., attitudes towards meal time, dieting, and thin ideal) play a role in the development of dieting and subsequent eating disorders (Leung et al., 1996). Parental preoccupation with weight, food, and dieting also contributes to the onset of dieting and disordered eating (Carper, Fisher, & Birch, 2000; Fairburn et al., 1999). This is particularly significant for young girls whose mothers restrict their caloric intake (Sinton & Birch, 2005). Furthermore, research has shown that daughters of mothers with poor body image have an increased internalization of the thin-ideal (Leung et al., 1996; Stice, Nemeroff, & Shaw, 1996). Finally, Fairburn and colleagues (1999) found that family members' criticism about eating, appearance, or weight is a significant predictor of eating disorders across the spectrum. As children begin to age and consume more media, it has also been suggested that this media use contributes to poor body image and eventual dieting.

In general, it is believed that media use leads to the development of poor body image and dieting, but this research has yielded mixed results. Correlational research has shown that increased exposure to fashion magazines and television shows featuring thin women is associated with increased body dissatisfaction and eating disorder symptomatology among females (Anderson, Huston, Schmitt, Linebarger, & Wright, 2001; Bissell & Zhou, 2004; Morry & Staska, 2001; Sands & Wardle, 2003; Thomsen, 2002; Tiggemann, 2013). Experimental research, however, has shown that the effects of exposure to the thin ideal are not universal. In fact, it has been suggested that previous levels of body dissatisfaction moderate women's

responses to media images, in that those who were initially dissatisfied end up being more dissatisfied following exposure (Posavac, Posavac, & Posavac, 1998). Other studies have found no immediate effect of thin-ideal media on body image concerns and eating behaviours (Halliwell, Dittmar, & Howe, 2005). Thus, there is still great debate on the actual impact of media use on body image and eating pathology. Despite the vast array of findings that exist to explore the impact of media use on body image and eating behaviours, very little is known about the impact of social media use, which has actually surpassed television and magazines in terms of daily consumption among youth (Lenhart, 2015; Lenhart, Purcell, Smith, Zickuhr, 2010).

Once youth enter into adolescence, some researchers have suggested that peers have a greater influence on the development of body dissatisfaction and onset of dieting than does media exposure (Ferguson et al., 2014). In fact, it is believed that peers act as a catalyst for the development of these problems (Ferguson et al., 2011; Muñoz & Ferguson, 2012) because youth feel pressure to compete with one another. Research has also shown that youth tend to learn problematic attitudes about food and risky dieting behaviours from one another (Field et al., 1999; 2008; Stice, 1998). Some researchers have also suggested that in the age of social media, social media celebrities (i.e., people with social media accounts with significant followings) can act as a proxy for peers. Thus, social media websites offer an endless amount of peers against which youth compare themselves (Muñoz & Ferguson, 2012).

Some of the most popular social media accounts are diet- and fitness-related (Lenhart, Purcell, Smith, Zickuhr, 2010). The problem with these accounts is that they are often run by uninformed youth who are not formally educated about diet and fitness and who rely on personal anecdotes to share diet and fitness tips. Essentially, many of these accounts promote misleading information about dieting, fitness, and eating disorders. Recent research has determined that

following these accounts is predictive of dieting and disordered eating in adolescents (Ferguson et al., 2014; Syed-Abdul et al., 2013). The active ingredient is believed to be the amount and degree of inaccurate information about dieting and exercise that is espoused by these sites (Syed-Abdul et al., 2013). The consequence of the internalization of this misleading information is dietary misinformation, which is a consequence not only of social media use, but also of online behaviours in general.

Dietary Misinformation. Misinformation refers to a state of believing “erroneous, incomplete or misleading [information] without any scientific basis at all” (Chandon & Wansink, 2007). Misinformation is a consequence of increased reliance on invalid and unreliable online sources of information (Escoffrey et al., 2010). Given that most people rely on websites for information about diet and exercise, and do not generally consider the quality of this information, dietary misinformation is a growing concern (Fox & Duggan, 2012; PewInternet Research Project, 2012). One of the most tangible products of the proliferation of inaccurate sources of health information is diet myths (Chandon & Wansink, 2007).

Diet myths are inaccurate claims about dieting and health that are not based on valid and reliable information. These myths often appear in flashy articles such as “Top 10 Tips to Lose Weight Fast” (Ruderman, 2010), for example “Eating multiple small meals per day speeds up your metabolism” (Fowler, 2012) or “Green coffee beans are a weight-loss miracle” (Dr. Oz, 2014). These ideas are attractive and marketed well. They offer maximum results with minimal effort, instilling a sense of hope in consumers. Over time, these ideas become popular and mainstream, and contribute to the “general knowledge” (however inaccurate) of the dieting culture (Stinson, 2001). Endorsing diet myths may seem innocuous when in fact it is quite harmful.

Research has already demonstrated some negative outcomes of dietary misinformation. The American Dietetic Association (Chandon & Wansink, 2007) released a statement warning about the consequences related to following “food fads” (exaggerated beliefs that certain foods or dieting practices have special health benefits), “health fraud” (promotion of health products for financial gain with false or misleading health claims), and “misdirected claims” (lead consumers to make incorrect inferences about the health benefits of food). These include economic consequences, such as being vulnerable to health scams, and long-term health consequences, such as delaying seeing a doctor or seeking treatment and making health-related decisions based on inaccurate information. Perhaps one of the most insidious qualities of misinformation is that it tends to spread unchecked through social media sites, particularly those geared at lifestyle disease management.

In fact, while misinformation has not received much attention in dieting and disordered eating research, an area where it has been studied at length is diabetes management. Williamson, Hunt, Pope, and Tolman (2000) determined that misinformation (from peers with diabetes and other health professionals, as well as the media) represents a significant barrier to dietary adherence in individuals with diabetes. Another study reviewed how individuals make decisions about their diabetes management after using health-related social networking websites (Weitzman, Cole, Kaci, & Mandl, 2011). They also evaluated the quality and safety of these sites. They determined that the quality of these websites was disconcertingly variable, with most sites often lacking proper references and medical disclaimers, and an over-reliance on community forums that are poorly regulated. On some sites, there were even ads for diabetes “cures” which were not empirically supported. They concluded that one of the consequences of relying on social media in this way is increased misinformation. The deleterious impact of

misinformation has also been observed in severe clinical populations, such as eating disorder patients.

Research has shown that eating disorders patients are sensitive and susceptible to diet rules. They tend to follow strict and rigid rules about food and react intensely and negatively when they inevitably break them (Cooper & Fairburn, 2011). These rules are often the result of inaccurate beliefs (based on myths) about the use of dieting strategies (i.e., believing that purging eliminates any calories from being absorbed by the body; Fairburn, 1995). These myths serve to maintain eating pathology. Despite the importance of understanding misinformation in this population, this construct has not been formally defined or explored and has not been integrated into any theoretical models to understand the development of pathology. This is problematic, given the growing popularity of social media sites that promote eating disorders as a “lifestyle” and provide many of these misinformed tips (Syed-Abdul et al., 2013). Finally, research has shown that even nonclinical individuals (particularly dieters) alter their behaviour according to diet myths (Wansink, 2005), but it is not clear whether this is preceded or followed by a change in attitudes about food or distorted thinking about food.

Perhaps the most significant consequence of misinformation across populations is that it can lead to negative thoughts, beliefs, and attitudes. This idea has already been studied at length across multiple health-related fields such as HIV (Price & Hsu, 1992), smoking (Bansal, Cumming, Hyland, & Giovino, 2004), sexual health (Hoff, Greene & Davis, 2003) and, as previously mentioned, eating disorders (Fairburn, Cooper, & Shafran, 2002). Thus, it is a goal of the current study to determine if dietary misinformation is an antecedent to distorted thinking about food in a nonclinical sample.

Measuring Dietary Misinformation. No measures of dietary misinformation currently

exist. Thus, the current research will also focus on the development and preliminary validation of a psychometric tool to measure dietary misinformation (see study 1). For the purpose of this research, “dietary misinformation” is defined as: the extent to which individuals endorse common diet myths. Given that distorted thinking about food is linked to symptomology in eating disorders patients, it is also a goal of this line of research to determine the link between dietary misinformation, distorted thinking about food, and eating behaviours in a nonclinical sample. Understanding the link between these three constructs (as suggested in Figure 1) could enhance current understanding of the development of subclinical eating pathology and point to more effective ways to structure dieting and disordered eating prevention and intervention strategies (discussed in study 2).

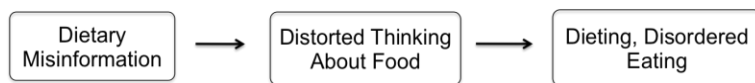


Figure 1. Proposed link between theoretical variables

Theories about Distorted Thinking About Food

Despite the value and importance of understanding distorted thinking about food, there are few theoretical models that have attempted to conceptualize it. Thus, an understanding of the theory behind distorted thinking about food is limited to an understanding of the Dietary Restraint Model (Polivy & Herman, 1985), eating-disorder-specific Cognitive Behavioural Therapy (Fairbun, Cooper, & Shafran, 2008), and broad biopsychosocial models of pathology.

Dietary Restraint Model. The Dietary Restraint Model was developed in order to understand why dieting is an ineffective and inappropriate solution for bingeing (Polivy & Herman, 1985). It posits that dieting leads to weight gain because it requires the acquisition of a “cognitive style” of eating, which is contrary to eating in response to physiological cues of hunger and fullness. Researchers suggested that cognitive control is a risk for disinhibited eating,

since the loss of cognitive control is likely. The result is that individuals alternate between periods of caloric deprivation, followed by periods of bingeing, which eventually leads to weight gain. Furthermore, dieting leads to an increased preoccupation with food, which begins to dominate the dieter's thinking. Garner, Garfinkel, and Bemis (1982) explained that this leads to the "entrenchment of dichotomous thinking", as dieters begin to distinguish foods as either "good" (diet) foods or "bad" (diet-breaking) foods. This rigidity is believed to contribute to the restrict/binge cycle. Thus, researchers determined that the causal link between dieting and bingeing is cognitive control and the resulting distorted thoughts about food. These ideas are consistent with models that have explored this phenomenon in clinical populations, such as eating disorder-specific Cognitive Behavioural Therapy (Fairburn, Cooper, & Shafran, 2002).

Cognitive Behavioural Therapy. The most well supported model for the treatment of eating disorders is eating disorders-specific Cognitive Behavioural Therapy (CBT-e; Fairburn & Cooper, 2014; Fairburn, Cooper, & Shafran, 2008; Hay, 2013). CBT provides a framework for understanding the role that distorted thinking can play in the development of beliefs about the self, and the subsequent behaviours that follow. Researchers have argued that CBT is the most appropriate treatment for eating disorders, given that the symptomatology is mostly cognitive in nature (i.e., the overvaluation of weight and shape; Murphy, Straebl, Cooper, & Fairburn, 2010). In fact, in order to address some of the limitations of bulimia-focused CBT (CBT-BN; which focused largely on reducing bingeing and purging), researchers broadened their understanding of maintaining factors of eating disorders to focus on cognitive factors (Fairburn et al., 2002). Researchers have also shown that eating disorder patients engage in other maladaptive cognitive processes, such as rumination, experiential avoidance, and dysfunctional thinking (Rawal, Park, & Williams, 2010). Thus, altering biased cognitive processes is

considered the mechanism of change in CBT for eating disorders (Beck, 1979; Fairburn et al., 2002). Given the similarity in cognitive processes among clinical and subclinical eating disorder populations, the CBT framework is used to conceptualize and measure distorted thinking about food in the current line of research.

Beck's cognitive theory of depression (Abela & D'Allesandro, 2002; Beck, 2008) conceptualized cognitive distortions as automatic, negative, and inaccurate thoughts that are amenable to change. Beck argued that these negative automatic thoughts are generated by dysfunctional beliefs and are the cause of depressive symptoms rather than the consequence. Cooper and Fairburn (2011) identified a similar link between cognitive and behavioural variables in eating disorder patients. They suggested that CBT theory can explain binge eating as a response to attempts at dietary restriction (whether actual or perceived) which are usually preceded by distorted thoughts about the self, weight, shape, and food (Fairburn et al., 2002). Furthermore, Beck suggested that dysfunctional beliefs (often created by early experience) are activated by future critical events, which could then trigger negative automatic thoughts about the self across a variety of domains. This is consistent with how many researchers view the development of dieting and disordered eating (Fairburn, 2009) as mentioned above, as well as models that consider environmental factors in addition to cognitive processes.

Biopsychosocial Models. Researchers have attempted to offer a parsimonious explanation of how eating pathology develops by addressing the biological, psychological, and societal factors that contribute to its onset. These models tend to differ from one another, as researchers outline different factors believed to contribute to eating pathology. Ricciardelli and colleagues (2003) showed that BMI, gender, and age were significant predictors of the onset of poor body image and weight change strategies. They also found that the main predictor of these

strategies was perceived pressure from parents, peers, and the media. Previous research has also demonstrated that there exists a genetic predisposition for eating disorders (Fairburn & Harrison, 2003); however in many instances it is not clear whether family members with eating disorders may also be exerting an influence through the modeling and teaching of problematic behaviours. Other research has shown that psychological factors such as self-esteem and negative affect are related to eating pathology and act as moderators of other socio-cultural factors (Ricciardelli & McCabe, 2001; Stice, 2001). Thus, within a biopsychosocial model, distorted thinking about food could be viewed as a mediating or moderating cognitive process. Finally, many biopsychosocial models of mental illness view culture as the surrounding influence affecting the onset and maintenance of certain problems.

Within Western cultures (and particularly in North America), there is an obsession with thinness and a fear of fatness, which is constantly reinforced through various media (Polivy & Herman, 2002). The overvaluation of these ideals has also been directly linked to the onset of pathological symptoms (Cooper & Fairburn, 1993; Goldschmidt, et al., 2010; Hrabosky, Masheb, White, & Grilo, 2007). Across cultures, access to various forms of media (including social media) and the adoption of Western values around food and thinness are associated with an increase in prevalence rates of body-dissatisfaction, eating disorders, and disordered eating (Becker, Burwell, Gilman, Herzog & Hamburg, 2002; Calado, Lameiras, Sepulveda, Rodrigueaz & Careera, 2010; Chang, et al., 2013; Syed-Abdul et al., 2013). These studies highlight the interplay between culture, society, and environmental factors.

Although none of these risk factors alone seem to be enough to “cause” eating disorders, they are particularly harmful in combination (Polivy & Herman, 2002). Ultimately, these types of models recognize the important role of cognition in the development and maintenance of

eating problems. The current line of research sought to build on these models by addressing some of their limitations.

Limitations of Theoretical Models

Despite the strengths of the models above, there are some limitations with respect to how they can conceptualize distorted thinking about food in nonclinical samples. First, the Dietary Restraint Model is simply out of date. Since the publication of this model, the way eating disorders and subclinical eating problems are defined and measured has drastically changed (i.e., binge eating is now a diagnosis in the DSM-5; American Psychiatric Association, 2013). Furthermore, this model limited its definition and scope of distorted thinking about food in dieters to dichotomous thinking, but research on eating disorder patients has shown that they engage in a variety of food-related cognitive distortions (e.g., mind reading, catastrophization; Rachman & Shafran, 1999; Shafran, Teachman, Kerry, & Rachman 1999). Thus, it is important to understand the breadth of food-related cognitive distortions in nonclinical samples, as it can improve the detection and understanding of this type of thinking. There has also been an increase in the incidence and variety of disordered eating behaviours among youth, which in part reflects the changing diet culture (Astrup, Larsen, & Harper, 2004). Access to media and time spent consuming various media has also drastically increased since then. Accordingly, this model requires modernization.

In terms of CBT-e, though it is relatively successful at capturing symptomatology and accounting for therapeutic gains in eating disorder patients, it is restricted in its scope. Although it considers developmental factors, cognitions, and behaviours, it does not explicitly account for societal factors that contribute to distorted thinking about food. With respect to biopsychosocial models, although they seem to recognize the vast array of factors that contribute to eating

pathology (including culture, which is often not explicitly addressed in other models), none of them have isolated distorted thinking about food as a specific process to explore. Furthermore, though they acknowledge societal factors, they have not addressed dietary misinformation, which is a very specific type of sociocultural factor that has become more relevant as people rely more on online sources for health information (Fox, 2005; Murero & Rice, 2013). In fact, recent research has demonstrated that social media use contributes to the onset and maintenance of pathological symptoms (Ferguson et al., 2014; Syed-Abdul et al., 2013). Therefore, there is a need for a formal model that considers current societal factors that may contribute to the onset and maintenance of distorted thinking about food, in addition to cognitive factors. Thus, the current research proposes a theoretical model to conceptualize subclinical eating problems that is based on Cognitive Behaviour Therapy (CBT; explained above) and the Theory of Planned Behaviour.

Theory of Planned Behaviour

The Theory of Planned Behaviour (TPB; Ajzen, 1985) is a social cognition model that is used to predict and explain health behaviours (Armitage & Conner, 2006; Conner & Sparks, 1996). The TPB has been used across disciplines to predict healthy behaviour uptake and has generally been successfully applied (Armitage & Conner, 2001). The model essentially predicts that someone's intention to perform or change a behaviour will strongly predict the actual performance of that behaviour (Figure 2).

There are three main factors that comprise a person's intention: (1) Attitude: someone's positive or negative thoughts about a behaviour, which are influenced by a) behavioural beliefs about the consequences of carrying out a behaviour, and b) the outcome evaluation of the consequences. (2) Subjective norms: the degree to which the person feels social pressure to

perform the behaviours, influenced by a) normative beliefs: perceptions about the expectations of significant others, and b) motivation to comply with perceived expectations of others. These sets of beliefs are particularly relevant to dieting, given the social pressure to conform to standards and ideals of beauty and weight (Paxton, Schutz, Wertheim, & Muir, 1999; Stice, Maxfield, & Wells, 2003). (3) Perceived behavioural control (PBC), influenced by a) control beliefs: the degree of personal control the individual perceives he or she has over the behaviour in question and barriers to performing the behaviour, and b) control frequency: how often barriers occur. PBC can also directly predict behaviour, insofar as the perceived control accurately reflects actual control (Hobbis & Sutton, 2005).

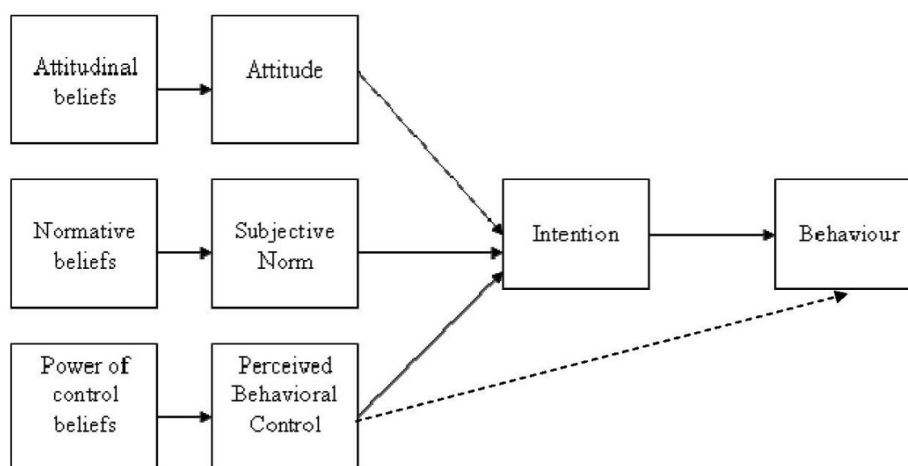


Figure 2. Theory of Planned Behaviour (Ajzen, 1985)

Essentially, the different beliefs underlying attitudes, subjective norms, and PBC influence the development of intentions. The more favourable the beliefs, the stronger the intention and the more likely one is to perform the behaviour. This theory dictates that in order to change behaviour, one must change the underlying beliefs. This model can enhance CBT models by explicitly addressing societal factors that influence behaviours, which in this case refer to inaccurate beliefs about food and dieting (dietary misinformation).

There is emerging evidence for the utility of combining CBT and TPB in the design of behaviour change interventions (Hobbis & Sutton, 2006). In terms of conceptualizing new variables in the current study, the combination of these two approaches has some important benefits. TPB provides an explanation of how social factors affect cognition, which can then translate into behaviour change, while CBT offers a parsimonious explanation of how thoughts, beliefs and behaviours are linked in the current research. Both of these models have also been studied at length for behaviour change across a range of different health problems. Thus, situating misinformation and distorted thinking about food within these models makes them more accessible to change in future interventions designed according to TPB and CBT. Finally, this approach addresses many of the weaknesses that exist in the extant literature regarding behaviour-change interventions (discussed in detail in study 2), and combining both approaches allows for the in-depth exploration of cognition from two distinct perspectives.

Theoretical Cognitive Variables

CBT and TPB have a primary focus on cognition and share the viewpoint that cognitive change is essential for behaviour change. Thus, they are each comprised of cognitive variables; however, these variables are distinct in the way they are conceptualized and measured according to their respective theories. Thus, they represent theoretically different constructs. For example, in TPB, the cognitive variables of “beliefs” refer to specific ideas about the behaviour in question. For example, if the behaviour is “dieting”, an individual’s attitude might be that “dieting is a good idea”, normative beliefs might be “people diet, I know people who diet so dieting is socially acceptable” and control beliefs might be “I believe I can diet effectively”. Together, these three types of beliefs contribute to an increase or decrease in intentions to perform the behaviour (Ajzen, 1985). These differ from “thoughts” in CBT, which are more

automatic and general in nature, not necessarily directly connected to the behaviour in question, and may not influence intentions (Hobbis & Sutton, 2006). For example, if the behaviour in question again is “dieting”, then this may have been preceded by thoughts such as “I must be perfect” or “If I’m not skinny, then I am a failure” (i.e. a cognitive distortion), which is not a belief about the behaviour itself, but rather a cognitive antecedent to the behaviour. Furthermore, CBT suggests different kinds of cognitions at different levels of consciousness, from core beliefs to dysfunctional assumptions and automatic thoughts, all of which may influence behaviour in different ways. The specific nature of the beliefs in TPB is also viewed as a strength of this model, since these are believed to have greater predictive power than non-behaviour-specific cognitions in CBT (Conner & Sparks, 1996). Finally, there may be some similarities in salient beliefs in TPB and automatic thoughts in CBT when these are behaviour-specific, but they would still be assessed and conceptualized as distinct according to each theory and have different functions (Hobbis & Sutton, 2006).

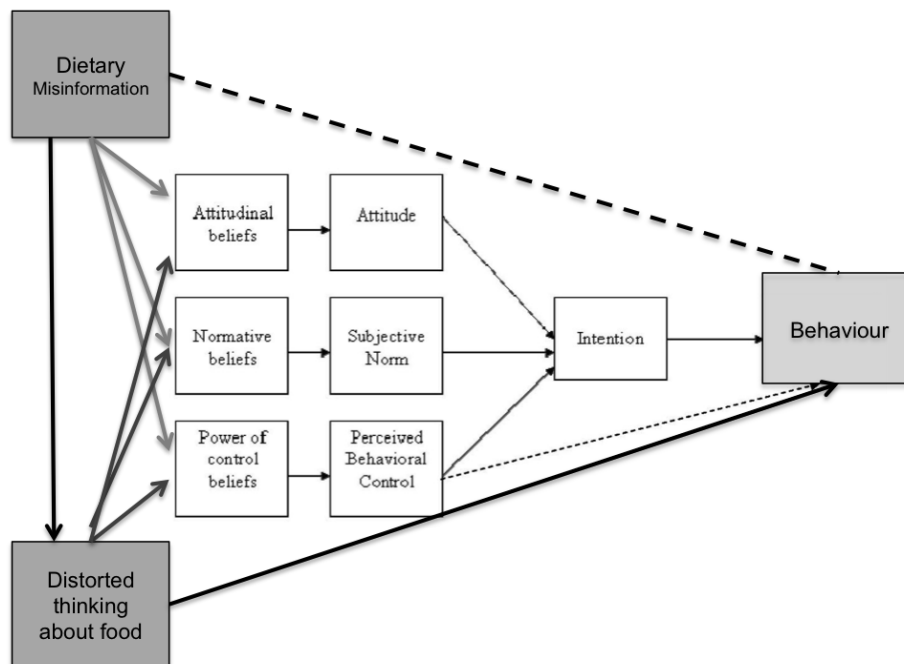


Figure 3. Integrated TPB and CBT Model

The model depicted in Figure 3 represents the hybrid model used to conceptualize new variables in study 1. Essentially, it is theorized that dietary misinformation precedes distorted thinking about food, and they both affect beliefs associated with eating behaviours. This model will be altered slightly in study 2 to reflect the design of the TPB and CBT workshop (described in detail in study 2).

