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Reducing behavioural risk factors for cancer: An affect regulation perspective

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Nearly half of all cancer deaths are attributable to preventable causes, primarily unhealthy behaviours such as tobacco use, alcohol use and overeating. In this review, we argue that people engage in these behaviours, at least in part, as a means of regulating their affective states. To better understand why people engage in these behaviours and how researchers might design interventions to promote the selection of healthier methods for regulating affect, we propose a conceptual model of affect regulation. We synthesise research from both the stress and coping tradition as well as the emotion and emotion regulation tradition, two literatures that are not typically integrated. In so doing, we indicate where researchers have made headway in understanding these behaviours as affect regulation and note how our model could be used to structure future work in a way that would be particularly advantageous to cancer control efforts.

Keywords: cancer; affect; emotion; stress; coping; emotion regulation

Nearly 600,000 people die of cancer each year in the United States, making it a leading cause of death (second only to cardiovascular disease; American Cancer Society, 2016). While some cancers develop as a result of intrinsic, unavoidable cellular transformations, it is thought that 70 to 90% of cancers are due to extrinsic – environmental and behavioural – factors (Wu, Powers, Zhu, & Hannun, 2015). Indeed, it has been estimated that up to 50% of cancer deaths may be due to preventable behaviours such as tobacco use, alcohol use and overeating (Colditz, Wolin, & Gehlert, 2012). There is an urgent need to better understand both what motivates people to engage in these behaviours and how these behaviours can be changed.

One promising approach is to apply an affective science perspective to cancer control efforts (Ferrer, Green, & Barrett, 2015). People are generally motivated to do things that make them feel more positive affect and less negative affect (Gross & John, 2003). In order to achieve these goals, some individuals engage in unhealthy behaviours – including all of those noted above – that increase their likelihood of developing cancer (Colditz et al., 2012; Ferrer et al., 2015). In this review, we present a conceptual model of affect regulation (AR) that (a) organises research efforts aimed at understanding how

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these behaviours operate as a means of AR and (b) provides insights as to how maladaptive forms of AR might be replaced by more adaptive AR strategies.

In the first section, we begin by reviewing evidence that three, highly prevalent and preventable, cancer-causing behaviours – tobacco use, alcohol use and overeating – are maladaptive forms of AR. Next, we outline important concepts in the stress and coping and emotion regulation literatures and synthesise them into a broader conceptual model of AR. We then apply this model to our review of preventable behaviours, describing the ways in which existing research can be framed by the model and highlighting areas for future work. In particular, we emphasise the ways in which our AR model can be used to guide ongoing cancer control efforts both in terms of (a) more precise diagnoses of the root causes of maladaptive AR behaviours and also (b) more effective prevention and treatment of these behaviours.

Three behaviours that increase risk for cancer

In the following sections, we will examine three common and important types of behaviours which are preventable causes of cancer death: (1) tobacco use, (2) alcohol use and (3) overeating. For each class of behaviour, we first briefly describe the evidence that it causes cancer. Next, in more detail, we review the evidence that each can be understood as a form of AR, both in terms of increasing positive affect and in terms of decreasing negative affect.

Tobacco use

Tobacco use is one of the chief causes of cancer both in the United States and internationally (National Cancer Institute, 2016). Tobacco use claims more lives domestically than any other type of preventable behaviour (Centers for Disease Control & Prevention, 2016) and can lead to cancer in the lungs, larynx, mouth, oesophagus, throat, bladder, kidney, liver, stomach, pancreas, colon and rectum and cervix (National Cancer Institute, 2016). Although rates of smoking in the United States have declined over the past decade, smoking remains far and away the most common method for ingesting tobacco (Centers for Disease Control & Prevention, 2016). Given that the primary active ingredient – nicotine – is present in all forms of tobacco, insights regarding smoking tobacco as a method of AR likely apply to consumption of smokeless forms as well.

Many people report smoking tobacco because it increases positive affect and alertness (Heishman, Kleykamp, & Singleton, 2010; Ikard, Green, & Horn, 1969). Multiple studies have shown that nicotine increases dopamine activity in the nucleus accumbens, similar to other addictive substances such as cocaine and amphetamines (Pontieri, Tanda, Orzi, & Chiara, 1996). Additionally, research has shown that nicotine improves motoric and cognitive function more generally (Heishman et al., 2010; Rusted, Sawyer, Jones, Trawley, & Marchant, 2009). By some estimates, more than three quarters of people claim to smoke for these benefits (Ikard et al., 1969).