Overview of the Research

The current line of research aims to accomplish two goals: the first is theoretical, and the second is applied. The theoretical goal is to understand, define, and measure distorted thinking about food and to determine its association with other negative outcomes, such as body dissatisfaction and eating pathology in a nonclinical sample. To gain a better understanding of societal factors that influence this type of thinking, a secondary goal of the research is to define and measure dietary misinformation. To achieve these goals, the first study will consist of a psychometric validation of two measures: the Calorie Catastrophizing Scale (CCS) and the Dietary Misinformation Questionnaire (DMQ).

The applied goal is to develop a brief CBT- and TPB-based workshop-style intervention that targets distorted thinking about food and dietary misinformation (study 2). The ultimate goal of the workshop is to reduce these two problems by modifying underlying beliefs around engaging in distorted thinking about food, dietary misinformation, and dieting behaviours. The aim is to encourage participants to engage in more “fair thinking” about food (see study 1), seek out more accurate information about food and to avoid myth-based dieting behaviours. This study will include a detailed review of existing interventions for a range of health problems and populations and why they tend to fail in general. The current research suggests that an understanding of distorted thinking about food and misinformation could enhance existing

interventions and be widely applicable. This intervention will be short-term (2 weeks), thus it is not expected that actual eating behaviour will change significantly. The scope of the current research is limited to understanding distorted thinking about food and dietary misinformation (see Figure 3).

Rationale

The current line of research enhances existing theory in a number of important ways. First, two new variables will be defined and measured (distorted thinking about food and dietary misinformation). Despite the fact that both of these constructs have been shown to be important in understanding eating behaviours in subclinical populations, few studies have formally explored them and so they remain poorly understood. Furthermore, no dieting or disordered eating prevention or intervention studies have investigated whether altering these beliefs and cognitions could lead to improved outcomes. Importantly, most models of subclinical eating pathology ignore current societal factors that impact the development and maintenance of inaccurate beliefs about food. Gaining an understanding of these factors could lead to improved conceptualization of the developmental trajectory of eating pathology and serves to modernize existing interventions.

Study 1

Measuring Distorted Thinking About Food: Development and Validation of the Calorie Catastrophizing Scale

Abstract

Despite the fact that distorted thinking about food has an impact on eating behaviours in nonclinical samples, there are no measures designed to assess this construct. This is particularly problematic given that this type of cognition is associated with negative outcomes, such as eating pathology. Exploring this construct could enhance the current understanding of how subclinical eating problems emerge and are maintained. Thus, the principal goal of this study was to develop and validate the 34-item Calorie Catastrophizing Scale (CCS) in a sample of 433 university students. The CCS measures ten types of distorted thinking (and fair thinking) related to how people process information about their food choices and food environment. As predicted, principal components analysis revealed two underlying factors (distorted thinking and fair thinking). Strong internal consistency was found for both the distortion subscale ($\alpha = .92$) and the fair thinking subscale ($\alpha = .90$) of the CCS. Construct validity for the CCS was established using several standardized measures of negative thinking, eating pathology, and body dissatisfaction. The CCS was shown to be a valid and reliable tool to measure distorted thinking about food in the general population. A secondary goal of this study was to define and measure dietary misinformation and understand its association with distorted thinking about food. The Dietary Misinformation Questionnaire (DMQ) demonstrated strong internal consistency with ($\alpha = .88$) and there is preliminary evidence for an underlying factor structure. Results showed that, as theorized, distorted thinking about food is related to dietary misinformation, supporting the view that distorted thinking about food is determined, at least in part, by societal factors.

Introduction

Distorted thinking refers to a pattern of cognitions that deviate from logical or realistic thinking (Beck, 1963). This construct that has been examined at length across various areas of study including mood and anxiety disorders (Beck, 1963; Cartwright-Hatton, Tschernitz, & Gomersall, 2005), and eating disorders (Fairburn, 2009). Cognitive models of eating disorders generally acknowledge that distorted thinking contributes to the development and maintenance of pathological symptoms (Fairburn, Cooper, & Shafran, 2002). Treatment, therefore, often focuses on the identification and reduction of this thinking.

Distorted Thinking about Food

Distorted thinking about food has not been formally defined in the literature, so for the current research, this term is defined as: having negative and unrealistic thoughts about the nature and impact of certain foods; it is the application of cognitive distortions to food-related situations. This type of thinking is extremely common among eating disorders patients (Fairburn, Cooper, & Cooper, 1986; Garner & Bemis, 1982) and represents a significant barrier to recovery in treatment (Fairburn, Cooper, & Shafran, 2008; Hay, 2013). This type of thinking often manifests as having exaggerated beliefs about food and eating (e.g. believing that one meal will lead to significant weight gain, or labeling oneself as a “failure” if diet rules are broken; Garner, Olmsted, & Bohr, 1982; Lake, Staiger, & Glowinski, 2000). Distorted thinking about food is also distinct from other cognitive distortions common to the eating disorder population, such as the overvaluation of the thin ideal, in that it is food- and eating-specific and related to the function and consequence of eating choices. Given the prevalence of cognitive symptoms among eating disorder patients, a number of clinical measure have been developed in order to further understand the complexity of these constructs. Importantly, the use of these measures on the

general population has suggested that nonclinical individuals also display problematic attitudes and beliefs about food.

Cognitive Antecedents to Eating in the General Population

Many clinical tools have been developed to assess and screen for eating disorders (e.g., Eating Attitudes Test; Garner & Garfinkel, 1979; Bulimia Cognitive Distortion Scale; Schulman, Kinder, Powers, Prange, & Gleghorn, 1986; Three Factor Eating Questionnaire; Stunkard & Messick, 1985, and many others). These measures typically contain a number of subscales assessing various components of eating pathology, with an emphasis on cognition (e.g., the “cognitive restraint” subscale of the TFEQ), beliefs (e.g., the “drive for thinness” scale of the Eating Disorder Inventory; Garner, Olmstead, & Polivy, 1983), and behaviours (e.g., the “dieting” subscale of the EAT). Research has shown that although specific eating disorder behaviours (such as restriction, bingeing and purging, etc.) may vary across eating disorder subtypes, what they have in common is distorted thinking (see “transdiagnostic theory of eating disorders”; Fairburn, Cooper, & Shafran, 2003). Additionally, despite their clinical focus, these measures have been used to understand eating patterns in the general population.

Studies assessing eating concerns in the general population using these measures have shown that cognitive antecedents (e.g., negative attitudes or beliefs about food) affect eating patterns in subclinical populations as well (de Lauzon, et al., 2004). Attitudes and behaviours (as measured by these tools) of individuals with and without eating disorders can look similar, to the extent that certain measures can often result in the false-positive identification of eating disorders (Carter & Moss, 1984). For example, Carter and Moss (1984) distributed the Eating Attitudes Test (EAT) to a large sample of female undergraduates and then followed up with half of the participants via interview. Although many participants endorsed items related to bingeing

(16.6%), purging (1.8%) and a fear of weight gain (53.7%; all of which are eating disorder symptoms), when diagnostic criteria were reviewed, few actually endorsed enough criteria to merit an eating disorder diagnosis. Overall, these studies have suggested that cognitive antecedents are related to eating in both clinical and nonclinical populations; however, scarce research has been conducted on the latter. Investigating food-related cognitions in nonclinical samples could illuminate how subclinical eating problems such as dieting and disordered eating develop in the general population.

Dieting. Dieting is extremely common in North America and the incidence is steadily rising among males and females across the lifespan (Haines & Neumark-Sztainer, 2006). Early research on dieting has shown that food-related cognitive distortion is an important antecedent to unhealthy dieting behaviours in nonclinical samples (Polivy & Herman, 1985; Urbszat, Herman, & Polivy, 2002). These researchers also found that dieters make conscious decisions about food based on these cognitions, rather than physiological factors like hunger and satiety. In this way, the cognition of dieters and eating disorder patients can look similar, highlighting the importance of understanding cognitive distortions across both populations (Polivy & Herman, 1985). Recent research on dieters (also known as “restrained eaters”) has shown that they tend to overeat following a perceived diet transgression (i.e., eating foods deemed as “bad”), which may explain why dieting often leads to weight gain (Urbszat et al., 2002). Indeed, this dichotomous thinking or all-or-nothing thinking is one of the most common manifestations of distorted thinking about food and is viewed as the catalyst to the diet-binge cycle among dieters (Herman & Polivy, 1996; Polivy & Herman, 2002). Despite the evidence that distorted thinking about food leads to disordered eating, no tools exist to measure this construct in nonclinical samples.

Using existing measures of eating disorder symptoms to assess distorted thinking among

nonclinical individuals may be inappropriate and potentially limiting for a number of reasons. First, the vast majority of these measures were initially developed in clinical samples (e.g., Garner, Olmsted, Bohr, & Garfinkel, 1982; Stunkard & Messick, 1985) and in many instances have not been adequately validated in nonclinical samples. Indeed, the cognition-focused subscales of existing measures are geared towards measuring severe eating-disorder-specific behaviours and cognitions such as preoccupation and rigidity (e.g., “I am preoccupied with thoughts of having fat on my body”; EAT; Garner et al., 1982). Though the cognitive profiles of nonclinical and clinical eating disorder patients may look similar, these measures likely do not capture the nature and variability of distorted thinking in nonclinical samples. Furthermore, no measures to date have attempted to formally measure distorted thinking about food as a distinct construct, so deeper exploration of this construct among nonclinical samples is merited.

Exploring the nature of distorted thinking about food in nonclinical samples offers the potential of improving the current understanding of the cognitive antecedents of subclinical eating problems (e.g., chronic dieting and disordered eating). Accordingly, the main goal of this study was to develop and validate a new measure of distorted thinking about food in the general population and to examine the prevalence of such thoughts. In order to gain a better understanding of this construct, societal factors were explored in the conceptualization of distorted thinking about food.

Conceptualizing Distorted Thinking about Food in a Broad Societal Model

It is widely recognized that eating disorders and disordered eating develop in the context of broad social pressures (Polivy & Herman, 2002). Researchers have determined that peers, traditional media (i.e., television, magazines, etc.), and cultural factors (i.e., the thin ideal) all exert pressure on individuals to conform to unachievable standards of weight and beauty and can

trigger body dissatisfaction and eventual dieting and disordered eating (Polivy & Herman, 2002; Stice, 2001; Stormer & Thompson, 1996). New sources of media, however, have received far less attention. There is emerging evidence to suggest that diet- and fitness- related social media contribute to body dissatisfaction through “thinspiration” and “fitspiration” (i.e., guilt-inducing content around weight and shape; Boepple & Thompson, 2016) and promote dieting and disordered eating through misleading information about diet and exercise (Syed-Abdul et al., 2013). The consequence is that consumers become misinformed about food. In general, dietary misinformation is viewed as a consequence of the increased reliance on online sources for diet and health information (Escoffrey et al., 2010; Fox & Duggan, 2012; Weitzman, Cole, Kaci, & Mandl, 2011). Thus, despite the fact that existing models of eating pathology have recognized the role of some societal factors, an emerging concern that remains poorly understood is dietary misinformation.

Dietary Misinformation. Recent lines of inquiry have shown that the consumption of dietary misinformation (i.e., common diet myths, such as “eating fat will make you fat”) can lead to the development of inaccurate beliefs about food (Chandon & Wansink, 2007; American Dietetic Association, 2006), which may itself be an important antecedent to the development of food-related cognitive distortions. This is consistent with other work that has demonstrated that broadly based societal attitudes about food can pressure individuals to label themselves as “good” or “bad” based on the food they eat, which may lead to unhealthy eating behaviours (i.e., avoiding all “bad” foods; Gronning, Scambler & Tjora, 2012; Rich & Evans, 2005). Thus, there is preliminary evidence that dietary misinformation might lead to distorted thinking about food, which in turn precedes dieting behaviours. Researchers have also argued that North American society is characterized by a “dieting culture” (Stinson, 2001), which describes a society with an

“insatiable desire for new information, programs, devices, books, and medicines that will make us healthier, slimmer, fitter, and more aesthetically appealing” (Brownell, 1991). The modern manifestation of this desire is the increased reliance on online sources of diet and health information, which are often misleading and inaccurate (Escoffrey et al., 2010). The consequence is the widespread proliferation and consumption of dietary misinformation.

Given the growing amount of research on dietary misinformation and societal attitudes about food, there is good reason to broaden existing models of clinical and subclinical eating pathology to include a role for dietary misinformation as an antecedent to distorted thinking about food which may ultimately increase the risk for and subsequent emergence of problematic eating behaviours. Accordingly, the current study aimed to embed distorted thinking about food in a theoretical model (see Figure 1) that also explicitly addresses dietary misinformation.

Measuring Dietary Misinformation. Dietary misinformation has mostly been studied in populations with lifestyle diseases such as diabetes and obesity, where it is viewed as a problem that interferes with the uptake of healthy behaviours and adherence to medical protocols (Funnell, 2006; Murrock, Taylor, & Marino, 2013; Osberg, Poland, Aguayo, & MacDougall, 2007; Weitzman et al., 2011). There is also evidence that eating disorder patients are susceptible to misinformation, and often base rigid diet rules around common myths (e.g., “laxatives eliminate all calories, so if I eat I must take laxatives to cancel out the calories”; Fairburn, 1995). Despite the importance of understanding this construct, no research to date has focused on the development of a scale to measure dietary misinformation; however, there has been some research dedicated to understanding problematic emotional beliefs in obese and overweight populations.

The Irrational Beliefs about Food Scale (IBFS; Osberg, Poland, Aguayo, & MacDougall,

2008) was developed from the authors' work with obese and overweight clients, and reflects thoughts and beliefs primarily of people who have trouble with weight control. Items on this scale reflect mostly individual beliefs about the emotional function of food in a person's life (e.g., believing only high-fat foods taste good, eliminating fatty foods will make you sad, eating will lead to happiness, believing that eating can help you overcome loneliness or sadness). Studies using this scale have provided evidence that dysfunctional beliefs about the emotional value of food are predictive of relapse in clinical populations including obese and bulimic patients (Osberg & Eggert, 2012).

Given the restricted scope and target group of this scale, items may not adequately capture the thoughts and beliefs of normal- or under-weight individuals, which would highlight risk factors in developing pathological eating problems in the general population. This scale also does not necessarily capture societal attitudes about food and dieting, as most items reflect the personal and emotional value of food. One of the strengths of this scale, however, is that some items seem to reflect inaccurate beliefs about food (i.e., "Foods like fruits and vegetables have no calories"), but such items were not shown to be distinct from the emotional items. Furthermore, authors found that having rational beliefs about food was not actually consistent with healthy eating behaviours, highlighting the fact that knowledge around healthy eating does not predict healthy eating (Worsley, 2002) and pointing to another weakness of this scale. The current study sought to broaden these findings by examining the role that common diet myths play in the development of food-related thoughts and subclinical eating problems in the general population. Thus, a secondary goal of the current study was to develop and validate a measure of dietary misinformation. Broadening the theoretical framework to include beliefs and attitudes about food is consistent with studies that have used the Theory of Planned Behaviour (TPB) to examine

eating habits in nonclinical samples.

Theory of Planned Behaviour. The TPB is a social-cognition theoretical model of behaviour change which suggests that an individual's behaviour is predicted by their intention to perform that behaviour, which itself is directly influenced by attitudes (and beliefs) about a behaviour, subjective norms (i.e., what relevant people think about the behaviour) and perceived behavioural control (the extent to which a person believes they can perform the behaviour). Several studies have established the importance of beliefs about food in the development of healthy eating habits (Bassett-Gunter et al., 2013; Blanchard et al., 2009; Gronhoj et al., 2013; McEachan et al., 2011).

These studies have generally assessed motivation to engage in healthy eating behaviours when medically necessary (e.g., monitoring calories in obese populations, modifying diets in diabetic populations, etc.) but have not looked at the impact of dietary misinformation or distorted thinking about food on beliefs, attitudes, or perceived behavioural control to perform healthy or unhealthy eating behaviours in nonclinical samples. Addressing this gap could reveal barriers to the uptake of healthy behaviours or factors that influence the onset and maintenance of pathological behaviours. Thus, the proposed theoretical model of the current study is a hybrid CBT-TPB model, which at once addresses the relationship between cognition and behaviour, but also addresses societal factors and individual beliefs that may predict behaviour.

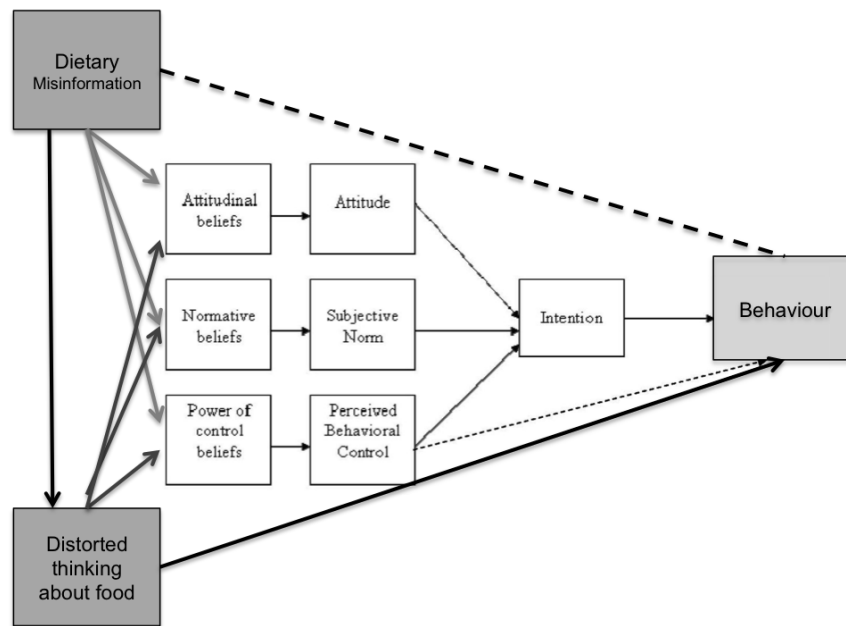


Figure 1. Theoretical Model

Enhancing Existing Models

In the model depicted in Figure 1, both dietary misinformation and distorted thinking about food are assessed as separate, theoretically related constructs. It is theorized that dietary misinformation leads to distorted thinking about food and that they both contribute to the beliefs associated with performing related behaviours, such as dieting and disordered eating behaviours. For example, if an individual were to hear and internalize that “multiple small meals a day increases your metabolism” (diet myth), they are likely to think “if I don’t eat this way I will fail my diet and be fat” (distortion) and then might eat accordingly (dieting behaviour). The development of separate instruments to measure these constructs is theorized to improve current understanding of how subclinical eating problems develop.

Calorie Catastrophizing Scale (CCS). The CCS was developed in order to measure distorted thinking about food in nonclinical samples. To develop this measure, traditional examples of cognitive distortions were altered to reflect food-related concerns and then

formatted into items for the questionnaire. This new measure of distorted thinking about food is similar to existing measures of eating pathology (such as the Eating Attitudes Test; Garner & Garfinkel, 1979, Three-Factor Eating Questionnaire; Stunkard & Messick, 1985, Body Shape Questionnaire; Cooper, Taylor, Cooper & Fairburn, 1987, etc.) in that it attempts to capture cognitive distortion, but differs in several important ways. First, it is not targeted towards a clinical sample, so items were developed with the general population in mind and reflect moderate rather than severe distortion. Second, all items are different reflections of food-related cognitive distortions (see Appendix A for examples) and there is no focus on specific overt behaviours or other cognitive distortions (such as body image perceptual problems, preoccupation with weight, etc.). This will improve the current understanding of distorted thinking about food by examining the variability of this construct. Third, there is a built-in “fair thinking” (see Method) subscale to ensure that the measure properly discerns fair thinking from distorted thinking. This also acts as an internal mechanism of validity (described below).

Dietary Misinformation Questionnaire. The proposed measure of dietary misinformation is the first of its kind (see Appendix B). No known measures currently examine the extent to which individuals endorse common diet myths. To develop this scale, the 54 most common diet myths (methodology described below) were formatted into items and then assessed using a Likert scale. Finally, the model depicted above implies a number of hypotheses about the nature of distorted thinking about food and its relationship to other clinical constructs.

Hypotheses

Hypotheses were tested in order to determine the CCS’s reliability and validity. It was hypothesized that (1) the CCS would demonstrate good reliability with a) good internal consistency and b) strong item-total correlation. It was also hypothesized that (2) the CCS would

demonstrate good validity by showing a) construct validity by revealing two underlying factors (distorted thinking and fair thinking; see below), b) good convergent validity with established measures of eating pathology, low mood and body dissatisfaction, and c) strong negative correlations between the two subscales. It was hypothesized that (3) the DMQ would demonstrate a) strong internal consistency and b) good convergent validity. Furthermore, (4) higher levels of dietary misinformation (as measured by the DMQ) were hypothesized to be associated with greater distorted thinking about food. Finally, (5) greater distorted thinking about food was hypothesized to be associated with greater frequency of unhealthy eating behaviours (as measured by the Problematic Eating Questionnaire; PEQ).

Method

Ethical approval for this project was granted by the University of Ottawa Research Ethics Board.

Participants

The participants for this study were 393 undergraduate students and 40 people from the community who completed an online survey for course credit or to be entered into a draw for a gift certificate. For factor analyses, previous research has recommended a minimum of 5 to 10 cases per item, and thus, this sample size is adequate (Everitt, 1975; Garson, 2008; Gorsuch, 1983; Velicer & Fava, 1998, p. 232). The majority of the participants were female ($n = 366$ or 84.5%) and the age of the participants ranged from 16 to 57 years old ($M = 19.5$, $SD = 4.0$). The majority of the population was white ($n = 308$, or 71.1%) and reported English as their first language ($n = 297$ or 68.6%). Finally, 5.3% of the sample reported ever having received an eating disorder diagnosis (excluded from analyses), and 38.8% reported ever having been depressed in the past. Demographic data is summarized in table 1 below.

Table 1

Descriptive Statistics

	Gender		Age	Ethnicity		EDx	Dep
	Male	Female		White	Other	Yes	Yes
n	67	366	--	308	125	23	168
Mean	--	--	19.5	--	--	--	--
%	15.5	84.5	--	71.1%	28.9%	5.4	38.8

Note: EDx: diagnosis of eating disorder; Dep: report of being depressed

Procedure and materials

The online survey consisted of a battery of measures and took approximately 45 minutes to complete. The survey was hosted on the website Survey Gizmo (surveygizmo.com), a company based out of Boulder, Colorado. No personal information was collected. The following measures were used:

Calorie Catastrophizing Scale (CCS). This measure (Appendix A) was developed for the purpose of this study. It is an attempt to capture distorted thinking about food, or the application of cognitive distortions to food-related situations. Burn's (1999) checklist of cognitive distortions was used to generate items. The descriptions and examples were reviewed and then applied to food-specific situations. For example, with "all-or-nothing thinking", Burns uses an example of considering yourself a failure if a situation falls short of perfect, so the item "If I have even one cookie, I have ruined my diet completely" was generated for the current scale. For "labeling", he used the example of a person making a mistake and calling himself a "loser", so the item "If I don't follow my diet perfectly, it means I'm a failure" was generated. Respondents were asked to state the extent to which they agree with these statements on a six-point Likert scale from (1) strongly disagree to (6) strongly agree. The "neutral" anchor point was avoided here to get a sense of what individuals are inclined to think (Matell & Jacoby, 1972; Leung, 2011). A list of "fair" items (see Fair Thinking below) was also generated for each

cognitive distortion.

The purpose of developing fair items was twofold: first, this acts as an internal mechanism for determining validity (i.e., participants cannot truly endorse both types of items for a given situation). For example, with mind-reading, the item indicating distortion would be “When I eat ice cream (or another treat) in public, I know people are judging me for it” and the fair item would be “When I eat ice cream (or another treat) in public, I don’t know what other people are thinking”. Second, it provides another measurable construct that would be sensitive to change in future intervention. There are twenty-five “distortion” items and nine accompanying “fair” items and thus this measure produces two subscale scores: a distorted thinking score (CCSdis; sum of twenty-five “distortion” items, $\alpha = .92$) and a fair thinking score (CCSfair; sum of nine “fair” items, $\alpha = .90$). These items were generated by the principle researcher and then distributed to a group of graduate and undergraduate psychology students in order to be reviewed for clarity and face validity. Following this step, no items were changed or dropped. Higher scores on the distorted thinking score indicate higher levels of distorted thinking about food. Psychometric qualities of this scale are described in detail below.

Fair Thinking. For the purpose of the current research, the inverse of distorted thinking is considered “fair” thinking. This type of thinking is generally more objective and accurate than distorted thinking, but the word “fair” acknowledges that a thought can be fair without being accurate, or it can be distorted without being decidedly inaccurate. For example, someone might have the thought “When I eat ice cream in public, people are judging me for it” (“mind reading”). This is distorted, in that it reflects negative expectations of others; however, it might actually be true. Conversely, the thought “I don’t know what people are thinking if I eat this” is more fair, but might actually be inaccurate based on previous experience, such as growing up in

a harsh and judging environment. Thus, it is not the accuracy of the thought that is the focus, but the distortion. This is consistent with previous research that has shown that the objectivity and accuracy of a thought are less important than the fairness and function of the thought (Persons, 2005).

Dietary Misinformation Questionnaire (DMQ). This measure (Appendix B) was developed for the purpose of this study. An extensive literature search was conducted to create a list of the fifty-four most common food- and exercise-related diet myths. These myths were cross-referenced with academic studies that confirm, beyond a reasonable doubt, that there is a lack of scientific evidence to support these claims. Participants were asked to state to what extent they agreed with the statements, on a seven-point Likert scale from (1) strongly disagree to (7) strongly agree. The highest possible score is 378. Higher scores indicate greater endorsement of myths, which indicate a higher degree of misinformation. Examples of items include: “Eating multiple small meals per day increases your metabolism”, and “Eating at night makes you gain weight”. The current study demonstrated that the DMQ is internally consistent, with $\alpha = .88$. Psychometric qualities of this scale are described in detail below.

Problematic Eating Questions (PEQ). This questionnaire (Appendix C) was developed for the purpose of this study. It is a frequency questionnaire that assesses how often (in the past month) respondents have performed the behaviours listed. This measure was used in lieu of using an existing behavioural measure because it captures specific disordered eating behaviours that are not necessarily part of eating disorder diagnoses and thus capture more variability in the general population. Furthermore, no current scales are formatted in order to capture behavioural outcomes of distorted thinking about food in nonclinical samples. Thus, this scale was constructed to represent the behavioural manifestation of food-related cognitive distortions, e.g.,

“I thought I ate too many calories, so I had to “exercise it off””. The scale type also captures more variability by probing about the specific frequency of behaviours among respondents. It includes 16 items that ask about unhealthy dieting behaviours (such as avoiding certain foods for being “too fatty”, taking diet pills, skipping meals, etc.) and disordered eating behaviours (e.g., bingeing, purging, fasting, etc.). Respondents are asked how often in the last month have they engaged in these behaviours, ranging from (0) not at all to (5) more than once per day. The psychometric qualities of the scale are described below, but the current study demonstrated that the PEQ is internally consistent with an $\alpha = .91$ and has concurrent validity, given that it was highly correlated with eating pathology (as measured by the TFEQ, $r=.48$, $p<.01$) and poor body image (as measured by the BSQ, $r=.61$, $p<.01$).

Body Shape Questionnaire (BSQ). This 34-item measure (BSQ; Cooper et al., 1987) assesses body dissatisfaction and includes items regarding preoccupations and distress about body size and shape, for example “Have you felt that it is not fair that other women are thinner than you?” Items are rated on a scale of 1 (never) to 6 (always), and higher scores reflect greater body dissatisfaction. The BSQ has demonstrated good reliability and validity (Rosen & Reiter, 1996) with internal consistency of $\alpha = .97$ (Di pietro & Silveira, 2009) and a two week test-retest reliability of .88 for all 34 items. This measure was used to validate the CCS, given that poor body image generally correlates with problematic eating (Ricciardelli & McCabe, 2001; Littleton & Ollendick, 2003; Olivardia, Pope Jr, Borowiecki, & Cohane, 2004) and distorted thinking (Cash & Hicks, 1990; Olivardia, 2001; Rosen, Reiter, & Orosan, 1995). The BSQ has also shown concurrent validity with the Eating Attitudes Test (EAT) and the Multidimensional Body-Self Relations Questionnaire (Rosen et al. 1996). Evans and Dolan (1993) suggested that this measure could be used on men, by switching out the gender of the example items, which was done in this

case.

International-revised Positive and Negative Affect Schedule Short-Form (I-PANAS-SF). This measure (Thompson, 2007) asks participants to report to what extent they generally feel five items describing positive affect (PA; alert, inspired, determined, attentive, active) and five items descriptive of negative affect (NA; upset, hostile, ashamed, nervous, afraid) from (1) never to (5) always. Thompson (2007) reported excellent psychometric characteristics across participants recruited from numerous nations and cultures. The I-PANAS-SF PA subscale had $\alpha = .78$ and the NA subscale had $\alpha = .76$, indicating adequate internal consistency. The correlation between the two subscales was $-.29$ ($p < .01$; Thompson, 2007). For the current study, this measure was used to validate the CCS, given that negative affect is believed to correlate with distorted thinking (Beck, 1963; Teasdale, 1983; Sears & Kraus, 2009) and thus should be negatively correlated with fair thinking (convergent and discriminant validity, respectively).

Three Factor Eating Questionnaire-R18 (TFEQ-R18). This tool (TFEQ-R18; Stunkard & Messick, 1985) is a self-report measure that assesses food intake behaviour. The original version contains 51 items with 4-point response scale (definitely true/ mostly true/ mostly false/ definitely false). For example, “When I feel lonely, I console myself by eating” and “I consciously hold back at meals in order not to gain weight”. These items factor out into three subscales measuring 1) cognitive restraint of eating, 2) disinhibition and 3) hunger. This scale has shown excellent reliability with $\alpha = .93$ for scale 1, $\alpha = .91$ for scale 2 and $\alpha = .85$ for scale 3 (Stunkard & Messick, 1985). The TFEQ-R18 is a shorter version, comprised of 18 items and is easier to use in studies where participants complete many questionnaires (de Lauzon et al., 2004), thus the shorter version was used in the current study. Hyland and colleagues showed that this instrument is valid to use in nonclinical individuals (Hyland et al., 1989). In the current

study, this measure was used to validate the CCS, given that distorted thinking about food should be correlated with eating pathology (convergent validity; Sanftner, Barlow, Marschall, & Tangney, 1995; Polivy & Herman, 2002; Steinglass, Eisen, Attia, Mayer, & Walsh, 2007).

Results

Descriptive statistics and frequencies were calculated for all of the study's variables, in order to discover any outliers, normality issues, and missing values. All assumptions for inferential statistics were met.

Validating the CCS

Principal components analysis. A principal components analysis (PCA) was conducted to evaluate the factor structure of the 34-item Calorie Catastrophizing Scale. A varimax rotation was used. The initial PCA produced three factors. This three-factor solution produced unique loadings (≥ 0.40) for one factor, while some cross-loading occurred for the third factor onto the first factor (see table 1 below). Results indicate that items with the strongest loadings on Factor 3 also had large ($>.30$) loadings on Factor 1, suggesting that those items were also a reasonable indication of variation captured in Factor 1. Thus, a two-factor solution was performed and examined for unique factor loadings. Twenty-five items loaded onto Factor 1 (CCSdis; distorted thinking about food) and nine items loaded onto Factor 2 (CCSfair; fair thinking about food). There were no cross-loadings with this structure. As predicted, these results confirmed the CCS's two-factor structure. Thus, hypothesis 2a) was supported and the CCS was scored two-dimensionally.

Table 2

Factor Loadings of the CCS

	Item	F1	F2	F3
CC8	If I don't follow my diet, I am a failure	0.76		
CC17	It doesn't matter how much I exercise or how well I eat, if I have a treat, the day is ruined for me	0.74		
CC4	If I ate 3 cookies when I planned to eat just 1 cookie, my diet is ruined for the day (if you don't like cookies, think of your favourite treat)	0.72		
CC20	When I have a treat (e.g. ice cream) in public, people think I have no self-control	0.70		
CC7	If I stray from my diet and eat candy when I was not supposed to, that makes me a bad person:	0.69		
CC29	My workout doesn't count if I had a treat on the same day	0.69		
CC2	Eating a treat that I crave, (e.g. ice cream, candy, etc.) once a week will ruin my diet	0.68		
CC16	If I ate well for most of the day, but had a treat, I worry about it	0.65		
CC14	I should not give into food cravings if I have not planned for them	0.62	-0.36	
CC19	When I have lunch or dinner with friends, I know they are judging me for what I order	0.61		
CC22	When I feel fat, I look fat	0.58		0.33
CC28	My day of healthy eating doesn't count if I skipped a workout	0.58		
CC26	I will never be able to enjoy food:	0.56		
CC25	If I ate unhealthily today, it means I will never be able to eat well	0.54		
CC1	Eating fat will make me fat, and being fat will make me unhappy	0.53		0.37
CC32	It is not fair that other people find it easier to eat well	0.48		
CC5	If I ate 3 cookies when I planned to eat just 1 cookie, then I might as well eat the rest of the box (or chips, ice cream, etc.)	0.45		
CC34	Dieting and exercising is so painful for me, that I know it will payoff one day	0.41		
CC33	Other people (roommate, family, partners) make me have a bad diet	0.40		
CC31	I can't help it if my diet is poor	0.65		
CC23	When I crave chocolate, even after a big meal, it is because I am hungry	0.54		
CC9	If I don't follow my diet, I don't worry about it	-0.51	0.46	
CC18	I don't let how much I exercise or what I eat affect my day	-0.53	0.46	
CC6	If I ate more cookies (or other treats) than I had planned, it won't affect my day	-0.62	0.36	
CC24	The way I feel does not affect the way I think about food		0.45	
CC12	I don't feel like I should have to follow any specific diet or exercise plan	-0.32	0.45	0.35
CC30	My diet or exercise does not affect what I do with the other		0.43	
CC3	Having treats once in a while is not a big deal	-0.44	0.59	
CC27	I feel differently about food from day to day		0.40	
CC21	I don't know what people think when they see me eat food		0.35	
CC11	<i>I should be able to lose weight</i>	0.34	-0.43	0.45
CC13	<i>I should not stray from my diet and/or exercise plan</i>	0.34	-0.34	0.41
CC10	<i>I should be able to follow a perfect diet</i>	0.35		0.49
CC15	<i>There is nothing I should not do when it comes to diet and exercise</i>	0.31		0.41

Note: Factor 1 (regular type) represents distortion items, Factor 2 (bolded) represents fair thinking items and Factor 3 (italicized) represents items containing the word "should".

Internal consistency. Strong internal consistency was found for both the distortion subscale ($\alpha = .92$) and the fair thinking subscale ($\alpha = .90$) of the CCS. There were strong item-total correlations for each item (between 0.89 and 0.95), so no items were removed from the scale. Thus, hypotheses 1a) and 1b) were supported.

Psychometrics. Construct validity was assessed through an examination of correlation coefficients presented in Table 2 below. Both subscales of the CCS were correlated with measures of negative thinking, eating pathology and body dissatisfaction in order to determine convergent and divergent validity (see Table 1).

As hypothesized, a higher level of distorted thinking about food (as measured by the CCSdis) was negatively correlated with positive mood (PANAS, $r=-.19$, $p<.01$) and positively correlated with negative mood (PANAS, $r=.37$, $p<.01$). Also as predicted, a greater distorted thinking about food score was negatively correlated with fair thinking about food (CCSfair, $r=-.32$, $p<.01$). It was also positively correlated with body dissatisfaction (as measured by the BSQ, $r=.57$, $p<.01$) and eating pathology (TFEQ, $r=.46$, $p<.01$). With regards to the TFEQ, greater distorted thinking about food was positively correlated with the total score, the cognitive restraint subscale, the unrestrained eating scale, and the emotional eating subscale. Thus, hypotheses 2b) and 2c) were supported.

The fair thinking score of the CCS (CCSfair) was correlated with the same measures to determine its relationship to these variables. As hypothesized, fair thinking about food was positively correlated with the positive thinking (PANAS, $r=.11$, $p<.05$) and negatively correlated with the negative thinking (PANAS, $r=-.33$, $p<.01$). Also as predicted, fair thinking about food was negatively correlated with eating pathology (TFEQ, $r=-.42$, $p<.01$; PEQ $r=-.45$, $p<.01$) and body dissatisfaction (BSQ, $r=-.50$, $p<.01$).

Further correlations were run to determine the relationship among distorted thinking about food, misinformation, and problematic eating (see Table 1). As predicted, greater misinformation (DMQ) was positively correlated with higher distorted thinking about food scores (CCSdis, $r=.46$, $p<.01$) and other measures of eating pathology. Distorted thinking about

food was also positively correlated with frequency of problematic food behaviours (PEQ, $r=.55$, $p<.01$). Thus, hypotheses 3b), 4 and 5 were supported.

Preliminary Validation of the DMQ

An exploratory factor analysis was conducted on the 54 items of the DMQ using principle axis factoring in SPSS. The rotated factor solution demonstrated a 16-factor structure; however, no single factor was dominant, with factor 1 accounting for only 12.43% of the variance. An examination of the items for each factor revealed that items seemed to be clustered according to the subject matter of the myth (e.g. macronutrient-related myths, exercise-related myths, etc.). Removing the factors comprised of few items (i.e. 3 or less) did not significantly improve the structure. A test of internal consistency was also conducted for the DMQ, which revealed an $\alpha = .88$. Thus, hypothesis 3a) was supported. As mentioned above, correlational analyses also revealed that the DMQ was related to measures of distorted thinking and eating pathology.

Table 3

Correlations among all variables

	CCSdis	CCSfair	DMQ	PEQ	PANAS+	PANAS-	TFEQtot	TFEQc	TFEQu	TFEQe	BSQ	Mean	SD
CCSdis	1	-.32**	.46**	.55**	-.19**	.37**	.46**	.36**	.35**	.36**	.57**	77.13	18.10
CCSfair		1	-.09	-.45**	.11*	-.33**	-.42**	-.46**	-.26**	-.28**	-.50**	20.70	6.49
DMQ			1	.27**	-.08	.18**	.17**	.15**	.12*	.13**	.23**	203.68	28.94
PEQ				1	-.09	.42**	.48**	.42**	.35**	.35**	.61**	12.80	12.20
PANAS+					1	-.23**	-.21**	.04	-.25**	-.26**	-.25**	2.94	.79
PANAS-						1	.32**	.23**	.25**	.27**	.47**	2.36	.86
TFEQtot							1	.60**	.87**	.81**	.60**	42.54	8.87
TFEQCRS								1	.20**	.28**	.51**	13.71	3.58
TFEQUES									1	.68**	.43**	21.51	5.27
TFEQEMO										1	.49**	7.30	2.61
BSQ											1	46.45	21.15

** Correlation is significant at the 0.01 level (2-tailed).

* Correlation is significant at the 0.05 level (2-tailed).

Note: PANAS+: Positive and Negative Affect Scale, positive affect subscale; PANAS-: negative affect subscale; CCSdis: distortion subscales; CCSfair: fair thinking subscale; TFEQ: total score of Three Factor Eating Questionnaire; TFEQ-CRS: cognitive restraint subscale; TFEQ-UES: unrestrained eating subscale; TFEQ-EMO: emotional eating subscale; BSQ: Body Shape Questionnaire; DMQ: Dietary Misinformation Questionnaire; PEQ: Problematic Eating Questionnaire.

Mediation Analysis. An exploratory mediation analysis (Preacher & Hayes, 2004) was conducted to further explore the relationship between the new constructs in this study. Key elements of the model presented in Figure 1 were evaluated through an examination of regression analyses. Given that the PEQ was slightly positively skewed, the log of PEQ was used. Results reveal that dietary misinformation (DMQ) is a significant predictor of problematic eating (PEQ; see figure 2). Furthermore, the relationship between dietary misinformation and problematic eating behaviours was mediated by distorted thinking about food (CCSdis). As Figure 3 illustrates, the standardized regression coefficient between dietary misinformation and distorted thinking about food was statistically significant, as was the standardized regression coefficient between distorted thinking about food and problematic eating. The standardized indirect effect was $(.3036)(.0130) = .0040$. The significance of this indirect effect was tested using bootstrapping procedures. Partially standardized and standardized indirect effects were computed for each of 5000 bootstrapped samples, and the 95% confidence interval was computed by determining the indirect effects at the 2.5th and 97.5th percentiles. The bootstrapped partially standardized indirect effect was .0094 with the 95% confidence interval ranged from .0073 to .0116. The bootstrapped standardized indirect effect was .2710 with the 95% confidence interval ranging from .2083 to .3386. Thus, the indirect effect was statistically significant. These results illuminate the relationship among the theoretical constructs in the current study.

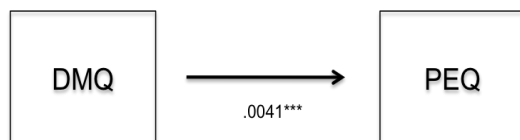


Figure 2. Regression predicting PEQ from DMQ

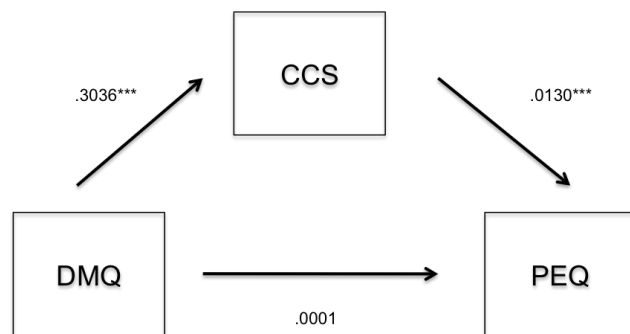


Figure 3. Mediation model

*** $p < .0001$

Table 4a

Regression predicting PEQ from DMQ (total effect)

Variable	R	R^2	β	F/t	p
Model	.2781	.0774		36.13	<0.0001
DMQ	--	--	.0041	6.01	<0.0001

Table 4b

Regression predicting CCS from DMQ

Variable	R	R^2	β	F/t	p
Model	.4538	.2059		111.75	<0.0001
DMQ	--	--	.3036	10.57	<0.0001

Table 4c

Simultaneous regression predicting PEQ from CCS and DMQ (indirect effect)

Variable	R	R^2	β	F/t	p
Model	.6005	.3606		121.24	<0.0001
DMQ	--	--	.0001	.16	.8692
CCS	--	--	.0130	13.80	<0.0001

Discussion

The purpose of this study was to broaden the current understanding of subclinical eating problems in the general population, by exploring important cognitive antecedents and acknowledging the impact that emerging societal factors (i.e., diet myths) have on eating attitudes and behaviours. Thus, this study sought to (1) define, operationalize and validate new measures of (a) distorted thinking about food and (b) dietary misinformation, and (2) to determine the relationship between these two new constructs and eating behaviours in a new theoretical model for nonclinical populations (Figure 1).

Understanding Distorted Thinking about Food

The 34-item Calorie Catastrophizing Scale (CCS) was developed on the basis of research that has shown that, just as with eating disorder patients, cognitive distortions have an impact on eating behaviours in nonclinical individuals. The need for this type of scale is highlighted by the fact that existing measures of eating pathology focus on clinical populations (Garfinkel & Newman, 2001; Garner et al., 1982; Stunkard & Messick, 1985), have not formally defined distorted thinking about food as a distinct construct and thus may not adequately capture food-related cognitions in nonclinical samples. Furthermore, despite the fact that some of these measures have items or subscales that tap into distorted thinking, this is usually limited to a few items, which limits the current understanding of the complexity of this phenomenon. Thus, by examining distorted thinking about food across different types of cognitive distortions, and by adding a “fair thinking” subscale, the CCS is the first measure of its kind to capture the nature and breadth of distorted thinking about food in nonclinical samples specifically.

Calorie Catastrophizing. The CCS is comprised of two subscales: a distortion subscale and a fair thinking subscale. Distortion items were developed using traditional examples of

cognitive distortions (Burns 1999) with a focus on eating and food-related situations. Fair thinking items were developed by determining the inverse of the distortion items. Results from this study provide preliminary evidence that the CCS is both internally reliable and valid. As predicted, distorted thinking about food was highly correlated with low mood, body dissatisfaction and eating pathology, and was negatively correlated with positive mood. These findings are consistent with previous research that has demonstrated this link among clinical populations (Fairburn, Cooper & Shafran, 2003) and subclinical populations (Urbszat et al., 2002).

Additionally, although the CCS is comprised of several different cognitive distortions, including all-or-nothing thinking, mind-reading, and catastrophizing, results from a principle components analysis supported a two-factor structure, as hypothesized. These results support the view that despite the vast number of potential cognitive distortions about food, distorted thinking about food assesses a single underlying factor or construct. These results are consistent with previous research on clinical populations, which has generally conceptualized food-related cognitive distortions in eating disorder patients as a singular construct (Shafran & Robinson, 2004). Findings also confirmed that fair thinking is distinct from distorted thinking in this context. This enhances existing research by suggesting that understanding fair thinking about food is also meaningful in the exploration of how eating problems are avoided or prevented.

Fair thinking. Fair thinking about food was highly correlated with positive mood, but negatively correlated with low mood, body dissatisfaction, and symptoms of eating pathology. This enhances existing research by suggesting that fair thinking may be a novel, reliable predictor of healthy eating. Indeed, previous research has shown that rational beliefs about food (defined as beliefs aligned with dietary recommendations, e.g., “One should strive for 5 servings

of fruits and vegetables a day”; Osberg et al., 2008) and knowledge about food (Worsley, 2002) do not accurately predict actual eating behaviours. Thus, understanding fair thinking about food (i.e., nonjudgmental thinking) could point to factors that protect against the development of distorted thinking about food and eventual dieting and disordered eating. Furthermore, the success of dieting and disordered eating prevention and intervention strategies may hinge on increasing fair thinking about food.

Ultimately, the development of the CCS will provide future researchers with a valid and reliable tool with which to measure distorted thinking about food in nonclinical samples and may be a reliable tool to measure change following intervention. In order to better understand how these distorted thoughts develop in subclinical populations, an exploration of societal factors, such as diet myths, was essential.

Understanding Societal Factors

Dietary misinformation is an emerging concern among clinical and nonclinical populations in North America, but it is particularly relevant to youth and young adults who search for health and diet information online or who subscribe to diet-related social media accounts. Research has begun to demonstrate the pernicious effects of this type of social media, and it is believed that these media promote disordered eating and eating disorders through misleading health information (Syed-Abdul et al., 2013). The consequence of the internalization of this misleading information is dietary misinformation, or the endorsement of diet myths. Importantly, cross-cultural research has also shown that increasing rates of eating disorders, body dissatisfaction, and disordered eating have been linked to increase access to various forms of media, from Asian to Latino populations (Becker, Burwell, Gilman, Herzog & Hamburg, 2002; Calado, Lamieras, Sepulveda, Rodrigueaz & Careera, 2010; Chang, et al., 2013; Syed-Abdul et

al., 2013). Despite the value of understanding this construct, it has received little attention in disordered eating research. Thus, it was a goal of the current study to formally define and measure it.

Dietary Misinformation. The 54-item Dietary Misinformation Questionnaire (DMQ) was developed based on research that has shown that consuming diet myths can lead to the development of problematic attitudes and beliefs about food, leading to adverse behavioural outcomes (Chandon & Wansink, 2007). This research, however, has mostly focused on specific clinical populations (such as eating disorder patients and diabetics; Fairburn, 1995; Murrock, Taylor, & Marino, 2013; Weitzman et al., 2011). Given the ubiquity of diet myths and their potential for harmful consequences, there is a need to understand dietary misinformation and its impact on thoughts and behaviours in nonclinical individuals.

The DMQ was designed to assess a single construct: the endorsement of diet myths. Items were developed based on Internet searches of the most common diet myths (i.e., popular ideas about food that were not supported by research, e.g., “Multiple small meals per day increases your metabolism”). Results show that the DMQ is internally consistent, valid, and reliable. Exploratory analyses also revealed a potential factor structure of the DMQ. Although it was beyond the scope of the current study to investigate this in depth, factor analyses suggest that this scale captures different categories of myths (i.e., items seem to cluster according to the theme of the myths, such as fad diets, meal timing/spacing, exercise, calories, and macronutrients). Future investigation could explore the value of refining this tool and determining the importance of the different factors. In addition, it may be useful to add items that reflect accurate information about food, in order to determine if this is distinct from being misinformed.

As predicted, dietary misinformation was highly correlated with symptoms of eating pathology and distorted thinking about food. This is generally consistent with previous research examining the link between dietary misinformation and behavioural outcomes in clinical populations (Fairburn, 1995; Murrock et al., 2013; Weitzman et al., 2011) and emerging evidence that misleading online information promotes disordered eating (Syed-Abdul et al., 2013). This greatly enhances existing research by clearly delineating the link between dietary misinformation and distorted thinking about food. Although past research has demonstrated the link between misinformation and behaviour, the current study is the first to suggest that distorted thinking about food may be the vehicle for this relationship.

Linking Cognition and Misinformation to Behaviour

The final goal of this study was to understand the link between dietary misinformation, distorted thinking about food, and eating behaviours. In addition to these constructs being highly related, exploratory analyses suggest that dietary misinformation could be a strong predictor of subclinical problematic eating and symptoms of eating pathology, and that this relationship is mediated by distorted thinking about food. This finding suggests that misinformation alone may not be sufficient to lead to unhealthy eating behaviour, but that it might be internalized through a cognitive mechanism such as distorted thinking. This relationship has never been demonstrated in the literature before, and though these findings are preliminary, they point to new considerations that could enhance existing developmental models of eating pathology. Indeed, an understanding of how dietary misinformation develops could point to an even earlier risk factor for the development of eating problems. Further investigation is necessary to fully understand this relationship, though, and would benefit from investigating whether fair thinking about food is also a mediator in a protective way.

Results indicated that distorted thinking about food and dietary misinformation are related to existing clinical measures with strong psychometric properties, including the Body Shape Questionnaire, Three-Factor Eating Questionnaire, and PANAS. These findings support the idea of expanding existing theoretical models of subclinical eating pathology to include both broader societal constructs (such as dietary myths) and distorted cognitions about food. This is consistent with existing research that has shown that broad societal attitudes and messages about food and dieting can have an impact on the onset and maintenance of disordered eating and eating disorders (Levine & Murnen, 2009; Thompson & Heinberg, 1999; Thompson & Stice, 2001), yet it enhances this knowledge by pointing to a concrete aspect of societal messaging, namely, diet myths. Overall, these findings have vast implications for how subclinical eating problems (such as dieting and disordered eating) are conceptualized and intervened on.

Implications

The theoretical model explored in this study has a number of important implications. First, this model has the potential to enhance the current understanding of how eating problems arise in subclinical populations. A natural extension of this idea is that this model may, in fact, improve understanding of how clinical eating disorders arise, given that subclinical eating problems are an important precursor. Furthermore, the CCS and DMQ represent two novel, valid and reliable tools for measuring important antecedents to subclinical eating pathology. These measures will allow researchers and clinicians to better understand, assess and conceptualize subclinical eating problems, which may inform novel ways of designing dieting and disordered eating prevention and intervention programs.

Second, this is the first study of its kind to formally establish the link between dietary misinformation and eating pathology, which was mediated by distorted cognitions about food.

As mentioned above, this finding is preliminary, but opens new avenues and possibilities for intervention. Clinicians and other practitioners will be able to work with these tools to identify specific beliefs and thought patterns upon which interventions can be planned and designed. For example, dieting prevention programs could focus on teaching youth how to process food-related information by finding accurate, reliable sources and thinking critically about diet myths shared through social media and the dieting industry's messages.

Finally, given that DMQ and CCS were directly related to measures of eating disorder symptoms, achieving lasting gains in treatment for eating disorder symptoms may depend in part on addressing pervasive dietary misinformation, which may not be directly addressed in existing treatment for eating disorders. Despite the fact that eating disorders patients are susceptible to diet myths, this construct has not been formally investigated or integrated into intervention programs. In fact, the current “gold standard” treatment for eating disorders is eating disorder-specific CBT and focuses primarily on reducing cognitive distortion and overvaluation of weight and shape (Fairburn, 2009) by improving body image and reducing pathological eating behaviours (such as dietary restriction). Furthermore, these considerations may be applicable to the treatment and prevention of overweight and obesity, given that distorted thinking about food, disordered eating, and dietary misinformation are significant concerns in these populations as well. Thus, we suggest that in addition to these foci, treatments would benefit from directly addressing dietary misinformation and discussing how to avoid and challenge diet myths, which continue to be ubiquitous.

Limitations and Future Directions

There are several important limitations to consider when interpreting the findings of this study. First, two new measures were constructed (the CCS and DMQ) and both were

instrumental in preliminary model testing. Despite findings of excellent psychometric properties, replication of these exploratory findings will be needed to provide greater support for use of these measures and for the hypothesized model. For example, it may be useful to assess the test-retest reliability of this measure, in order to lend further support for its use and to speak to the stability of the construct of distorted thinking about food. Furthermore, the DMQ is currently comprised of 54 items, all of which may not be necessary to capture the construct being measured. As mentioned above, there is also some preliminary evidence for the factor structure of this measure. Thus, this measure should be refined. In addition, future research would benefit from formally testing the proposed model by directly measuring beliefs associated with eating behaviours and examining the relationship among variables with more advanced statistical procedures, such as structured equation modeling, which may include investigating whether reciprocal connections between the components of TPB and distorted thinking exist.

Second, despite the fact that dietary misinformation was measured in this study, sources of misinformation were not formally investigated. It is suggested that one of the main sources of misinformation is social media, but there are many other potential sources. Gaining an understanding of the origins of misinformation will illuminate its pathogenesis. It is possible that in addition to social media, individuals absorb diet myths from family members, peers or other sociocultural sources. Future research would benefit from exploring the course of misinformation as it could enhance the understanding of how it leads to distorted thinking and eating pathology and at what age individuals are particularly vulnerable.

Third, this study was conducted with a non-clinical, university student sample of convenience. Thus, generalizability of findings may be limited in the general population or clinical samples. Yet, since it has been shown that university students generally experience

higher levels of disordered eating behaviours and eating disorder symptomatology (Abrams, Allen, & Gray, 1993; Ackard, Croll, & Kearney-Cooke, 2002; Thome & Espelage, 2004), they are an important population to study in terms of measuring distorted thinking about food.

Furthermore, given that the subject pool is comprised of convenience participants, and the fact that this study was largely exploratory in nature, no statistical corrections or measures of effect size were used. Future research would benefit from collecting a larger and more diverse sample in order to conduct more refined statistical procedures. Additionally, assessing the use of the CCS and the DMQ in clinical populations, such as eating disorders or obesity could lend support for its use in these populations.

Fourth, the design of this study is cross-sectional. This limits the ability to infer causality from analyses (in particular the mediation analysis), and results should be interpreted with caution. Thus, despite the fact that findings suggest relationships in certain directions, it is possible that these relationships are reciprocal, and, for example, behaviour may lead to changes in cognition and not vice versa. Thus, future research could strengthen these findings by conducting further research with a longitudinal design, for example, manipulating either thinking or misinformation and measuring change in all variables over time.

Finally, this sample is predominantly female. Even though research has shown that women are more likely to diet and develop negative body image than males, these problems also occur and are on the rise in males (McCabe & Ricciardelli, 2004). This sample is also predominantly white. Given that attitudes about food and dieting practices may vary culturally (Rozin, Fischler, Imada, Sarubin & Wrzesniewski, 1999), these findings may differ in relevance based on culture or ethnicity of participants. Future research would benefit from attempting to sample more male and culturally diverse participants in order to explore this relationship.

Conclusion

The current line of research offers a novel way of understanding the cognitive antecedents of problematic eating behaviours in subclinical populations. This enhances the theoretical and clinical understanding of the onset and maintenance of pathological eating symptoms within a cognitive-behavioural framework. Findings from this study suggest that dietary misinformation could be a precursor to eating pathology, and this relationship appears to be bridged by distorted thinking about food. This highlights the importance of targeting these cognitions in eating disorder prevention strategies. A reasonable extension of this finding is that prevention and intervention efforts for eating pathology should also target these societal factors that influence the development of cognitive distortions and dietary misinformation for greater change and reduced relapse. Indeed, the treatment and prevention of dieting and disordered eating behaviours may depend on targeting other longer-standing, more chronic beliefs and thoughts about food which are reinforced through social media and may have an earlier onset in childhood than symptoms of eating disorders.

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Study 2

Reducing Distorted Thinking About Food in a Nonclinical Sample: Intervention

Development and Evaluation

Abstract

Distorted thinking about food is common in the eating disorder population. Recent studies have suggested that food-related cognitive distortions are present in the general population as well, and that they are associated with disordered eating, weight management problems, and body dissatisfaction; however, prevention and intervention programs for subclinical eating pathology often neglect this construct. Existing programs have also been shown to be generally ineffective, due in part to the fact that they are often not based on established models of behaviour change. Another weakness of current programs is that they fail to buffer against the negative impact of societal factors, such as dietary misinformation. Thus, the current study sought to build on eating pathology prevention and intervention models by designing a workshop according to the principles of CBT and TPB that explicitly addresses distorted thinking about food and dietary misinformation through various activities including diet myth busting and teaching CBT skills. A randomized controlled trial was conducted with 132 participants from the University of Ottawa. Participants completed an online battery of measures (assessing body image, eating pathology, and mood) at pretest and posttest. Doubly MANOVAs were conducted and revealed that workshop participants experienced a significant reduction in symptoms of eating pathology, body dissatisfaction, food-related cognitive distortions and dietary misinformation. Control group participants experienced no significant change. Results suggest that targeting distorted thinking about food and dietary misinformation within an established model of behaviour change can enhance existing prevention and intervention programs for eating pathology across the spectrum and may lead to greater, longer-standing change.

Introduction

Clinical and subclinical eating-related pathologies are on the rise in North America. Dieting, disordered eating, eating disorders, overweight, and obesity are becoming increasingly prevalent in males and females across the lifespan (Desai, Miller, Staples, & Bravender, 2008; Drummond, 2002; Flament et al., 2014; Hill, 2002; Hudson et al., 2007; Kenardy et al., 2001; O'Dea & Abraham, 2002; Prouty et al., 2002; Thome & Espelage, 2004; Weltzin, et al., 2005, see General Introduction). Effective interventions exist to treat the more severe clinical problems of this spectrum (e.g., CBT-e for eating disorders; Fairburn, 2009; Fairburn et al., 2009), yet few effective strategies exist to prevent or intervene on subclinical eating problems, such as chronic dieting and disordered eating, despite the vast array of available programs. The goal of the current study was to build on existing prevention and intervention theory by addressing the limitations of current programs.

Existing Interventions

Subclinical Eating Pathology. There are numerous types of prevention programs for subclinical eating problems. They tend to be similar in style and structure and generally fall under one of three categories: (1) psychoeducational programs, where the focus is on changing knowledge, (2) skill-based programs, where participants are taught various skills to challenge pathology, and (3) interactive programs, where participants' attitudes are altered through activities (Cororve Fingeret et al., 2006). These programs are typically child- or young adult-focused and are run in elementary schools or universities, respectively.

School-based prevention programs tend to have two goals in common: 1) to teach girls life skills in order to deal with stressors that contribute to the onset of poor body image and dieting, and 2) to teach them media literacy skills in order to challenge the internalization of the

thin ideal, which is associated with negative outcomes (McVey, Lieberman, Voorberg, Wardrop, & Blackmore, 2003; McVey et al., 2010). University-based programs are similar, and are generally centered on educating participants about the consequences of certain eating behaviours, discussing body image/self-esteem, and providing some kind of behaviour modification plan (Baranowski & Hetherington, 2001; Kater, Rohwer, & Levine, 2000; O'Dea & Abraham, 2000; Smolak & Levine, 2001; Smolak, Levine, & Schermer, 1998). Programs may also outline the impact of socio-cultural factors in the development and maintenance of these problems, but this is usually within the context of discussing body image, and is limited to a discussion of media use and the thin ideal, social media and “thinspiration”, among others (McVey & Davis, 2002; Neumark-Sztainer, Sherwood, Collier, & Hannan, 2000). These programs have been reviewed at length and despite having a number of strengths, they have shown only modest benefits.

Success rates for this type of prevention program have been mixed (Stice & Shaw, 2004). Some studies have shown that significant changes can occur in eating attitudes (O'Dea & Abraham, 2000) and other important variables like self-esteem and body image (McVey et al., 2004). Conversely, other studies have shown that changes in knowledge and body image are temporary (Neumark Sztainer et al., 2000) and that these rarely translate into behaviour change (Carter et al., 1997; Killen et al., 1993). Similar results have been found with adult-focused interventions. In fact, one review of 27 studies showed that information- and psycho-education-based interventions are least effective at improving body image and disordered eating among university students (Yager & O'Dea, 2006), and that behaviour-change protocols focused on healthy eating are not effective in the long-term (Ammerman, Lindquist, Lohr, & Hersey, 2002). A large meta-analysis on psychoeducational, skill-based and interactional programs found that they were mostly successful in changing knowledge ($d=0.75$, $p<.001$), had only small net

effects on reducing maladaptive eating attitudes ($d=0.18$, $p<.01$) and had no significant impact on eating behaviours ($d=0.13$, $p=.42$; Cororve Fingeret et al., 2006). Similar findings have been shown in studies on the prevention and intervention of overweight and obesity in children and adults.

Overweight and Obesity. Indeed, reviews of school-based interventions for overweight and obesity have demonstrated that most of them are ineffective (Jeffrey et al., 2000; Stice, Shaw, & Marti, 2006). These programs generally focus on educating students and parents about food, nutrition, and exercise, and many of them implement parts of the program (such as physical education) throughout the curriculum. Numerous reviews of this type of study have repeatedly shown that they fail to produce lasting change (Brown & Summerbell, 2009; Caballero et al., 2003; Donnelly et al., 1996; Luepker et al., 1996).

A review of 38 studies showed that 60% of interventions showed no difference between experimental and control groups over time (Brown & Summerbell, 2009). This review showed that successful interventions typically included physical education as well as dietary education, but their effectiveness was limited to short-term results. Even interventions that included teacher training, modification of school meals, school action plans, physical education and playground activities had no impact on BMI after year 1 (Sahota et al., 2001). Other reviews of long-term school-based interventions have shown similar results, essentially demonstrating that most programs either show no benefit, or that the small benefit they do yield does not last (Caballero et al., 2003; Donnelly et al., 1996; Luepker et al., 1996). Reviews of adult-focused intervention and prevention efforts for overweight and obesity have produced similar results (Brown et al., 2009; Doak, Visscher, Renders, & Seidell, 2006; Jeffrey et al., 2000; Stice, Shaw, & Marti, 2006), leading researchers to question what can account for such poor results.

Why Existing Prevention Programs are Ineffective

Reviewing existing interventions can be challenging from a theoretical perspective, since many studies do not explicitly state the theory upon which they were designed. In fact, critics of health prevention and intervention programs have highlighted that many are not actually based on well-supported models of behaviour change (Dusenbury & Falco, 1997; Nation et al., 2003), which could account for their poor results. Theory can improve interventions by identifying the theoretical constructs that should be targeted and by determining fundamental behaviour change techniques that should be incorporated (Webb, Sniehotta, & Michie, 2010). Thus, using established models of behaviour change could enhance existing dieting and disordered eating prevention and intervention programs, which is a goal of the current study. Another weakness of existing programs is their target audience or population.

Target Population. When reviewing programs it is important to consider the population that is targeted for intervention. A review of these programs showed that successful behavioural prevention and intervention programs are mostly successful in changing behaviours in populations that are already at-risk (Ammerman et al., 2002). This is a common limitation among prevention programs. In fact, most eating disorder prevention research conducted with university students and adults is targeted to students who have already been identified as having eating disorder symptoms (Stice & Shaw, 2004). Furthermore, in most behaviour-change interventions, lower-risk groups are given little attention, even though they actually account for much of the population affected by nutrition-related diseases in North America (Glanz & Mullis, 1988). Therefore, there is a need to arm people who have yet to show signs of eating pathology with tools to boost their resilience, which may be more effective than waiting for individuals to show signs of pathology (McVey et al., 2010). This non-specific vulnerability stressor model

(Cowen & Durlak, 2000) focuses on enhancing general psychological strengths and building resilience in order to prevent health problems (Levine & Smolak, 2006). This model has been applied with success to disordered eating and body dissatisfaction prevention programs (Levine & Smolak, 2006; McVey et al., 2010), yet these programs continue to have limitations. Specifically, another important limitation of existing programs is that they neglect a key construct that has been shown to be important to understanding subclinical eating pathology, namely, distorted thinking about food.

Distorted Thinking About Food. Most theoretical models of severe eating pathology highlight the important role that cognition plays in the development and maintenance of eating problems (see transdiagnostic model, Fairburn, Cooper, & Shafran, 2003; Fairburn & Harrison, 2003), and modifying cognitions is central to intervention. Recent research has shown that distorted thinking about food negatively impacts eating behaviour in subclinical populations (see Study 1), which is consistent with previous research that has underlined the negative consequences of all-or-nothing thinking among dieters (Polivy & Herman, 1985; Polivy & Herman, 2002); however, few if any prevention studies have attempted to modify this construct, pointing to the need for a new design for intervention. Indeed, targeting distorted thinking about food could lead to more lasting change in nonclinical samples. Thus, the current study sought to design an intervention that explicitly targets this cognitive variable in a nonclinical sample, while also targeting a crucial societal factor that has been linked to unhealthy eating in this population, namely, dietary misinformation.

Dietary Misinformation. Research has shown that seeking health information online or through social media leads to the consumption of diet myths, and ultimately misinformation, which in turn is associated with unhealthy eating behaviours across different clinical populations

(Funnell, 2006; Mensing et al., 2007; Murrock, Taylor & Marino, 2013; Osberg, Poland, Aguayo, & MacDougall, 2007; Weitzman, Cole, Kaci, & Mandl, 2011). Without addressing misinformation, current interventions cannot challenge the negative and inaccurate messages about food that people receive when they turn to online sources for information (Weitzman et al., 2011; Williamson, Hunt, Pope, & Tolman, 2000). The ubiquity of the fitness- and diet-focused social media accounts and the proliferation of inaccurate sources of information may in fact compete and interfere with these prevention efforts, particularly those geared at dieting among youth and young adults whose social media use is high (Lenhart, Purcell, Smith, & Zickuhr, 2010).

In sum, the poor results of existing programs highlight a number of important considerations for the design of dieting and disordered eating prevention and intervention programs. First, information- and knowledge-based programs are insufficient and do not lead to lasting change (Molaison, 2002). Second, behavioural strategies can be effective, but they are generally not effective in the long-term (Jepson, Harris, Platt & Tannahill, 2010; Shepherd et al., 2006). Third, since many intervention programs are targeted towards at-risk groups, there is gap in service provision that cannot be addressed with existing interventions (Ammerman et al., 2002). Fourth, these programs ignore an important component of subclinical eating pathology, distorted thinking about food. Fifth, they generally fail to consider the culture and environmental context of the participants by neglecting to address dietary misinformation, which could directly compete with the knowledge generally exchanged through these programs.

Accordingly, the goal of the current study was to develop an intervention targeting distorted thinking about food and dietary misinformation and to evaluate the impact of this intervention on a number of attitudinal, cognitive, and behavioural outcomes. Unlike previous

studies that have primarily targeted food knowledge, body image, dieting behaviours, and media literacy skills, the current intervention targeted the thoughts, beliefs, and attitudes likely to govern problematic eating behaviours in nonclinical samples. Most importantly, the current intervention was designed according to well-established models of behaviour change, the Theory of Planned Behaviour (Ajzen, 1991) and Cognitive Behaviour Therapy (Beck, Rush, Shaw & Emery, 1979).

Theory of Planned Behaviour

The Theory of Planned Behavior (TPB; Ajzen, 1991) is one of the most robust theories of behaviour change that can be used to explain the mechanisms by which people change their behaviour. It can also explain how people engage in strategies to change their thinking patterns in the context of CBT and related prevention programs. This theory is a cognitive or information-processing approach which postulates that in order to perform a particular behaviour, one must have the intention to perform that behaviour and this intention is influenced by three categories of cognitive antecedents: attitudes, subjective norms, and perceived behaviour control, which themselves are influenced by beliefs (Ajzen, 1991; see Figure 1). This model has a number of advantages over other health behaviour models, such as the Health Beliefs Model (Hochbaum, Rosenstock, & Kegels, 1952), which has shown poor predictive ability across a number of health behaviours (Harrison, Mullen, & Green, 1992), and the Transtheoretical Model of Behaviour change, which has been criticized for weakly defining the stages of change, which has called into question the validity and reliability of staging algorithms (Adams & White, 2004; Brug, Conner, Harre, Kremers, McKellar, & Whitelaw, 2004; Spencer, Pagell, Hallion, & Adams, 2002).

The TPB has successfully been applied in order to understand a range of health-related behaviours including dieting (Garcia & Mann, 2003; Nejad et al., 2005), healthy eating (Gratton

et al., 2007; Kothe et al., 2012; Lien, Lytle, & Komro, 2002), and eating pathology (Garcia & Mann, 2003; Nejad et al., 2005; Pickett et al., 2012). Research has repeatedly shown that this model is very effective at explaining the relationship between cognitive antecedents, intentions to change behaviour, and the actual behaviour change that follows in diet and exercise related interventions (Godin & Kok, 1996; Kelly, Melnyk, & Jacobson, 2011). In fact, the TPB is being used more and more frequently to inform the development of health behaviour change interventions (Hardeman, et al., 2002; Stead, Tagg, MacKintosh, & Eadie, 2005).

TPB Interventions. Developing an intervention using the TPB has been described in detail elsewhere (Ajzen, 2006; Hobbis & Sutton, 2005; Sutton, 2007). Essentially, the first step involves defining the target population and eliciting beliefs associated with the outcome behaviour. For the current intervention, two types of outcome behaviours were targeted: 1) changing distorted thinking about food, and 2) engaging in behavioural strategies (i.e., thought tracking and challenging) in order to change this thinking. The second step is to design a questionnaire (TPBQ; see below) based on these beliefs in order to determine pre-intervention attitudes, subjective norms, perceived behaviour control, and intentions and to measure post-intervention changes in these variables. Finally, the last step is to design and carry out an intervention (described below) that targets these beliefs in order to change these cognitive antecedents thereby altering intentions and actual behaviour. Given that the main target of the intervention is distorted thinking, this intervention was also designed according to Cognitive Behaviour Therapy (CBT; Beck, Rush, Shaw, & Emery, 1979) in order to determine the strategies that could lead to change.

Cognitive Behaviour Therapy

CBT operates by addressing how individuals process information: through automatic

thoughts, underlying assumptions, and core beliefs (James, 2001), and it is one of the most tested behaviour-change theories in health care interventions to change diet, exercise, and lifestyle (Spahn, et al., 2010). In order to change TPB beliefs in the current intervention, CBT strategies such as thought tracking and challenging were used. The merits of combining TPB and CBT have been discussed at length elsewhere (Hobbis & Sutton, 2005; Fishbein & Ajzen, 2005) but essentially, CBT can enhance a TPB-based interventions by identifying strategies to alter beliefs (that determine attitudes, subjective norms, and PBC), while TPB can enhance CBT by explaining how people engage in strategies to change their thinking. Figure 1 below illustrates this hybrid model.

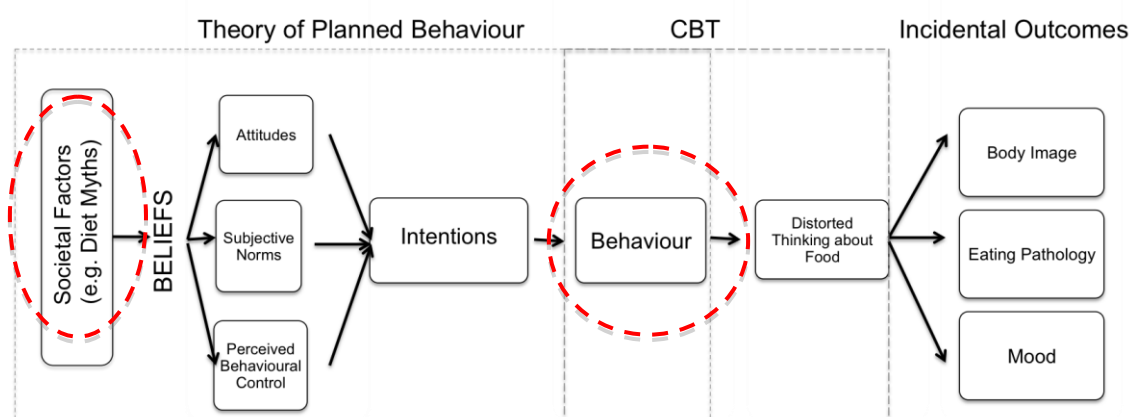


Figure 1. Theoretical Model of Change

Hybrid Model

The model depicted above illustrates the central aspects of the intervention designed in the current study. The targets of the intervention are identified in the model with dashed circles. Essentially, the current intervention (described below) focused on two goals. The first goal is to target beliefs (i.e., societal factors; dietary misinformation) associated with distorted thinking about food within the context of TPB. The second goal is to teach participants to track food-related cognitions (behaviour) and to challenge them within the context of CBT. By combining

these two strategies, the overarching goal is to increase intentions to engage in fair thinking about food and to monitor food-related cognitive distortions. The predicted outcomes include increases in TPB antecedents (i.e., attitudes, subjective norms, PBC) and a reduction in distorted thinking about food. Given that body image, eating pathology and mood are also associated with this variable, it is predicted that these will change despite not being directly targeted for intervention.

Summary of the Current Research and Hypotheses

To address the limitations of existing interventions in the extant literature, the current study focused on the development and validation of a workshop-style intervention. This intervention is based on TPB and CBT and targets dietary misinformation and distorted thinking about food. Specifically, a randomized controlled trial was conducted in order to investigate whether attending the workshop lead to change across a number of variables. Thus, two groups of hypotheses were examined in this study: tests of the validity of the TPB model and tests of the effectiveness of the intervention. Specifically, it is hypothesized that 1) the TPB questionnaire will demonstrate good psychometric qualities by having a) good reliability and b) good validity, and 2) intentions to engage in fair thinking about food will predict level of distorted thinking about food at time 1. It is also hypothesized that 3) attitudes, subjective norms, and PBC will predict intentions to change thinking about food and that 4) participants in the intervention group will experience significant increases in intentions to change their thinking about food and engage in thought monitoring activities. Finally, it is hypothesized that 5) participants in the intervention group will experience a greater reduction in distorted thinking about food over time when compared to controls and that 6) they will also experience a greater reduction in a) misinformation, b) symptoms of eating pathology, c) body dissatisfaction and d) low mood.

Method

Ethical approval for this project was granted by the University of Ottawa Research Ethics Board.

Participants

Participants for this study were 145 undergraduate students recruited from the University of Ottawa campus, using the Integrated System of Psychological Research (ISPR). The ISPR is an online system that allows first year psychology students to participate in research being conducted by graduate students and faculty. A power calculation using the G*Power program indicated that in order to assess pre-post differences between the intervention and control groups on hypothesized variables with a power of 0.80, 112 participants are required (approximately 56 participants per group). Thus, this sample size is adequate.

The majority of the participants were female ($n = 130$ or 90%) and the age of the participants ranged from 17 to 39 years old ($M = 19.7$, $SD = 3.3$). The majority of the sample was white ($n = 102$, or 70%) and reported English as their first language ($n = 94$ or 65%). Finally, 11.7% ($n = 17$) of the sample reported having received an eating disorder diagnosis in the past and therefore were excluded from analyses, while 10% ($n = 14$) reported having received a diagnosis of depression in the past.

Intervention

The “Calorie Catastrophizing” workshop (Appendix E) is based on cognitive behavioural principles and techniques from various CBT protocols, such as those of Jacqueline Persons and Aaron Beck (Beck et al., 1979; Persons, 2005). During the 60-minute workshop, the facilitator begins by normalizing the experience of consuming diet myths, participating in diets, developing negative attitudes about food and problematic eating behaviours. Participants are encouraged to

elicit the specific beliefs they have about these ideas and engaging in fair thinking about food. The workshop then focuses on debunking common diet myths. Participants are educated about the concept and consequences of distorted thinking, and how cognitive distortions can be applied to food. They are encouraged to think of ways to challenge this type of thinking by examining the evidence that supports or contradicts such thinking. Participants are then encouraged to think of how knowledge, thinking, and behaviour are connected. The workshop is intended to be interactive, and participants are encouraged to participate through different activities (such as true or false quizzes, matching games, etc.). Finally, the last part of the workshop involves explicitly teaching CBT skills. Participants are given a workbook to complete for fourteen days as a method of self-monitoring and thought tracking (i.e., light homework). In the booklet they are asked to list myths they have heard or acted on that day and how this might be harmful. They also list the specific cognitive distortions that they experienced throughout the day relating to food and how they challenged them. The purpose of the booklet was to encourage participants to practice CBT skills.

Sham Treatment

The control group for this study received an active control (Boot et al., 2013) treatment, which consisted of watching a 20-minute video about the Canada Food Guide (CBC, 2012), followed by a short questionnaire about what they had watched. Although expectations were not measured at the outset, both control and experimental group participants received the same description of the intervention, which indicated that they would be “learning about food”. Thus, it is reasonable to assume that expectations across participants were consistent.

Measures

Three different categories of measures were administered together as an online battery for

this study: (1) Model measures, which include tools to specifically measure the new constructs of this study, (2) Theory of Planned Behaviour (TPB) measures that capture the cognitive antecedents to behaviour and whether they change following intervention, and (3) Outcome variable measures, in order to determine whether clinical variables such as eating pathology, mood, and body dissatisfaction change following intervention.

Model Measures. The main components of the proposed theoretical model of this study are dietary misinformation, distorted thinking about food, and subclinical eating problems. Thus, the following measures were used.

Dietary Misinformation Questionnaire (DMQ). This self-report tool (Appendix B) was developed for previous research (see study 1) and assesses the extent to which individuals endorse common diet myths. Higher scores indicate more myths believed, which indicate a higher degree of misinformation. Examples of items include: “Eating multiple small meals per day increases your metabolism”, and “Eating at night makes you gain weight”. In the current study, the DMQ was used as a baseline and outcome measure for dietary misinformation. The DMQ has been shown to be internally consistent, with $\alpha = .88$ (see study 1) and has demonstrated good convergent validity by having a strong correlation with eating pathology (as measured by the TFEQ; $r = .17$, $p < .01$) and body dissatisfaction (as measured by the BSQ; $r = .23$, $p < .01$). A previous study of over 400 undergraduate students revealed that this population is severely misinformed about diet and exercise. For example, 65.6% of participants agreed that “Calories eaten at night turn to fat”, 78.6% believed that “Certain foods can speed up your metabolism”, and 55.1% believed that “Skipping a meal will slow down metabolism” (see study 1). Furthermore, this research showed that misinformation is linked to subclinical eating pathology, and that this relationship is mediated by distorted thinking about food.

Calorie Catastrophizing Scale (CCS). This scale (Appendix A) was developed for the purpose of previous research (see study 1). It captures distorted thinking about food, or the application of cognitive distortions to food-related situations. There are twenty-five “distortion” items and nine accompanying “fair” items and thus this measure produces two subscale scores: a distorted thinking score (CCS_{dis}; sum of twenty-five “distortion” items, $\alpha = .92$) and a fair thinking score (CCS_{fair}; sum of nine “fair” items, $\alpha = .90$). Higher scores on the distorted thinking score indicate higher levels of distorted thinking about food. In the current study, the CCS was used as a baseline and outcome measure for distorted thinking about food. Recent research has shown that in addition to being internally consistent, this scale has demonstrated strong construct validity by being strongly associated with measures of eating pathology symptoms (as measure by the TFEQ, $r=.46$, $p<.01$), mood (as measure by the PANAS, $r=.37$, $p<.01$, and body dissatisfaction (as measured by the BSQ, $r=.57$, $p<.01$; see study 1).

Problematic Eating Questionnaire (PEQ). This tool (Appendix C) is a frequency questionnaire that assesses how often (in the past month) respondents have performed the behaviours listed. It includes 16 items that ask about unhealthy dieting behaviours (such as avoiding certain foods for being “too fatty”, taking diet pills, skipping meals, etc.) and disordered eating behaviours (e.g., bingeing, purging, fasting, etc.). Respondents are asked how often in the last month have they engaged in these behaviours, ranging from (0) not at all to (5) more than once per day. In the current study, the PEQ was used as a baseline and follow up measure for unhealthy eating behaviours. Previous research has shown that the PEQ is internally consistent with an $\alpha = .91$ and has concurrent validity, given that it is highly correlated with eating pathology (as measured by the TFEQ, $r=.48$ $p<.01$) and poor body image (as measured by the BSQ, $r=.61$, $p<.01$). As with study 1, this scale was used in lieu of established measure of

eating pathology in order to gain a better understanding of subclinical eating problems in the general population.

Theory of Planned Behaviour Measures. The Theory of Planned Behaviour is the established model of behaviour change that was used in the design of the current intervention, and proposes that three types of cognitive antecedents (attitudes, subjective norms and perceived behavioural control) determine an individual's intentions to engage in a behaviour, which is the most proximal determinant of behaviour (Ajzen, 1985). Given that the intervention targets these antecedents, measuring them at baseline and post-intervention is essential. Thus, the following measure was included in the online battery.

Theory of Planned Behaviour Questionnaire (TPBQ). The TPBQ was developed based on Ajzen's (2006) guidelines. It consists of 20 items on a 7-point likert scale and assesses attitudes, subjective norms, perceived behaviour control, and intentions around 1) reducing distorted thinking about food and 2) completing self-monitoring activities about distorted thinking about food (and how to challenge them) for the next two weeks. This tool also measures past behaviour, given that some researchers have suggested that past behavior is also a strong determinant of future or current behaviour (Norman & Conner, 2006). Greater scores indicate increasingly positive antecedents and intentions to engage in these behaviours. Results from the current study were used to establish internal consistency and construct validity (see below).

Clinical Outcome Measures. In order to determine the efficacy of the current intervention, outcome measures were included to assess the indirect or incidental effects of the intervention. Specifically, despite not being directly targeted in the intervention workshop, the following measures were used to assess changes in body image, eating pathology symptoms and mood.

Body Shape Questionnaire (BSQ; Cooper et al., 1987). This scale is a 34-item self-report measure of body dissatisfaction. It includes items regarding preoccupations with and distress about body size and shape, for example “Have you felt that it is not fair that other women are thinner than you?” Items are rated on a scale of 1 (never) to 6 (always), and higher scores reflect greater body dissatisfaction. The BSQ has demonstrated good reliability with internal consistency with $\alpha = .97$ (Di pietro & Silveira, 2009) and a 2 week test-retest reliability coefficient of .88 for all 34 items. Concurrent validity has been demonstrated through strong correlations with the Eating Attitudes Test in women with subthreshold concerns (Cooper et al., 1987). Evans and Dolan (1993) demonstrated the reliability of using two 16-item shortened forms of the BSQ. In the current study, the BSQ was used as a baseline and outcome measure of body dissatisfaction.

Three Factor Eating Questionnaire -R18 (TFEQ-R18; Stunkard & Messick, 1985). This scale is a self-report measure that assesses food intake behaviour and disordered cognitive processes. The original version contains 51 items with 4-point response scale (definitely true/mostly true/mostly false/definitely false). For example, “When I feel lonely, I console myself by eating” and “I consciously hold back at meals in order not to gain weight”. These items factor out into three subscales measuring 1) cognitive restraint of eating, 2) disinhibition and 3) hunger. This scale has shown excellent reliability with: $\alpha = .93$ for scale 1, $\alpha = .91$ for scale 2 and $\alpha = .85$ for scale 3 (Stunkard & Messick, 1985). The TFEQ-R18 is a shorter version, comprised of 18 items and is easier to use in studies where participants complete many questionnaires (de Lauzon et al., 2004). Hyland and colleagues showed that this instrument is valid to use in normal individuals (Hyland et al., 1989), by reconfirming its factor structure. In the current study, the TFEQ-18 was used as a baseline and outcome measure of eating pathology.

Eating Attitudes Test (EAT-26; Garner & Garfinkel, 1979; Garner, Olmsted, Bohr & Garfinkel, 1982). The EAT-26 is a self-report measure that assesses symptoms and characteristics of eating disorders. The original version included 40 items, but Garner later validated a 26-item version that was found to be useful for determining eating attitudes in nonclinical samples. Items on the EAT-26 are rated on a 6-point frequency scale ranging from (0) Never to (3) Always. Totals are then summed and three subscale scores are computed for dieting, bulimia and food preoccupation and oral control. Examples of items include: “I feel that food control my life”, “I avoid eating when I am hungry”, and “I feel extremely guilty after eating”. The EAT-26 has both demonstrated good reliability with an internal consistency of $\alpha = .85$ and validity with strong correlations with other measures of eating concerns (Garner & Garfinkel, 1979; Garner, Olmsted, Bohr, & Garfinkel, 1982). For the current study, the EAT was used as a baseline and outcome measure for eating pathology and attitudes about food.

Scale for Positive and Negative Affect (SPANE; Diener et al., 1985). The PANAS scales (which were used to measure mood in study 1) have been criticized for measuring high-valence emotions (excited, cheerful), which tend to be rarer than others measured simultaneously (e.g., happy; Diener et al., 1985). To address these weaknesses, Diener and Emmons (1985) created the Scale for Positive and Negative Affect, which measures emotions that are more similarly valenced to one another. This is a 9-item, 7-option Likert-type scale questionnaire with 4 positive affect items and 5 negative affect items. This measure produces two unique factors (positive and negative affect) that have strong convergent and discriminant validity with other measures of positive and negative emotions, such as the General Well-Being Schedule (Diener & Emmons, 1985; Fazio, 1977). For the current study, this scale was used as a baseline and outcome measure for mood.

Procedure

This study was completed in three steps. First, all participants completed an online survey comprised of the measures listed above, in addition to some demographic questions (e.g. age, gender, ethnicity, etc.). A detailed consent form, outlining the purpose and context of the study was included at the start of the questionnaire. Participants were required to acknowledge that they had read and understood the terms before they moved on to the first question. Responses were anonymous, no personal data were collected. The survey was posted on the survey website Survey Gizmo (surveygizmo.com), a company based out of Boulder, Colorado.

Next, participants were randomized into an experimental (workshop) group or control (sham treatment) group using randomizer.org. Identical instructions were sent for study participation to each participant, indicating the time and location of the treatment. Once all participants had arrived, the principle researcher administered the workshop to those who had been assigned to the experimental group, while a research assistant delivered the sham treatment to the control group in the room next door. The workshop and sham treatment were delivered in small groups of 6-8 participants. At the end of the sham treatment, control group participants were offered the chance to participate in the workshop once they had completed the trial. Finally, two weeks after the workshop (or sham treatment), both groups completed the battery of measures again. All ISPR students were compensated with two points towards their course fulfillment.

Results

Results are organized according to psychometric analyses, theoretical model testing, and intervention outcomes.

Psychometric Analysis

Psychometric analyses were conducted to establish validity and reliability of the TPBQ. Chronbach's alphas were computed for the attitudes, subjective norms, perceived behaviour control, and intentions to reduce distorted thinking about food and increase fair thinking. Correlations were computed between TPB variables and clinical and well-being variables as hypothesized to establish convergent and discriminant validity. It was not hypothesized that this scale would reveal two underlying factors, despite the fact that it assesses both intentions to change thinking and intention to engage in thought tracking. Thus, items were collapsed.

A test of internal consistency for the TPBQ questionnaire revealed a Cronbach's alpha of .92 and a Spearman Brown split-half reliability coefficient of .90. Correlations suggest that the TPBQ is valid. Specifically, the total score of the TPBQ was correlated with the CCS ($r = -.29$, $p < .01$), the PEQ ($r = -.25$, $p < .01$), the negative affect subscale of the SPANE ($r = -.17$, $p < .05$), the positive affect subscale of the SPANE ($r = .24$, $p < .01$), as well as its subscales: TPB ATT ($r = .91$, $p < .01$), TPBSN ($r = .47$, $p < .01$), TPBPBC ($r = .89$, $p < .01$), TPBINT ($r = .88$, $p < .01$) and TPBPST ($r = .38$, $p < .01$). Individual subscales were also strongly positively correlated with each other (see Table 1). Thus, hypotheses 1a and 1b were supported.

Table 1

Model and TPB Correlations at Time 1

	CCS	CCSfair	DMQ	PEQ	TPBatt	TPBsn	TPBpbc	TPBint	TPBpas	Mean	SD
CCS	1	-.42**	.46**	.54**	-.28**	.03	-.39**	-.23**	-.28**	64.82	21.81
CCSfair		1	-.15	.01	-.12	-.04	.06	-.09	.17*	38.56	9.93
DMQ			1	.25**	-.12	-.01	-.18*	-.09	-.12	116.89	15.74
PEQ				1	-.26**	-.01	-.33**	-.22*	-.16	31.46	11.59
TPBatt					1	.22**	.76**	.77**	.26**	22.87	5.28
TPBsn						1	.18*	.23**	.25**	11.13	3.41
TPBpbc							1	.78**	.39**	16.69	4.11
TPBint								1	.31**	10.65	3.35
TPBpast									1	6.58	3.60

** Correlation is significant at the 0.01 level (2-tailed).

* Correlation is significant at the 0.05 level (2-tailed).

Note: CCS: distortion subscale; CCSfair: fair thinking subscale; DMQ: Dietary Misinformation Questionnaire; PEQ: Problematic Eating Questionnaire; TPB: Theory of Planned Behaviour; ATT: attitude; SN: Subjective Norms, PBC: Perceived Behavioural Control; PST: Past behaviour;

Table 2

Outcome Variable Correlations at Time 1

	BSQ	TFEQ	TFEQcr	TFEQue	TFEQem	EATdiet	EATbul	EATcntl	SPANE+	SPANE-	Mean	SD
BSQ	1	.54**	.48**	.30**	.43**	.71**	.60**	.25**	-.43**	.48**	50.79	20.89
TFEQ		1	.53**	.84**	.76**	.49**	.48**	.06	-.29**	.26**	43.44	8.41
TFEQcr			1	.06	.18*	.61**	.33**	.33**	-.17*	.20*	14.79	3.63
TFEQue				1	.57**	.24**	.40**	-.05	-.16	.19*	21.34	5.32
TFEQem					1	.20*	.28**	-.14	-.36**	.16	7.31	2.66
EATdiet						1	.68**	.43**	-.26**	.34**	8.92	8.33
EATbul							1	.30**	-.27**	.35**	1.54	2.57
EATcntl								1	-.08	.33**	2.39	2.95
SPANE+									1	-.46**	22.45	4.18
SPANE-										1	16.37	4.43

** Correlation is significant at the 0.01 level (2-tailed).

* Correlation is significant at the 0.05 level (2-tailed).

Note: BSQ: Body Shape Questionnaire; TFEQ: total score of Three Factor Eating Questionnaire; TFEQ-CRS: cognitive restraint subscale; TFEQ-UES: unrestrained eating subscale; TFEQ-EMO: emotional eating subscale; EAT: Eating Attitudes Test; EATdiet: dieting subscale; EATbul: bulimia subscale; EATcntl: control subscale; SPANE +: positive affect subscale; SPANE -: negative affect subscale

Theoretical Model Testing

To determine the suitability of the TPB to predict the performance or change of behaviour, simultaneous regressions were computed on the three antecedent variables (attitudes, subjective norms, perceived behavioural control) to predict intentions and then intentions to predict behaviour. This analysis was conducted at time 1 with all participants in order to collapse groups and increase statistical power. For the purpose of this analysis, TPB variables were split into intentions to change thinking and intentions to track thoughts and only the former were used, given that there is no outcome measure for tracking thoughts. These regressions were conducted using path analysis in AMOS (see Figure 2). At time 1, intentions to engage in fair thinking predicted a lower CCS score (standardized coefficient of $-.23$, $p < 0.05$). At this time, most of the variance in intentions was accounted for by PBC (standardized coefficient of $.45$, $p < 0.05$) but attitudes were also significant in determining intentions (standardized coefficient of $.41$, $p < 0.05$). Thus, hypotheses 2 and 3 were supported.

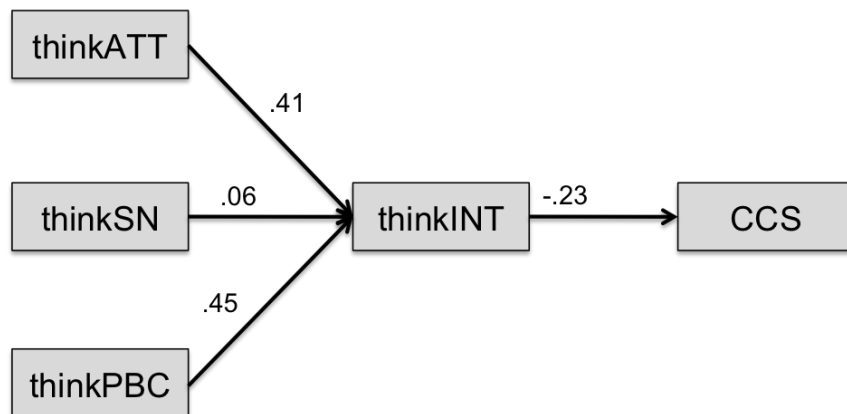


Figure 2. Path Analysis Time 1

Intervention Outcomes

To determine the efficacy of the intervention, doubly-multivariate ANOVAS were conducted to assess pre-post intervention changes on three categories of variables: model

variables, TPB variables, and clinical outcome variables. A doubly-MANOVA design refers to an analysis where several different dependant variables are measured at more than one time point (p. 311, Tabachnick & Fidell, 2007). The dependant variables were grouped based on whether or not they were theoretically related (i.e., all eating pathology variables were grouped in one analysis). These analyses were followed up by simple effects analyses of group and time effects when multivariate interaction effects were significant.

This sample included n=52 participants in the control group and n=70 in the intervention group, after excluding participants who had time 2 missing data (n=10 from control group). Groups were equivalent across all relevant demographics (see Table 3). There were no univariate or multivariate outliers at $\alpha=.01$ and assumptions of the central limits theorem were met. Results of the assumptions of doubly-multivariate analysis of variance were satisfied. Given that this study is exploratory in nature and type 2 error is a concern, no Bonferroni correction was applied to this analysis. Partial eta squares are reported as an indicator of effect size, but should be interpreted with caution, given that participants were recruited from a sample of convenience and participant self-selected into the study. Cell means and standard deviations for the dependent variables over group and time are in Table 5 and selected analyses are depicted in charts (Figures 3 through 12; bars represent confidence intervals).

Table 3

Descriptive Statistics

	Gender		Age	Ethnicity		EDx	Dx
	Male	Female		White	Other	Yes	Yes
Control							
Mean	--	--	19.71	--	--	--	--
%	10.7	89.3	--	68.5	31.5	14.3	5.4
Experimental							
Mean	--	--	19.65	--	--		
%	10.1	89.1	--	70.6	29.4	10.1	12.4

Note: EDx: diagnosis of eating disorder; Dep: diagnosis of depression

Model Variables (CCS, DMQ and PEQ).

Multivariate analysis 1. In order to determine the efficacy of the intervention on the main variables of interest in this study, a doubly-multivariate analysis of variance (MANOVA) was performed on 1 between-subjects independent variable (IV; experimental condition - intervention or control; group), 1 within-subjects IV (time – pre or post), and 4 dependent variables (DV): distorted thinking about food (CCS), fair thinking about food (CCSfair), dietary misinformation (DMQ), and unhealthy eating behaviours (PEQ). GLM Repeated Measures in SPSS version 21.0. was used. The results for all multivariate analyses appear in Table 4.

At the multivariate level, there was a main effect of group [$F(4,117) = 6.28, p=.001$, partial $\eta^2 = .17$], time [$F(4,117)=17.65, p=.000$, partial $\eta^2 =.32$], and the group x time interaction was significant [$F(4,117)=8.23, p=.000$, partial $\eta^2 =.19$]. At the univariate level, for the CCS there was a main effect of group [$F(1,120)=7.97, p=.001$, partial $\eta^2 =.06$], time [$F(1,120)=9.23, p=.001$, partial $\eta^2 = .07$], and a significant interaction [$F(1,120)=8.13, p=.001$, partial $\eta^2 =.063$]. There was also main effect of group [$F(1,120)=14.29, p=.001$, partial $\eta^2 =.106$], time [$F(1,120)=49.29, p=.001$, partial $\eta^2 =.29$], and interaction [$F(1,120)=22.28, p=.001$, partial $\eta^2 =.16$] for the DMQ. As for the CCSfair, only the group effect was significant [$F(1,120)=8.83, p=.004$, partial $\eta^2 =.07$] No univariate effects were significant for the PEQ.

For a more fine-grained analysis, significant interactions were followed up by testing simple main effects, doing all pairwise comparisons among the four cells. The results appear in Table 4 (Analysis 1), where means that were significantly different are noted in the far right column ($p \leq 0.01$). This analysis revealed a simple main effect of the interaction between time and group for the CCS [$F(4,117) = 9.44, p=.001$, partial $\eta^2 = .068$] and the DMQ [$F(4,117) = 22.28, p=.000$, partial $\eta^2 =.157$].

Thus, for participants in the experimental group, attending the workshop lead to a significant reduction in distorted thinking about food and dietary misinformation, whereas for participants in the control group, attending the sham treatment had no effect. Therefore, hypotheses 5 and 6a were supported.

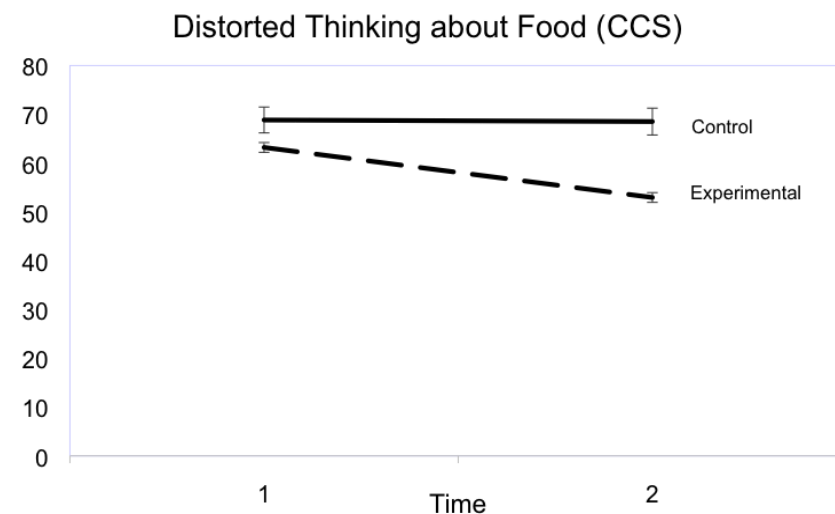


Figure 3. Change in CCS score over time

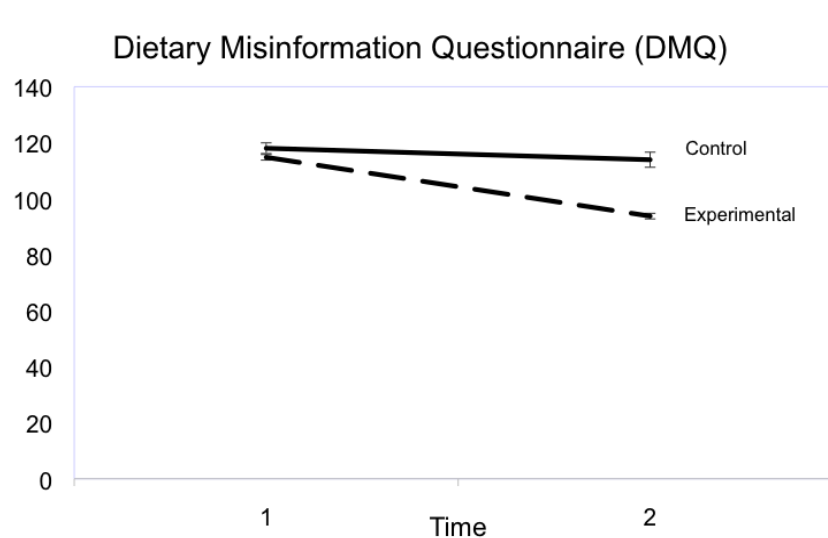


Figure 4. Change in DMQ score over time

TPB Outcomes.

Multivariate analysis 2. In order to determine whether the intervention had an impact on

either of the TPB variables (i.e., intent to engage in fair thinking about food and track food behaviours), a doubly MANOVA was performed on experimental condition (group), time, and 2 DVs: total scores for TPB scale for “changing thinking about food” (TPBthink) and the TPB scale for tracking thoughts about food (TPBtrack).

At the multivariate level, only the main effect of group was significant [$F(2,119) = 3.99$, $p = .021$, partial $\eta^2 = .058$]. The interaction was not significant. At the univariate level, the main effect of group was significant for TPBthink [$F(1,120) = 8.03$, $p = .005$] partial $\eta^2 = .058$ and TPBtrack [$F(1,120) = 3.77$, $p = .05$, partial $\eta^2 = .028$].

Thus, the main effect of time on TPB scales was not significant, but the main effect of group was significant such that the participants who attended the workshop had higher overall TPB scores to increase fair thinking about food and track thoughts about food, irrespective of time point.

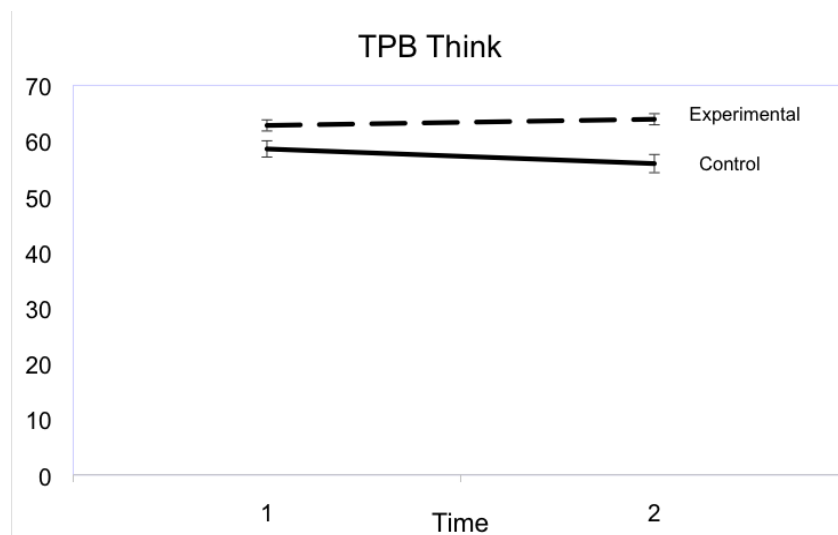


Figure 5. Change in TPB “think” score over time

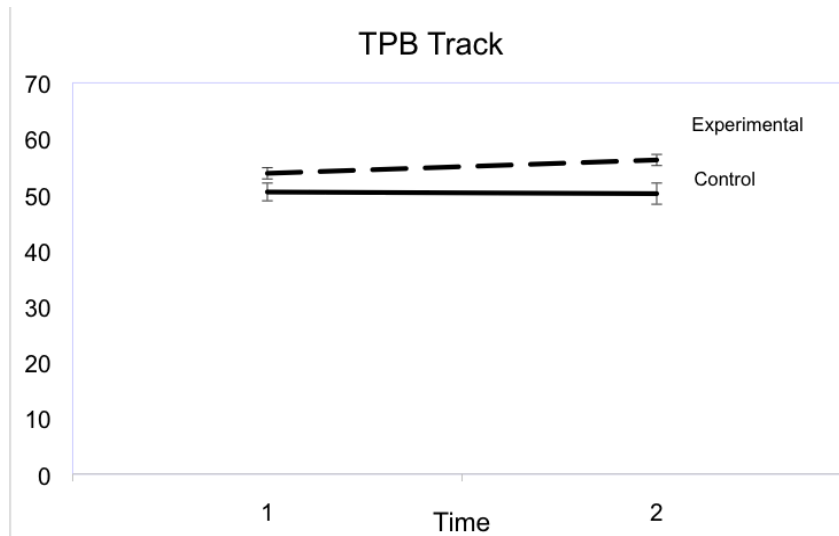


Figure 6. Change in TPB “track” score over time

Multivariate analysis 2b: TPB Think Subscale Outcomes. In order to determine which components of the TPB Think model changed as a result of the intervention, a doubly multivariate analysis of variance was performed on experimental condition (group), time, and 5 DVs: the subscales of the TPB scale for “engaging in fair thinking about food” (TPBthink): attitudes (ATT), subjective norms (SN), perceived behavioural control (PBC), intentions (INTENT) and past behaviour (PAST).

At the multivariate level, both main effects of group [$F(5,116) = 2.91, p = .016$, partial $\eta^2 = .104$] and time [$F(5,116) = 2.45, p = .037$, partial $\eta^2 = .089$] as well as the group x time interaction [$F(5,116) = 3.01, p = .013$, partial $\eta^2 = .107$] were significant. At the univariate level, the main effect of time was only significant for PAST [$F(1,120) = 9.44, p = .003$, partial $\eta^2 = .068$] and the main effect of group was significant for ATT [$F(1,120) = 5.45, p = .021$, partial $\eta^2 = .040$], PBC [$F(1,120) = 12.21, p = .001$, partial $\eta^2 = .086$], INTENT [$F(1,120) = 9.37, p = .003$, partial $\eta^2 = .067$] and PAST [$F(1,120) = 4.32, p = .040$, partial $\eta^2 = .032$]. At this level, the time x group interaction was only significant for INTENT [$F(1,120) = 7.32, p = .008$, partial $\eta^2 = .053$]. The significant interactions were followed up by testing simple main effects. This analysis revealed a simple

main effect of the interaction between time and group for the TPB think intentions (INTENT) subscale [$F(1,20) = 7.32, p = .008, \text{partial } \eta^2 = .053$].

Thus, participants who attended the sham treatment experienced a decrease in intentions to engage in fair thinking about food, while workshop participants experienced a statistically non-significant increase in these intentions over time 1 and time 2. Thus, hypothesis 4 was not supported. Furthermore, participants in the experimental group endorsed higher scores for ATT, PBC, INTENT, and PAST than control participants, irrespective of time point.

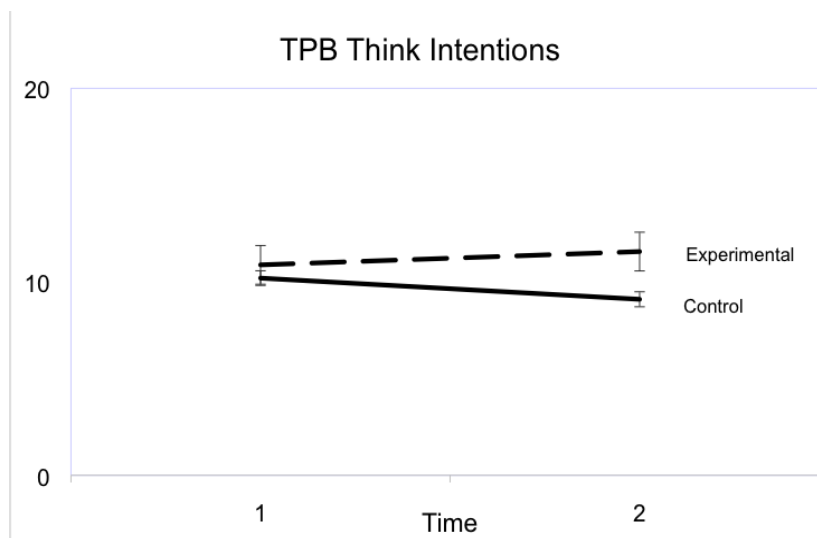


Figure 7. Change in intentions to “change thinking” over time

Multivariate analysis 2c: TPB Track Subscale Outcomes. A doubly multivariate analysis of variance was performed on experimental condition (group), time, and 5 DVs: the subscales of the TPB scale for “tracking thinking about food” (TPBtrack): attitudes (ATT), subjective norms (SN), perceived behavioural control (PBC), intent (INT) and past behaviour (PAST).

At the multivariate level, only the main effect of time was significant [$F(5,116) = 5.09, p = .000, \text{partial } \eta^2 = .168$] and there was no significant interaction. At the univariate level, the main effect of time was only significant for SN [$F(1,120) = 7.73, p = .006, \text{partial } \eta^2 = .056$] and PAST [F

(1,120)=11.33, $p=.001$, partial $\eta^2=.080$].

Thus, the main effect of group on these TPB antecedents was not significant, but the main effect of time was significant for subjective norms and past behaviour such that, over time, participants experienced an increase in the socially perceived value of tracking thoughts about food irrespective of group.

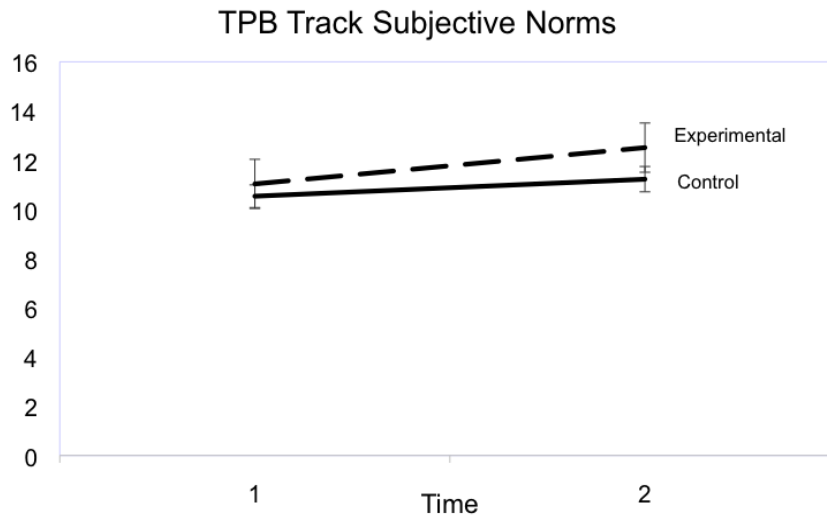


Figure 8. Change in subjective norms of “tracking” over time

Incidental Clinical Outcomes.

Analysis 3: Body Image Outcomes. Given that there was only one measure of body image used, a repeated measures ANOVA was performed on experimental condition (group), time, and 1 dependent variable: Body Shape Questionnaire (BSQ). Results showed that the interaction was significant at the univariate level [$F(1,120) = 3.95$, $p=.05$, partial $\eta^2=.029$]. Thus, for participants in the experimental group, attending the workshop lead to a reduction in body dissatisfaction, whereas for participants in the control group, attending the sham treatment had no effect on body image. Therefore, hypothesis 6c was supported.

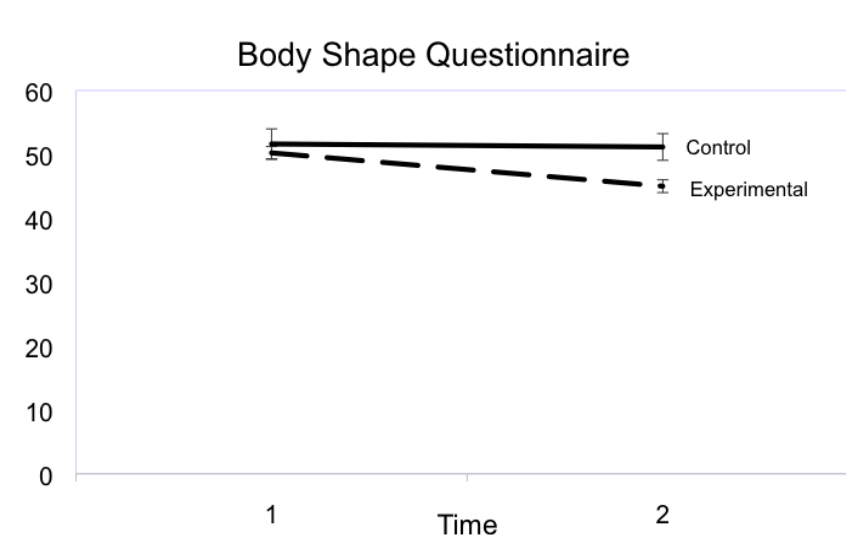


Figure 9. Change in BSQ score over time

Multivariate analysis 4: Eating Pathology Outcomes. To measure the impact of the intervention on eating pathology symptoms, a doubly MANOVA was performed on experimental condition (group), time, and 2 dependent variables: total scores for the Three Factor Eating Questionnaire (TFEQ-18) and Eating Attitudes Test (EAT).

At the multivariate level, both the main effect of time [$F(2,119)=4.63$, $p=.01$, partial $\eta^2=.072$] and the group x time interaction [$F(2,119)=3.37$, $p=.03$, partial $\eta^2=.057$] were significant. At the univariate level, the main effect of time was significant for the TFEQ [$F(1,120)=4.99$, $p=.03$, partial $\eta^2=.040$] but not for the EAT. Conversely, at this level, the group x time interaction was significant for the EAT [$F(1,120)=6.89$, $p=.01$, partial $\eta^2=.054$] but not for the TFEQ. The significant interaction was followed up by testing simple main effects. This analysis revealed a simple main effect of the interaction between time and group for the EAT [$F(1,120)=6.89$, $p=.010$, partial $\eta^2=.054$].

Thus, for participants in the experimental group, attending the workshop lead to a reduction in eating pathology symptoms (as measured by the EAT), whereas for participants in the control group, attending the sham treatment actually lead to an increase in eating pathology.

Therefore, hypothesis 6b was supported.

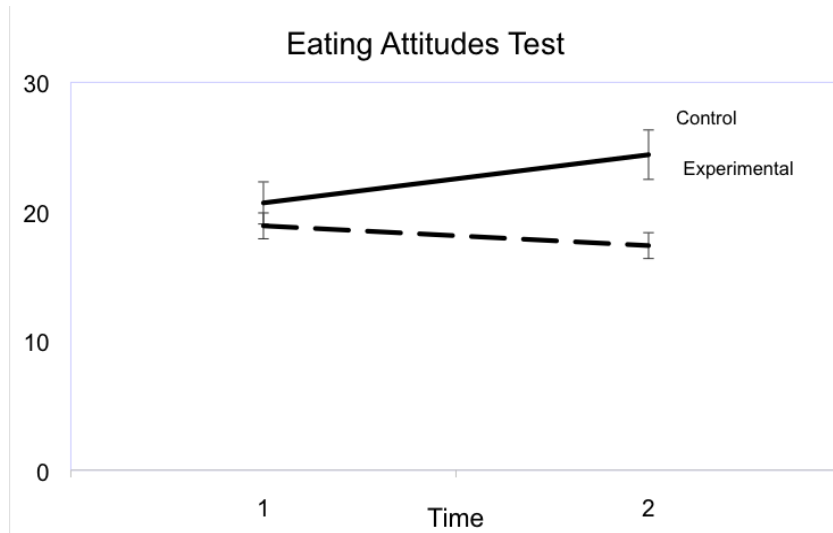


Figure 10. Change in EAT score over time

Multivariate analysis 4b: Eating Pathology Subscale Outcomes. A doubly MANOVA was performed on experimental condition (group), time, and 6 dependent variables: the three subscales of the TFEQ: cognitive restraint scale, unrestrained eating scale and emotional eating scale, as well as the three subscales from the EAT: dieting, bulimia and control subscales.

At the multivariate level, both the main effect of time [$F(6,115) = 2.906, p = .01$, partial $\eta^2 = .132$] and the time x group interaction [$F(6,115) = 2.13, p = .05$, partial $\eta^2 = .10$] were significant. At the univariate level, the main effect of time was significant for the TFEQ unrestrained eating subscale [$F(6,115) = 5.50, p = .02$, partial $\eta^2 = .044$] and the bulimia subscale of the EAT [$F(6,115) = 4.25, p = .04$, partial $\eta^2 = .034$]. The interaction was only significant for the dieting subscale of the EAT [$F(6,115) = 0.75, p = .002$, partial $\eta^2 = .075$]. The significant interactions were followed up by testing simple main effects. This analysis revealed a simple main effect of the interaction between time and group for the dieting subscale of the EAT [$F(6,115) = 9.75, p = .002$, partial $\eta^2 = .075$].

Thus, for participants in the experimental group, attending the workshop lead to a

reduction in eating pathology symptoms (as measured by the dieting subscale of the EAT), whereas for participants in the control group, attending the sham treatment actually lead to an increase in this score.

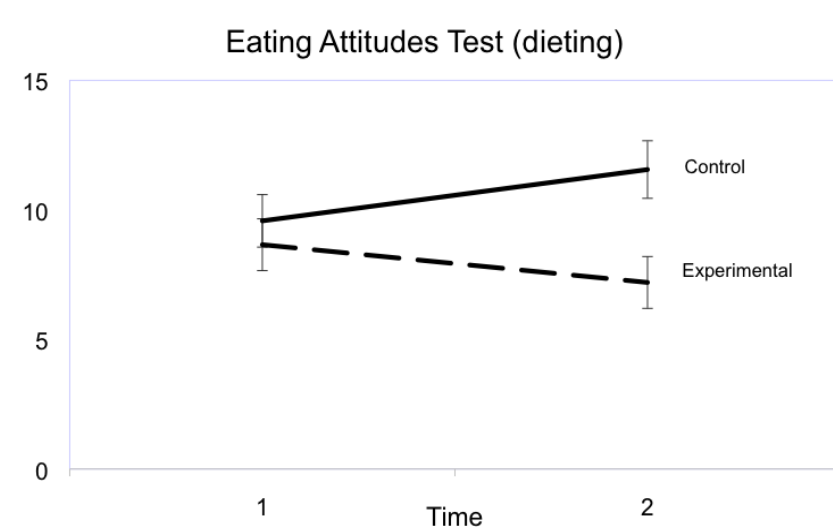


Figure 11. Change in "dieting subscale" of the EAT over time

Multivariate analysis 5: Mood Outcomes. In order to determine the impact of the workshop on participants' mood, a doubly MANOVA was performed on experimental condition (group), time, and 3 dependent variables, the subscales of the SPANE: positive affect, negative affect and affect balance.

At the multivariate level, only the main effect of group was significant [$F(3,118) = 3.99$, $p = .021$, partial $\eta^2 = .058$]. There was no significant interaction. At the univariate level, the main effect of group was significant for the positive affect [$F(1,120) = 7.95$, $p = .006$, partial $\eta^2 = .058$] and affect balance subscales [$F(1,120) = 4.95$, $p = .028$, partial $\eta^2 = .037$].

Thus, the main effect of time on affect was not significant, but the main effect of group on affect was significant such as that participants who attended the workshop had more positive mood, irrespective of time point. Thus, hypothesis 6d is not supported.



Figure 12. Change in positive mood over time

Table 4

F values for doubly multivariate GLM analyses where experimental condition, time, and the interaction between them are used to predict study outcomes

Variable	Group	Time	Interaction
Model Outcomes			
Analysis 1: Model variables			
Multivariate effect	6.28**	17.65**	8.82**
Univariate effects			
CCS	7.97**	9.23**	8.13**
CCSfair	8.83**	3.24	2.56
DMQ	14.29**	49.29**	22.28**
PEQ	.006	.22	2.36
Theory of Planned Behaviour Outcomes			
Analysis 2: TPB total Variables			
Multivariate effect	3.99*	.98	.30
Univariate effects			
TPBtotTRACK	3.77*	.56	.94
TPBtotTHINK	8.03**	.39	2.4
Analysis 2b: TPBthink subscales Variables			
Multivariate effect	2.91**	2.45*	3.01**
Univariate effects			
ATT	5.45*	.78	.02
SN	.18	.00	1.79
PBC	12.22**	.09	1.03
INT	9.37**	.38	7.32**
PAST	4.32*	9.44**	.27
Analysis 2c: TPBtrack subscales Variables			

<i>Multivariate effect</i>	1.55	5.09**	1.16
<i>Univariate effects</i>			
ATT	1.55	.52	.39
SN	1.75	7.73**	1.01
PBC	7.17**	.62	.04
INT	2.99	.09	3.42
PAST	.08	11.33**	1.79
Incidental Clinical Outcomes			
Analysis 3: Body Image variable			
<i>Univariate effects</i>			
BSQ	1.28	5.7 *	3.95*
Analysis 4: Eating Pathology variables			
<i>Multivariate effect</i>	1.66	4.63**	3.57*
<i>Univariate effects</i>			
TFEQ	.23	4.99*	2.25
EAT	3.03	1.21	6.89**
Analysis 4b: Eating Pathology subscales			
<i>Multivariate effect</i>	1.92	2.91**	2.13*
<i>Univariate effects</i>			
TFEQ-CRS	.01	1.71	2.23
TFEQ-UES	.02	5.50*	2.51
TFEQ-EMO	3.05	.26	.19
EAT-diet	2.96	.23	9.75**
EAT-bul	1.59	4.25*	2.82
EAT-cont	.86	1.70	2.50
Analysis 5: Mood variables			
<i>Multivariate effect</i>			
<i>Univariate effects</i>	3.99*	.56	1.80
SPANepos			
SPANeneg	7.95**	.16	2.77
SPANefbal	1.19	.88	1.20
	4.95*	.13	3.46

*significant at $p \leq 0.05$

**significant at $p \leq 0.01$

Table 5

Means (standard deviations) for all outcome variables at Time 1 and 2

	INT		CON		Sig. Mean Differences	
Variable	T1 (1)	T2 (2)	T1 (3)	T2 (4)	1-2	3-4
	M(SD)	M(SD)	M(SD)	M(SD)		
Model Variables						
Analysis 1:						
CCS	62.95(21.97)	52.46(20.43)	68.84(21.81)	68.52(25.29)	10.49*	.327
DMQ	114.79(16.34)	93.77(23.92)	118.03(15.34)	113.92(20.53)	21.00*	4.12
PEQ	19.96(12.10)	19.21(2.88)	18.77(5.24)	20.17(9.41)	.74	.14
CCSfair	39.82(10.12)	42.93(10.52)	36.58(8.81)	36.75(10.19)	3.10*	.17

TPB Variables						
Analysis 2:						
TPBtrack	53.81(15.45)	56.20(17.40)	50.48(11.92)	50.17(16.20)	2.39	.308
TPBthink	62.80(13.52)	63.91(13.58)	58.58(12.33)	55.94(16.10)	1.11	2.64
Analysis 2b:						
TPBthink						
ATT	23.50(5.52)	23.13(5.54)	21.58(4.89)	21.20(5.78)	.36	.481
SN	10.98(3.76)	11.49(3.70)	11.29(2.73)	10.75(3.91)	.51	.54
PBC	17.45(4.19)	17.73(4.23)	15.52(4.00)	15.00(5.00)	.28	.52
INT	10.88(3.46)	11.56(3.18)	10.19(3.28)	9.10(3.87)	.69	1.1*
PAST	7.04(3.75)	8.10(3.85)	5.79(3.03)	7.23(3.62)	1.05*	1.48*
Analysis 2c:						
TPBtrack						
ATT	18.13(6.33)	18.89(6.88)	17.30(4.60)	17.36(6.08)	.75	.06
SN	11.04(4.53)	12.51(4.73)	10.54(3.37)	11.23(4.40)	1.48**	.69
PBC	15.73(4.00)	15.33(4.25)	14.11(3.38)	13.87(4.16)	.41	.25
INT	8.90(4.16)	9.48(4.38)	8.52(3.61)	7.71(3.81)	.58	.81
PAST	4.11(2.91)	6.00(4.34)	4.50(3.44)	5.31(3.83)	1.88**	.81
Clinical Variables						
Analysis 3:						
BSQ	50.23(20.98)	45.01(17.98)	51.63(20.39)	51.15(19.73)	5.21*	.481
Analysis 4:						
TFEQ	43.14(8.61)	40.99(9.17)	42.96(7.22)	42.53(8.55)	2.16*	.42
EAT	18.90(13.39)	17.37(14.17)	20.67(13.95)	24.40(18.17)	1.53	3.73*
Analysis 4b:						
TFEQ-CRS	15.00(3.40)	14.34(3.75)	14.71(3.80)	14.75(3.94)	.66*	.04
TFEQ-UES	21.32(5.46)	19.84(5.59)	20.61(4.52)	20.32(5.50)	1.49*	.29
TFEQ-EMO	6.81(2.78)	6.80(2.53)	7.63(2.42)	7.46(2.24)	.01	.17
EAT-diet	8.66(8.07)	7.20(8.11)	9.58(9.00)	11.56(10.70)	1.46*	1.98*
EAT-bul	1.64(2.68)	1.64(2.94)	1.71(2.60)	2.70(2.76)	.10	.98*
EAT-cont	2.34(3.03)	2.27(3.16)	2.44(3.00)	3.19(4.10)	.07	.75
Analysis 5:						
SPANepos	22.89(4.34)	23.35(4.55)	21.56(3.85)	20.81(4.60)	.46	.75
SPANeneg	16.20(4.46)	15.46(5.15)	16.62(4.30)	16.67(4.49)	.74	.06
SPANeafbal	6.69(7.46)	7.89(8.61)	4.94(6.88)	4.13(6.61)	1.2	.81

Note. Within a given row, means that are significantly different from one another at $p < .05$ (*) and $p < .01$ (**) are noted in the farthest column. Simple effects analyses were conducted for significant univariate interactions.

Discussion

The main purpose of this study was to offer an alternative to current models of dieting and disordered eating prevention by addressing the weaknesses of existing programs.

Specifically, I sought to develop and evaluate a TPB and CBT-based intervention in order to reduce dietary misinformation and distorted thinking about food and to evaluate its impact on clinical variables such as body image, eating pathology symptoms, and mood. Results suggest

that this intervention has the potential to enhance existing models in a number of important ways, ranging from prevention theory to clinical practice.

Prevention Theory

One of the principal weaknesses of existing prevention and interventions programs for eating problems is that they are often not based on substantiated models of change (Nation, et al., 2003; Dusenbury & Falco, 1997). The result is that it is difficult to determine which components of the programs are successful or how to account for poor outcomes. Programs also often rely on methods to elicit change that have not been supported in the literature, such as knowledge dissemination, teaching about healthy eating and exercise, and discussions on body image (Molaison, 2002; McVey, David, Tweed, & Shaw, 2004). Indeed, these elements have rarely led to successful long-term change. The current study addressed these limitations by designing an intervention according to two well-established models of behaviour change (CBT and TPB) and by targeting two important variables that have been largely ignored in past research (distorted thinking about food and dietary misinformation).

Cognitive Behaviour Therapy (CBT). CBT theory was used in this study to understand how thoughts, beliefs and behaviours are connected in the current theoretical model (see Figure 1). Despite the success of CBT-based interventions for clinical eating disorders (i.e., CBT-e; Fairburn, 2009), few dieting and disordered eating prevention programs are designed according to CBT principles or implement CBT strategies. This is a missed opportunity, given that research has shown that CBT can be effective when disseminated to large groups of subclinical populations (e.g., low-intensity CBT; Richards, 2010). Thus, the current study designed the Calorie Catastrophizing Workshop according to CBT theory and implemented a number of key CBT strategies. Essentially, participants were socialized to the CBT model and they learned

about cognitive distortions, how they apply to food, and how these thoughts can affect eating behaviour. Then, participants were asked to monitor and challenge their own food-related cognitive distortions for two weeks. Another component of the homework was to monitor and challenge any diet myths that they encountered during this two-week period.

CBT Outcomes. Distorted thinking about food and dietary misinformation were the main targets of the CBT strategies. Participants in the intervention group experienced a significant reduction in distorted thinking about food and dietary misinformation, but the increase in fair thinking about food was nonsignificant. Despite the fact that clinical variables were not targeted for intervention, these participants also experienced a reduction in body image dissatisfaction and symptoms of eating pathology. Control participants experienced no change in distorted thinking or dietary misinformation, but did exhibit an increase in symptoms of eating pathology after watching a brief video of the Canada Food Guide (CBC, 2012).

The eating pathology scale most sensitive to change over both groups was the “dieting” subscale of the EAT (Garner & Garfinkel, 1979). This subscale is comprised of items that assess problematic dieting behaviours, such as preoccupation with counting calories, avoiding certain foods and feeling guilty about food and exercise. Thus, despite the fact that the intervention did not lead to a statistically significant change in behaviour measured by the PEQ, it did result in significant reduction in dieting behaviours as measured by the EAT. Importantly, learning about the Canada Food Guide seemed to lead to increases in this behaviour. It is unclear why this change occurred, but it could be the case that receiving any information about how one “should” eat activates underlying distorted cognitions about food, but further research is necessary to fully understand this relationship. Overall, these results point to the effectiveness of the intervention and highlight the utility of using CBT in the design of prevention programs for eating problems.

These findings were also likely strengthened by the integration of TPB into the design of the intervention.

Theory of Planned Behaviour (TPB). The TPB was used in this study to explain how societal factors (i.e., beliefs) influence intentional antecedents (i.e., attitudes, subjective norms, and perceived behavioural control [PBC]), which determine actual behaviour. The TPB has been applied with success across a wide range of clinical populations (Armitage & Conner, 2001). With respect to eating, the TPB has mostly been applied in order to understand dieting behaviour (Nejad et al., 2004) and to design interventions geared at changing eating behaviour, such as increasing fruit and vegetable intake (Godin & Kok, 1996; Kelly et al., 2011). It has never been applied within the context of disordered eating prevention or within a theoretical model including distorted thinking about food and dietary misinformation. Thus, the Calorie Catastrophizing Workshop is the first intervention of its kind to be designed according to the principles of TPB. Specifically, participants were asked to share their beliefs associated with the target behaviours and then attitudes, subjective norms and PBC were targeted for change through workshop activities. Importantly, for the current study there were two behavioural targets in TPB: decreasing distorted thinking about food and tracking food cognitions. This is due to the fact that TPB interventions typically target overt, observable behaviours and generally do not target cognitive processes (Armitage & Conner, 2001). By targeting thought monitoring, this intervention also bridges TPB with CBT.

Psychometrics and Model Testing. Psychometric testing revealed that the TPBQ was reliable and valid. This questionnaire is able to measure changes in attitudes, subjective norms, perceived behavioural control (PBC) and intentions to engage in fair thinking about food and thought monitoring. Theoretical model testing at time 1 indicated that attitudes and PBC

accounted for most of the variance in intentions to engage in fair thinking about food. This is consistent with previous research on eating behaviours that has shown that attitudes and PBC tend to be the strongest predictors of intentions (Nejad et al., 2004; Armitage & Conner, 2001).

TPB Outcomes. Results reveal that, overall, there was little change in TPB antecedents or intentions to engage in fair thinking about food or to track food-related thoughts; however, the experimental group did experience significant increases in subjective norms and past behaviour related to tracking thoughts about food. This may be due to the fact that tracking food thoughts is a new behaviour to them, so learning about it among peers may have lead to an increase in believing that it is socially prescribed. Not surprisingly, the increase in past behaviour is due to the fact that workshop participants were asked to track their behaviour for two weeks following the intervention.

Despite the fact that this intervention led to little change across TPB variables, this should not be taken as evidence of the ineffectiveness of the TPB model (see General Discussion for more detail). In fact, participants across groups presented with high levels of antecedents and intentions to change and track thinking at time 1. Thus, there was little space for upward movement in these scores. There are many potential explanations for this “ceiling effect”, but given the fact that university students are exposed to pressure to be constantly improving their eating and lifestyle habits (Stice, Maxfield, & Wells, 2003), it could be that they already have heightened antecedents and intentions to change eating-related behaviour. In fact, the success of the intervention may be accounted for by teaching participants strategies that allow them to act on their existing intentions. At the very least, the intervention maintained levels of antecedents and intentions, whereas control participants actually experienced a decrease in certain variables. Additionally, by targeting two relatively unexplored variables, the current intervention offers a

novel, potentially more accurate way of conceptualizing and treating subclinical eating problems.

Distorted Thinking about Food. Research has repeatedly shown that nonclinical individuals engage in distorted thinking about food and that this predicts dieting and disordered eating behaviour (Polivy & Herman, 2002). Yet no dieting or disordered eating prevention programs to date have explicitly targeted this variable for change. Thus, by specifically targeting this type of thinking, the current intervention ostensibly improves upon existing intervention models. This intervention essentially armed participants with tools that they needed to challenge distorted thinking and dietary misinformation. Indeed, targeting this cognitive variable could be the key to successful, lasting change in prevention and intervention programs for eating problems across the spectrum of severity.

Results revealed that this intervention was successful at producing meaningful change in distorted thinking about food. This is consistent with research that has shown that CBT-based interventions for eating disorders are successful at changing related distorted cognitions (Fairburn, 2009) and it builds on this research by showing that this type of intervention is useful for subclinical populations. Furthermore, meaningful change was observed in other clinical variables such as eating pathology symptoms (as measured by the EAT) and body dissatisfaction, which were not directly targeted during intervention. This enhances current models by suggesting that distorted thinking about food could be a pernicious root cause of these pathological symptoms, which can be altered without directly targeting them. Importantly, mood was not sensitive to the intervention and this finding suggests that mood (as measured by the SPANE) might not be related to the target variables of this study, or that perhaps there is a more appropriate scale available to capture this construct.

Finally, these findings suggest that teaching basic CBT skills related to distorted thinking

about food may have some benefits that spill over into other areas of distorted thinking, such as poor body image. The current intervention informs prevention theory by addressing the environmental or societal context of participants and considering the impact of dieting culture and negative messaging about food. This serves to modernized the intervention, by acknowledging societal factors that compete with prevention or intervention strategies.

Dietary Misinformation. One of the most tangible consequences of diet culture is misinformation (i.e., endorsing diet myths). Misinformation is a consequence of increased reliance on online sources for diet and health information. In youth it is believed to be a consequence of increased use of social media, and specifically an increase in attention to diet- and fitness-based accounts. Essentially, Instagram, YouTube, facebook and other social media sites provide platforms for people to share and widely disseminate inaccurate information about diet and exercise to young people, and these types of accounts are very popular (Chou, Hunt, Beckjord, Moser, & Hesse, 2009; Syed-Abdul et al., 2013). By not addressing misinformation, existing interventions do not provide participants with any skills to buffer against the negative impact of this messaging, which competes with any other skills gained during intervention. Although some studies have focused on teaching media literacy skills (McVey, et al., 2004), these have mostly focused on body image and how participants can avoid internalizing the “thin ideal”. Thus, by explicitly targeting misinformation, the current intervention actually has more real-world utility.

Results revealed that this intervention was successful at producing a significant reduction in misinformation, which was associated with a reduction in distorted thinking about food and other clinical variables. This is a novel finding, given that no research to date has demonstrated that changing misinformation can improve intervention outcomes. Although misinformation has

generally been ignored in prevention research to date, these findings are consistent with research that has shown that societal factors and diet messaging are associated with eating pathology across clinical populations (Syed-Abdul et al., 2013; Ferguson, Muñoz, Garza, & Galindo, 2014). Altogether, the findings from this intervention study have a number of important theoretical and clinical implications.

Implications

This is the first study of its kind to combine Cognitive Behaviour Therapy and the Theory of Planned Behaviour in the design of a subclinical eating pathology prevention program. It is also the first study to target distorted thinking about food and dietary misinformation in this population. These findings, if replicated, have a number of important implications for the future of intervention design, eating pathology prevention, and the treatment of subclinical eating problems.

Program Design. As stated above, the current intervention has the potential to improve upon existing programs in a number of important ways. First, this intervention was designed according to two well-established models of behaviour change. This clarified which variables should be targeted for change, and what strategies could be implemented to bring about this change. The target audience was also a non-specific, subclinical population. Despite the fact that the sample is limited in generalizability (see limitations below), results lend support to the idea that the intervention is widely applicable, as it led to significant changes across clinical domains. Furthermore, although some information was exchanged during the intervention, psychoeducation was not a focus of the workshop. In fact, the workshop focused mostly on interactive activities and CBT strategies, which were effective. Overall, the promising results of the current intervention can be extended to inform the design of future prevention and

intervention programs.

Indeed, researchers have suggested that effective prevention theory requires a developmental theory that links risk factors, mediating processes, and maladaptive patterns of behavior (Coie et al., 1993). The findings of the current research have provided preliminary evidence for a new adjunct developmental theory of eating pathology in which dietary misinformation creates the risk of distorted thinking about food which then leads to problematic eating behaviour (as supported in study 1). It could be the case that exposure to diet myths and misinformation (e.g., “Carbs are bad”) leads to the development of distorted thinking (e.g., “If I eat carbs I am a failure”) and unhealthy eating (e.g., restriction of carbohydrate intake). This points to a new way of structuring eating-related intervention and prevention programs. Importantly, it is unclear from the current research how or why people develop disordered eating in relation to dietary misinformation and distorted thinking about food. Indeed, there likely exists a multitude of vulnerability and protective factors that could contribute to or protect from the development and maintenance of these problems. Future research would benefit from further exploring this relationship, given that it may have important implications for eating pathology prevention.

Eating Pathology Prevention. Beyond theoretical design implications, the current results point to some practical implications for eating pathology prevention. First, results showed that it is possible for participants to experience change following one 60-minute (ultra brief) psychological intervention session and some light homework. This vastly improves on existing programs, which sometimes require that participants attend multiple sessions across many weeks (Stice, Shaw, & Marti, 2007). This study also used a low-intensity CBT model, coupled with a non-specific vulnerability model. Essentially, a large group of people who had yet to be

designated as “at-risk” were selected for this program and demonstrated significant gains following intervention. This is consistent with research that has shown that it is beneficial to target large groups of nonclinical individuals in order to arm them with skills to prevent future pathology (McVey et al., 2010) and it enhances prevention theory by highlighting the importance of a non-specific audience. Furthermore, although it was not addressed in this study, it is possible that paraprofessionals could be trained to deliver this workshop in community or school settings, which would greatly reduce the cost and increase the reach of prevention. Finally, although not explicitly addressed in this study, these findings lend further support to the idea that social media has a pernicious impact on eating behaviours in youth by increasing misinformation. Future eating pathology prevention in youth may benefit from a discussion of the critical use of social media, and future research would benefit from exploring these ideas.

Treatment of Subclinical Eating Problems. This study has demonstrated the potential utility of targeting distorted thinking about food and dietary misinformation in order to improve intervention outcomes. Targeting these variables lead to a notable change in other important clinical variables (body dissatisfaction and eating pathology). The clinical implications of these findings extend beyond the study of prevention and intervention programs to the individual treatment of subclinical eating problems in private practice and other settings. Essentially, this study has identified new targets for intervention that can be incorporated into talk therapies from counseling to CBT in order to treat subclinical eating problems. In fact, individual therapists could benefit from broadening their view of the onset and maintenance of these problems to formally address the role of distorted thinking about food and dietary misinformation. This would allow therapists to assess the individual impact of these constructs and determine how to tailor therapy for their clients. For example, a therapist might determine that dietary

misinformation is a severe concern for their client, and that it is directly related to social media use (e.g., following fitness- and diet-related accounts) and associated with low mood and body dissatisfaction. Thus, treatment would focus on limiting social media use or choosing to follow more body-positive or accurate accounts in order to reduce dietary misinformation and other clinical variables.

Limitations and Future Directions

These findings must be considered in the light of a few important limitations. First, a new intervention was designed according to two theories: CBT and TPB. Despite the fact that this intervention had a significant impact on variables of interest, replication of these findings will be needed to provide greater support for the efficacy and utility of this workshop and the theoretical model.

Second, as with previous research on this topic (see study 1), this RCT was conducted on a non-clinical, university student population that was essentially a sample of convenience. Although these might limit the generalizability of results, research has shown that university is an ideal setting to teach older adolescents and young adults media literacy skills and how to challenge distorted cognitions (Rickwood, Deane, Wilson, & Ciarrochi, 2005). Furthermore, time 1 sample characteristics indicate that this sample experienced higher-than-average levels of body dissatisfaction, eating pathology and low mood. Therefore, despite the fact that the generalizability is limited, this might indicate that university students are the ideal population for this intervention. In order to expand findings, future studies on this topic could aim to recruit younger school-aged samples or older adult samples in order to understand these processes among different age categories and whether or not societal factors (i.e., misinformation) have a differential impact based on age. This could have important implications towards how messages

about food and healthy active living are targeted towards these populations.

Third, this sample was predominantly white and female. Once again this limits the ability to generalize results. Despite the fact that research has shown that women are more likely to diet and participate in the dieting culture by purchasing books, magazines and diet products, these issues still impact males (Calderon, Catherine, & Jambazian, 2004). Research has also shown that eating and body image concerns can vary culturally (e.g., Latinas often endorse feeling pressured to be curvy versus thin; Schooler, 2008). Future research should aim to recruit more males and ethnically diverse individuals in order to understand whether these results are consistent across groups.

Fourth, this sample was comprised of small groups of nonclinical participants. Despite this being the focus of the current intervention, it is unclear how these results are relevant to clinical populations, such as eating disorders, overweight, and obesity. Thus, though it is reasonable to suggest that this workshop could be useful in these populations, further investigation is necessary. Future research would benefit from trialing this workshop with these populations. Additionally, future research is necessary to determine whether this workshop is appropriate to deliver to larger groups of participants.

Fifth, participants' treatment expectations were not explicitly measured prior to the intervention. Despite the fact that it is reasonable to assume that both groups had similar expectations given the broad description of the study, followed by the random assignment of participants into groups, it is possible that changes over time could be accounted for by difference in motivation and expectations about treatment (Boot et al., 2013). Thus, future research would benefit from measuring expectations prior to the intervention.

Finally, some methodological limitations must be considered. This study serves as a

preliminary test of the efficacy of the intervention. As such, the design had only two time points and two groups. This limits the understanding of the longer-term impact of the intervention, as well as the individual impact of the sham treatment. Future research on this topic could benefit from adding a “wait list” control group. This would allow more intricate analyses to be conducted to determine whether the sham treatment had a differential effect on participants than nothing at all. Further, future studies would benefit from adding another time point. Having a third time point, perhaps some months in the future, would enhance findings by determining whether these cognitive gains are present later than 2 weeks following the intervention. Additionally, it could also be determined if changes in eating behaviour are concomitant with these cognitive changes over time.

Conclusion

The current study has the potential to improve the way eating problems across the spectrum are prevented and treated. By designing the intervention according to two robust models of behaviour change (i.e., TPB and CBT), this research addresses many of the weaknesses of existing interventions. By explicitly targeting distorted thinking about food and dietary misinformation, this intervention has modernized the way nonclinical eating problems from dieting to overweight are conceptualized. Ultimately, this intervention targets societal-cognitive factors and provides participants with skills to challenge inaccurate messaging about food, which wholly enhances the practical utility of the program and points to how other programs can be similarly improved.

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General Discussion

Purpose of the Research

The purpose of this series of studies was to improve upon existing theoretical and clinical models of subclinical eating pathology in two ways: 1) by formally defining and measuring a construct that has been linked to subclinical eating pathology but remains poorly understood (i.e., distorted thinking about food), and 2) to address the weaknesses of existing prevention and intervention programs by developing an evidence-based workshop that also explicitly targets this construct. A secondary goal of the research was to address societal factors that contribute to distorted thinking about food, namely, dietary misinformation. This served to modernize the intervention by considering societal factors that compete or interfere with intervention strategies. This series of studies has addressed a number of gaps in the extant literature and findings enhance current knowledge in a number of important ways.

Summary of Findings and Relevance to Existing Literature

To achieve the goals listed above, the following studies were conducted: Study 1 was a psychometric validation study in which distorted thinking about food and dietary misinformation were defined and measured. In this study, Cognitive Behaviour Therapy (CBT) and the Theory of Planned Behaviour (TPB) were used to develop a theoretical model in which dietary misinformation contributes to the onset and maintenance of distorted thinking about food, which is then associated with pathological eating. Study 2 was a randomized controlled trial that evaluated whether the “Calorie Catastrophizing Workshop” could reduce distorted thinking about food and other clinical variables. In this study, the workshop was design based on CBT and TPB, and the theoretical model was adapted to conceptualize the relationship between the variables targeted for intervention. The findings of these two studies and the success of the

intervention inform both the theory and clinical practice of measuring and treating eating problems across the spectrum of severity. Perhaps the most significant contribution of the current research to eating pathology theory is the conceptualization and measurement of two new variables.

New Theoretical Constructs. The main goal of study 1 was to define and measure “distorted thinking about food”. In order to understand how societal factors (such as dieting culture and diet myths) affect this type of thinking, dietary misinformation was also defined and measured. Thus, the “Calorie Catastrophizing Scale” and “Dietary Misinformation Questionnaire” were developed.

Psychometric analyses revealed that both measures were valid and reliable and essentially measure what they were designed to measure. Further analyses showed that distorted thinking about food is related to important clinical variables such as low mood, body dissatisfaction, and symptoms of eating pathology. Preliminary model analyses suggest that the impact of dietary misinformation on eating pathology is mediated by distorted thinking about food. This relationship has not been demonstrated in research to date, and suggests that dietary misinformation alone may not be sufficient to lead to unhealthy eating. Indeed, it appears that dietary misinformation could be internalized by a cognitive mechanism, which may then translates into unhealthy eating; however, these findings are preliminary. By identifying an important antecedent to distorted thinking about food, and highlighting the relationship between these two variables and symptoms of eating pathology, the current research has suggested a novel way of conceptualizing eating pathology.

Whereas existing models of clinical and subclinical eating pathology generally acknowledge biopsychosocial factors that contribute to the development of eating problems, no

theories have outlined the specific role that dietary misinformation and distorted thinking about food play. Indeed, no research to date has isolated diet myths as an antecedent to distorted thinking in nonclinical populations. Furthermore, no research to date has attempted to formally define or measure distorted thinking about food in this population, despite evidence of its existence (Polivy & Herman, 1985; 2002). Thus, these findings enhance current knowledge by identifying these two key constructs that have largely been ignored.

Dietary Misinformation. Previous research has shown that there is a relationship between misinformation and increased eating pathology in eating disorder patients (e.g., if a patient believes that purging eliminates all caloric absorption, they are more likely to binge and purge; Mines & Merrill, 1987; White, McKee, & O'Malley, 2007). The current research has shown that this relationship exists among nonclinical individuals as well, further supporting the idea that the profiles of individuals with and without eating disorders can look similar (Polivy & Herman, 1985). This highlights the notion that strategies used to treat clinical problems (such as CBT-e) may in fact be appropriate to use on nonclinical population as a means of prevention. Furthermore, the current research built on past research that has demonstrated that societal factors (such as the media) can have a deleterious impact on eating behaviours.

By identifying and measuring dietary misinformation, the current research has highlighted a new, more pertinent societal factor that may be influencing eating behaviours in youth and young adults. Indeed, youth today are spending more time consuming social media than all other media combined (Lenhart, 2015). Youth and young adults also rely wholly on online sources (including related social media accounts) for diet and fitness information. Unsurprisingly, these sources are often inaccurate and have been shown to be linked with poor body image and eating pathology (Syed-Abdul et. al., 2013). This research has suggested that a

consequence of this consumption is misinformation. Thus, it is essential to acknowledge and consider misinformation when conceptualizing eating problems in the modern age. In light of this new evidence, it is also important to consider other sources of misinformation.

Although misinformation is often a consequence of seeking health information online and following health-related social media accounts, it is possible that people become misinformed about food in other ways. Previous research has demonstrated that thoughts and beliefs about food are transmitted through families, insofar as parents with maladaptive beliefs about food teach these beliefs to their children (Cutting, Fisher, Grimm-Thomas, & Birch, 1999). Research has also shown that children and youth learn diet myths and behaviours from their peers as they approach adolescence (Field, Camargo, Taylor, Berkey, & Colditz, 1999; Field et al., 2008; Stice, 1998). Thus, it could be the case that children develop inaccurate beliefs about food from their parents, which are then reinforced through diet myths and other sources of misinformation, such as social media. This may be particularly problematic for families who share illnesses, such as diabetes or obesity, which are two areas where misinformation has been shown to be rampant (Williamson, Hunt, Pope, & Tolman, 2000; Murrock, Taylor, & Marino, 2013). These findings, therefore, also support a new developmental model of eating pathology in which dietary misinformation is viewed as a risk factor or antecedent to distorted thinking about food, which can be reinforced through various developmental processes and is a proximal determinant of dieting and disordered eating. Future research would benefit from exploring the origins of misinformation, and whether the onset of inaccurate beliefs about food is related to future pathology.

Indeed, success across prevention programs could hinge on explicitly addressing dietary misinformation. This is encouraging, given that this construct is relatively easy to define and

measure; however, it is also disheartening, given that sources of dietary misinformation are ubiquitous and protecting against them presents a significant challenge. Ultimately, the identification of dietary misinformation as an important construct to study within eating pathology prevention greatly enhances current knowledge and suggests a novel, perhaps more modern way of understanding the developmental course of pathology in the current age. This construct also greatly enhances the understanding of other subclinical symptoms, such as distorted thinking about food.

Distorted Thinking about Food. Research that has shown that, just like eating disorder patients, nonclinical individuals engage in distorted thinking about food (Polivy & Herman, 1985). In the past, this research was conducted mostly by asking nonclinical individuals to complete clinical measures of eating pathology (de Lauzon et al., 2004). Generally, results showed that many nonclinical individuals scored in a clinical range across a number of domains, but did not merit an actual diagnosis when followed up by interview (Garner, Olmstead, & Bohr, 1982). Polivy and Herman (1985) showed that dieters showed clear patterns of distorted thinking by labeling themselves as “good” or “bad” based on their food choices and engaging in dichotomous thinking about food. Despite these findings, and the fact that eating pathology has been on the rise for decades across North America (Hoek, 2006), no studies have attempted to explore this construct further. This has pointed to the need for understanding cognitive antecedents to eating pathology in the general population. Indeed, no studies have attempted to define or measure distorted thinking about food, so it is an area that remained poorly understood. Thus, the current research enhanced the literature in a number of important ways.

First, distorted thinking about food was formally conceptualized, defined, and measured. This will allow future theoretical or applied research to use the CCS or improve on it in order to

measure this type of thinking in individuals or to measure change. Importantly, the CCS contained a “fair thinking” subscale. In doing so, this new scale offers an additional outcome to be measured at baseline or following intervention. Fair thinking was shown to be distinct from distorted thinking about food and associated with more positive clinical outcomes. Thus, attempting to increase fair thinking about food (rather than just reducing distorted thinking) may point to another potential target for intervention. Finally, this research also showed that distorted thinking about food is quite common among university students. This may not be surprising, given their tendency to consume social media and engage in dieting behaviours; however, it has highlighted that distorted thinking can occur across a spectrum of severity and be harmful, suggesting that the earlier individuals are armed with tools to combat it, the better. These findings are consistent with prevention theory that has promoted a non-specific vulnerability model (Cowen & Durlak, 2000; McVey, et al., 2010), and the success of the intervention in study 2 further supports this notion.

Program Outcomes. The main goal of study 2 was to develop and evaluate the Calorie Catastrophizing Workshop. This workshop was designed based on the tenets of Cognitive Behaviour Therapy and the Theory of Planned Behaviour and targeted distorted thinking about food and intentions to reduce distorted thinking about food. The purpose was to address the many weaknesses of existing prevention and intervention programs (see study 2). Outcomes of this study have some important implications for current research.

TPB Outcomes. Model analyses revealed that the TPB was successful in predicting behaviour, supporting its use for the current intervention. Results showed that the participants who attended the intervention experienced significant reductions in distorted thinking about food, dietary misinformation, and other important clinical variables such as body dissatisfaction

and symptoms of eating pathology. The intervention, however, had no impact on intentions to engage in fair thinking about food or to track food-related cognitions. This may be due in part to the fact that participants presented with high levels of attitudes and perceived behavioural control (PBC) related to both of these intentions, pointing to a potential “ceiling effect” at time 1 with little room for improvement in these scores. A possible explanation for this finding could be that university students are motivated to change diet and eating-related behaviours in general (Neighbors & Sobal, 2007), and these new behaviours were no exception. This is relevant to TPB research in a number of ways.

First, this study has highlighted the importance of considering the nature of a behavioural target in a TPB-based model or intervention. In this case, there may have been a pre-existing favourable bias towards engaging in fair thinking about food and tracking food-related cognitions. The current research has suggested that attempting to alter the intentional antecedents (i.e., attitudes, subjective norms, PBC) of a socially prescribed behaviour may not be successful if such a ceiling effect is observed, and in some cases it may be worth choosing a different behavioural outcome whose antecedents are more sensitive to change. Furthermore, the success of the current intervention may have hinged on providing participants with the skills necessary to act on existing intentions. This highlights the utility of combining CBT with TPB. Indeed, the combination of both models in the conceptualization of these variables and the design of the intervention was essential. TPB theory identified the key targets for the intervention (i.e., beliefs), while CBT identified an additional target (i.e., cognitions) and provided key strategies to elicit change in these variables. The success of this intervention may suggest that dieting and disordered eating prevention programs would benefit from a similar design, given that it simultaneously acknowledges societal factors and cognitions and it lead to significant clinical

outcomes. Despite these considerations, it may be valuable to consider these findings in light of recent criticisms of the TPB.

The TPB has been criticized for issues related to the validity and utility of the model (Sniehotta, Pesseau, & Araújo-Soares, 2014). Specifically, there have been few experimental tests of the model. Those that have been conducted are generally unsuccessful in targeting intentional antecedents, yet when these antecedents have changed successfully, it has rarely led to behaviour change (Hardeman, et al., 2002; McCarty, 1981). Other researchers have suggested that, depending on the behaviour in question, changes in beliefs can lead to changes in behaviour without modifying intentions (AraújoSoares, Rodrigues, Pesseau, & Sniehotta, 2013; Conner et al., 2013). The theory has also been criticized for having low predictive validity, suggesting that much of the variability in behaviour is not accounted for by the various components of TPB but by extraneous factors such as age, physical health, socio-economic status, and environmental factors (Sniehotta et al., 2014). Orbell and Sheeran (1998) also point out the problem of individuals who have the intention to engage in a behaviour but fail to act (also known as ‘inclined abstainers’), which has not been address by the theory. Finally, it has been suggested that other theoretical measures may be more successful at predicting behaviour change than the TPB, such as habit strength (Gardner, De Bruijn, & Lally, 2011), motivational measures (e.g. self-determination, anticipated regret and identity; Conner & Armitage, 1998) or self-regulatory measures (e.g. planning; Carraro & Gaudreau, 2013).

Sniehotta and colleagues’ (2014) condemnation of the TPB model is rigorous, but has itself been criticized for being overly harsh. For starters, it completely ignores and downplays the positive features of the model and the wealth of studies that have supported its use (Armitage, 2015). For example, the parsimony and potential for expansion is one of the strengths of the

model. Furthermore, authors promote other approaches to changing behaviour (such as ‘nudging’) that have far less evidence to support their use than the TPB (Hollands et al., 2013). With respect to “inclined abstainers”, the claim that this has not been addressed in research ignores many works by Ajzen that acknowledge that events occurring between the measurement of intention and the observation of behaviours can actually produce changes in intentions and these “unanticipated obstacles” can prevent people from carrying out their intentions (Ajzen, 2012; Ajzen & Sexton, 1999). Furthermore, the “extraneous variables” that affect behaviour could also be viewed as factors that affect underlying beliefs in TPB, which actually lends support to the model. Overall, the main weaknesses outlined by Sniehotta et al. (2014) suggest not that the TPB should be “retired”, but that more experimental research is needed in this area and that further theory development may be merited.

Given the fact that the current research is exploring a new area of research, it is expected that the TPB variables will be adjusted in future replication studies. Ajzen (2015) acknowledges that changing intentions and behaviour is not an easy task, and so TPB-based interventions require extensive piloting and formative research in order to determine the most optimal and appropriate targets for change. In many ways, the current research represents the important first step of this process. Findings, therefore, should not be taken as evidence that the TPB is not a suitable model, but should inform future directions of this research. This is not “apologizing” for TPB as Sniehotta et al. (2014) suggest, or ignoring “dismissing falsifying evidence”, but rather acknowledging the limits of the parameters of the current research. Indeed, there is not enough evidence, given the preliminary nature of the current research, to claim firmly that the TPB has failed in this case. In fact, using the TPB to conceptualize variables and design the intervention had a number of advantages. Most importantly, the TPB addresses societal factors that contribute

to behaviour change. In the case of dieting prevention, considering social norms and social messaging is essential, and these are two factors not explicitly addressed in other social cognition models and motivational theories.

Clinical Outcomes. As mentioned above, the current intervention was successful in changing a number of important clinical variables. First, it reduced distorted thinking about food and dietary misinformation. This is perhaps not surprising, given that these constructs were the main targets of the intervention. This is also encouraging, since these constructs could represent significant risk factors for the development of eating pathology. What may be surprising is the fact that this intervention was effective at changing variables that were not directly targeted.

Indeed, the workshop was successful in reducing body dissatisfaction and symptoms of eating pathology (as measured by the dieting subscale of the EAT). Taken together, these findings lend further support to the idea that misinformation, distorted thinking, and eating pathology are all linked in subclinical populations; however, further research is needed to fully understand how and why these variables changed together. It could be the case that teaching participants to challenge distorted thinking about food lead to lower internalization of diet myths, which then lead to fewer dieting behaviours; or it could be that reducing dietary misinformation meant that there were fewer sources of content for food-related cognitive distortions, which could also have lead to fewer dieting behaviours. Or, it may be the case that targeting both factors simultaneously really is the key to success in this study, highlighting the value of combining CBT and TPB in intervention design. Importantly, participants in the control group experienced little change other than an increase in eating pathology (also measured by the EAT). This may suggest that consuming any information about how one “should” eat potentially triggers dieting behaviours or possibly distorted thinking about food. Additionally, mood was not

sensitive and did not change following intervention. This may suggest that mood (as measure by the SPANE) is unrelated to the target variables in the current study. If this is the case, it lends further support to the intervention, given that participants were ostensibly not motivated to improve. If not, it suggests that perhaps a measure of mood other than the SPANE would better capture mood changes in the current study.

Ultimately, these findings suggest that successful prevention programs for eating pathology may depend on targeting these two constructs. In fact, dietary misinformation may be one of the earliest predictors or antecedents of eating pathology, and distorted thinking may be the process that mediates a number of problems related to eating pathology, ranging from misinformation to body dissatisfaction. Again, further research is needed to fully understand the relationship among all of these constructs. Finally, this research has a number of practical implications for prevention policy.

Intervention/prevention and policy. In order to combat the growing rates of dieting, disordered eating and lifestyle diseases, brief, low-cost and easily disseminated diet and lifestyle interventions are needed. The consequences of these problems range from weight management problems to the development of a clinical eating disorder (Field et al., 2003). Therefore, the onset of dieting and disordered eating should be avoided when possible. The “Calorie Catastrophizing Workshop” program is a brief (2 week) intervention/prevention program that significantly decreased distorted thinking about food and dietary misinformation, among other clinical variables which have been shown to be predictors of dieting and disordered eating. This intervention has the potential to be useful and affect policy in a number of ways.

First, this type of intervention is widely applicable. Although it has only been evaluated on a nonclinical university population, it is possible that it may be appropriate for individuals

across the age range and spectrum of severity. For youth, this workshop could serve as a form of primary prevention against dieting and disordered eating. This workshop indirectly teaches media literacy skills by encouraging participants to be critical of common diet myths and messages from the dieting industry. It also teaches a very important CBT skill: thought recognition and challenging. By learning to identify their own thoughts, youth will be able to challenge the negative thoughts that emerge that are associated with dieting and trying to lose weight. Essentially, the workshop provides skills to buffer against future pressure and pathology.

This workshop could also be widely disseminated to other university campuses or could target older populations. As such, it would serve as an intervention effort against distorted thinking about food, which could potentially lead to a reduction in current dieting and eating pathology. Finally, there may be some merit in evaluating this workshop on a clinical population of participants with eating disorders or obesity, in order to determine if it could serve as an adjunct to long-term therapy. In each case, further research is necessary to examine whether this workshop would be successful long-term at delaying the onset of dieting and overweight, changing dieting behaviour, or improving outcomes in eating disorder treatment.

Second, this intervention is convenient and low-cost. Although originally administered by a graduate student, paraprofessionals could be trained to successfully implement this intervention, which would further reduce the cost and increase accessibility. Overall, given the general failure of eating-related interventions geared towards various problems, a new way of treating these problems is necessary and the current intervention could be part of this novel approach.

Finally, this line of research has highlighted the importance of teaching youth and young adults how to consume health-related information responsibly. Given the high rate of dietary

misinformation in this university student sample, it suggests that more interventions are needed that explicitly teach participants how to search for accurate and reliable information, and how to challenge misinformation when they encounter it. This is particularly important for youth who consume this type of information through social media, given that previous research has shown that social media consumption is associated with misinformation and problematic eating behaviours (Syed-Abdul et al., 2013). Indeed, youth and young adults would benefit from learning new media literacy and critical thinking skills that focus on diet and fitness myths.

Limitations and Future Research Directions

These findings must be interpreted in the light of some important limitations. First, these studies were conducted on a convenience sample of university students, largely from undergraduate psychology. This might limit the generalizability of the results and greatly affects the utility of measures of effect size. Participants also self-selected into this study, which could explain the “ceiling effect” observed in study 2. Indeed, it is reasonable to assume that the subject pool is comprised of participants who are already motivated to change their thoughts about food. On the other hand, there are theoretical reasons to believe that these concerns are significant in this population anyway (Neighbors & Sobal, 2007), so this convenience sample could actually represent the ideal target population. In any case, future research would benefit from collecting a larger sample and conducting this research in different populations, such as adolescents or older adults in order to see how these concerns vary in different populations and to gain a better understanding of the effect sizes.

Second, certain participant characteristics were not considered in analyses. These factors (such as gender, eating disorder diagnosis, depression diagnosis) could be important to consider in future work. Given the exploratory nature of the current research, the focus was limited to

analyses on fundamental theoretical and clinical outcomes. Further splitting groups by another factor may have significantly reduced power to detect intervention effects. This sample was also predominantly white and female, so the understanding of how these concerns and this intervention apply to males or other cultures is limited. Future research would benefit from recruiting a more diverse sample in order to understand if these factors have a differential effect on eating pathology across different populations.

Third, given that this research was exploratory and two measures and an intervention were design and developed, future research is necessary to fully support the strength and use of these tools. For example, the Dietary Misinformation Questionnaire (DMQ) may need to be trimmed or adapted based on its preliminary factor structure. Furthermore, a new theoretical model of change was suggested, and it is the first time TPB and CBT were combined to understand subclinical eating pathology. More research is necessary to support this theoretical model, and it may be beneficial to run additional iterations of the model, such as a model in which cognitive variables from CBT are merged with TPB antecedents.

Finally, this research focused heavily on understanding dietary misinformation, but sources of misinformation were not explored. Understanding how misinformation develops (i.e., either from family, peer, or social media influences) may illuminate the role that it plays in the developmental course of eating pathology. This might also reveal the best method for addressing dietary misinformation, whether it is through some kind of knowledge inoculation (i.e., preempting social media misinformation) or the development of critical thinking skills for social media specifically. Furthermore, it is still unclear what individual factors contribute to the endorsement of diet myths (i.e. what makes certain individuals more likely to be misinformed?). Future research would benefit from exploring the origins of misinformation and how people

become misinformed in order to determine when it would be most appropriate and important to protect against it.

Conclusion

In this series of studies, dietary misinformation and distorted thinking about food were defined and measured using strong psychometric tools. A successful joint CBT-TPB workshop was also developed in order to target these problems and was successful at reducing them and other clinical variables, such as body dissatisfaction and some symptoms of eating pathology, despite not being explicitly targeted. This research greatly enhances current knowledge around the social and developmental nature of eating pathology and suggests a novel way of conceptualizing and preventing subclinical eating problems. Findings may even point to important considerations for the successful treatment of eating disorder patients or overweight youth in the modern age.

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Appendix A

Calorie Catastrophizing Scale

State to what extent you agree with the following statements:

1. Strongly Disagree 2. Disagree 3. Somewhat Disagree 4. Somewhat Agree 5. Agree 6. Strongly Agree

- CD: Catastrophizing
 1. Eating fat will make me fat, and being fat will make me unhappy
 2. Eating a treat that I crave (ice cream, candy, etc.) once a week will ruin my diet
 3. Having treats once in a while is not a big deal
- CD: All-or-Nothing
 1. If I ate 3 cookies when I planned to eat just 1 cookie, then my diet is ruined for the day
 2. If I ate 3 cookies when I planned to eat just 1 cookie, then I will eat the rest of the box of cookies
 3. If I ate 3 cookies when I planned to eat just 1 cookie, I don't let it affect my day
- CD: Labeling
 1. If I stray from my diet and eat candy when I was not supposed to, that makes me a bad person
 2. If I don't follow my diet, I am a failure
 3. If I don't follow my diet, I don't let it affect my day
- CD: Should Statements
 1. I should be able to follow a perfect diet
 2. I should be able to lose weight
 3. I don't feel like I should have to follow any specific diet or exercise plan
- CD: Should Not Statements
 1. I should not stray from my diet and/or exercise plan
 2. I should not give into food cravings if I have not planned for them
 3. There is nothing I feel I "should not" do when it comes to diet and/or exercise
- CD: Dwelling on Negative
 1. If I ate well most of the day but had a treat (such as ice cream or other), I am preoccupied with and worry about the ice cream.
 2. It doesn't matter how much I exercise or how well I eat – if I have a treat (ice cream, cookie, etc.), the day is ruined for me
 3. I don't let how much I exercise or what I eat affect my day
- CD: Mind-Reading
 1. When I have lunch or dinner with friends, I know they are judging me for what I order
 2. When I have a treat (ice cream, donut, etc.) in public, people think I have no self-control
 3. I don't know what people think when they see me eat food
- CD: Emotional Reasoning
 1. When I feel fat, it is because I have gained weight.

2. When I feel like eating a bunch of chocolate (or other treat), it is because I am hungry.
 3. The way I feel does not affect the way I think about food
- CD: Overgeneralization
 1. I ate poorly today – this means I will never be able to eat well.
 2. I will never be able to enjoy food.
 3. I feel differently about food from day to day.
 - CD: Disqualifying the Positive
 1. My day of healthy eating doesn't count if I skipped a workout
 2. My workout doesn't count if I had a treat (ice cream, etc.) today
 3. My diet and exercise don't affect how I feel about the other.

Appendix B

Dietary Misinformation Questionnaire

State to what extent you agree with the following statements:

1. Strongly Disagree 2. Disagree 3. Somewhat Disagree 4. Somewhat Agree 5. Agree 6. Strongly Agree

- All calories are created equally
- Calories eaten late at night turn to fat
- I absorb all of the calories I eat
- A small percentage of the calories I burn goes toward normal bodily functions (e.g., repairing body tissue, breathing, etc.)
- I burn most of my calories by the exercise I do
- Eating 4-6 smaller meals per day will increase my metabolism
- Skipping a meal will slow down my metabolism
- Metabolism slows down at night
- Celery is a “negative calorie food” (i.e., it takes more energy to digest it than it actually contains)
- A slow metabolism will make you fat
- Vegan and gluten-free junk food is healthier than “normal” junk food
- Humans are built to eat meat
- Humans don’t have the jaw or claws to catch and kill animals, thus they should not eat animals
- Modern humans should eat the way our ancient ancestors ate (i.e., follow the “Paleo diet”)
- Specialty diets, such as the Southbeach diet or the Atkins diet, etc. are better than the Health Canada food guidelines (i.e., the Food Pyramid)
- Drinking less than 8 cups (2 litres) of water per day is unhealthy
- Superfoods (gogi berries, acai berries, raw cacao, etc.) will make me super healthy
- Eating healthy is more expensive
- White bread (or pasta, rice) is unhealthy
- Most vegetarians and vegans lack protein in their diets
- Low-fat foods are healthier than higher fat foods
- Eating fat will make me fat
- Eating carbohydrates will make me fat
- Fruit is high in sugar and therefore is not healthy
- Eating lots of protein will guarantee muscle gain
- All cholesterol is bad for me
- Gluten is not healthy
- If I exercise, I can eat whatever I want and not gain weight
- After exercise, my body continues to burn more calories, so I can get away with eating higher calorie food
- Doing exercise on an empty stomach will burn more calories
- Cardio (e.g., running) is better for weight loss than weight-training (e.g., lifting weights)
- One pound of muscle weighs more than one pound of fat
- Women should not lift weights, because they become masculine
- You can target your exercise to lose weight in a specific body area

Appendix C

Problematic Eating Questionnaire

State how often you have had these thoughts (or engaged in these behaviours) in the last month:

0. Not at all. **1.** 1-2x per month **2.** 1-2x per week **3.** 3-4x per week **4.** 1-2x per day **5.** 3-4x per day **6.** 5 or more times per day

- I tried a new diet
- I tried a new exercise regime
- I deliberately restricted my calories
- I skipped a meal
- I had a “cheat day” (a day where I indulged/ gave into cravings/ did not follow my diet)
- I binged (ate a lot of food in a short amount of time – enough to feel uncomfortable)
- I felt out of control of what I was eating
- I purged by vomiting
- I purged by using laxatives
- I smoked a cigarette to avoid eating
- I fasted

- I ate too many calories or something unhealthy, so I had to “exercise it off”
- I ate too much at one meal, so I skipped the following meal to make up for it
- On a day that I was planning to drink alcohol, I skipped a meal to make up for the calories
- I compared what I eat to what my friends ate
- I felt disappointed in myself for eating too much

Appendix D

Theory of Planned Behaviour Questionnaire (TPBQ)

Attitudes

Tracking my thoughts about food for the next two weeks would be:

a bad thing :__1__: __2__: __3__: __4__: __5__: __6__: __7__: a good thing

unpleasant :__1__: __2__: __3__: __4__: __5__: __6__: __7__: pleasant

Changing my thinking about food over the next two weeks would be:

a bad thing :__1__: __2__: __3__: __4__: __5__: __6__: __7__: a good thing

unpleasant :__1__: __2__: __3__: __4__: __5__: __6__: __7__: pleasant

Changing my thinking about food is:

unimportant :__1__: __2__: __3__: __4__: __5__: __6__: __7__: important

Reducing my negative thinking about food will be:

unhelpful :__1__: __2__: __3__: __4__: __5__: __6__: __7__: helpful

Reducing my dietary misinformation about food will be:

unhelpful :__1__: __2__: __3__: __4__: __5__: __6__: __7__: helpful

a bad thing :__1__: __2__: __3__: __4__: __5__: __6__: __7__: a good thing

Subjective Norms

Most people who are important to me approve of me changing my thinking about food.

agree :__1__: __2__: __3__: __4__: __5__: __6__: __7__: disagree

Most people like me can benefit from changing their thinking about food

unlikely :__1__: __2__: __3__: __4__: __5__: __6__: __7__: likely

My friends would make fun of me if they knew I was trying to change the way I think about food

False :__1__: __2__: __3__: __4__: __5__: __6__: __7__: True

My friends would make fun of me if they knew I was tracking my thoughts about food

False :__1__: __2__: __3__: __4__: __5__: __6__: __7__: True

Most people who are important to me approve of me tracking my thoughts about food

agree :__1__: __2__: __3__: __4__: __5__: __6__: __7__: disagree

Perceived Behavioural Control

I am confident that I can track my thoughts about food for most days over the next 2 weeks

true :__1__: __2__: __3__: __4__: __5__: __6__: __7__: false

I am confident that I can change my thinking about food over the next 2 weeks

true :__1__: __2__: __3__: __4__: __5__: __6__: __7__: false

Tracking my thoughts about food is up to me

disagree: __1__: __2__: __3__: __4__: __5__: __6__: __7__: agree

Changing my thinking about food is up to me

disagree: __1__: __2__: __3__: __4__: __5__: __6__: __7__: agree

I am confident that I can track my food for most days over the next 2 weeks.

true: __1__: __2__: __3__: __4__: __5__: __6__: __7__: false

I am confident that I can change my thinking about food over the next 2 weeks.

true: __1__: __2__: __3__: __4__: __5__: __6__: __7__: false

Intentions

I intend to track my thoughts about food most days over the next 2 weeks.

likely: __1__: __2__: __3__: __4__: __5__: __6__: __7__: unlikely

I intend to change my thinking about food over the next 2 weeks.

likely: __1__: __2__: __3__: __4__: __5__: __6__: __7__: unlikely

Past Behaviour

In the past two weeks, I have tracked my thoughts about food.

false: __1__: __2__: __3__: __4__: __5__: __6__: __7__: true

In the past two weeks, I have changed my thinking about food.

false: __1__: __2__: __3__: __4__: __5__: __6__: __7__: true

Appendix E

Calorie Catastrophizing Workshop Intervention

Please contact authors for electronic version of slides.