Despite the high rates of smoking for mood enhancement, researchers have disproportionately focused on smoking as a means of reducing negative affect. Many of these studies focus on smoking as way of coping with stress (Bindu, Sharma, Suman, & Marimuthu, 2011; Revell & Warburton, 1985; Steptoe, Wardle, Pollard, Canaan, &

Davies, 1996; Wills, 1986). According to numerous cross-sectional, self-report studies, smokers indicate that smoking helps them deal with stressful events (Niaura, Shadel, Britt, & Abrams, 2002; Parrott, 1995; Steptoe et al., 1996). The use of tobacco as a coping strategy is of course not limited to stress, however, and a substantial number of smokers report using tobacco to reduce negative emotions more generally (Ikard et al., 1969). Longitudinal investigations of the precursors to smoking have repeatedly demonstrated that negative affect and negative life events increase the likelihood that an individual will begin to smoke tobacco (Dugan, Lloyd, & Lucas, 1999; Wills, Resko, Ainette, & Mendoza, 2004). These studies have shown that not only is the intensity of negative affect associated with the onset of smoking, it also predicts increases in smoking over time (Kassel, Stroud, & Paronis, 2003; Wills, Sandy, & Yaeger, 2002).

Alcohol use

Alcohol use accounts for 4% of cancer incidence and, to date, seven types of cancer have been directly associated with alcohol use (Connor, 2016; Cancer Research UK, 2016; Parkin, 2011). Specifically, alcohol use has been associated with the development of mouth, throat, larynx, oesophagus, liver, colorectal and breast cancer, with the strongest associations for head and neck cancers (American Cancer Society, 2016; National Cancer Institute, 2016). The majority of these findings are for consumption of more than 3 to 3.5 drinks per day (National Cancer Institute, 2016). However, with regard to breast cancer in particular, even light to moderate drinking can increase risk (Connor, 2016).

Decades of research have firmly established that both normal and pathological drinkers use drinking as an AR strategy (Aldao, Nolen-Hoeksema, & Schweizer, 2010; Sher & Grekin, 2007). As with smoking, a substantial proportion of people who drink report doing so for mood enhancement, primarily to increase positive affect (Cooper, 1994; Cooper, Agocha, & Sheldon, 2000; Cooper, Frone, Russell, & Mudar, 1995; Pihl & Smith, 1988). Like nicotine, the consumption of alcohol increases dopaminergic activity in the nucleus accumbens (Yoshimoto, McBride, Lumeng, & Li, 1992). Unlike tobacco, however, alcohol is not known to enhance cognitive function. Rather, it exerts a sedative effect on the brain, primarily through interactions with inhibitory, GABAerigc receptors (Stevenson, 2013). The majority of people report drinking both to upregulate positive and to downregulate negative affect, but a substantial proportion (around a quarter of people) report only having one of these motives (Cooper et al., 1995).

There is unambiguous self-report evidence that people drink alcohol to regulate stress and negative emotions (Cooper, Russell, Skinner, Frone, & Mudar, 1992; Park, Armeli, & Tennen, 2004; Steptoe et al., 1996; Wills, 1986). Importantly, while negative affect leads to drinking in individuals with a range of drinking patterns, data from ecological momentary assessment (EMA) studies indicate that stress often immediately precedes binge drinking episodes, during which people drink well in excess of the 3 to 3.5 drinks per day level mentioned above (Grzywacz & Almeida, 2008). Longitudinal research shows that singular stressful events and living in chaotic environments also increase the likelihood that adolescents will develop problems with drinking (Mulia, Schmidt, Bond, Jacobs, & Korcha, 2008). Strengthening the causal story, several studies have shown that controlled laboratory stressors increase drinking behaviour in alcoholics as well as individuals who have social anxiety (Thomas, Bacon, Randall, Brady,

& See, 2011; Thomas, Bacon, Sinha, Uhart, & Adinoff, 2012; Thomas, Randall, & Carrigan, 2003).

Overeating

By some estimates, 20% of all cancer deaths are due to cancers resulting from excess body weight (American Cancer Society, 2016). The weight-related conditions brought on by overeating that most strongly confer an increased risk of developing cancer are obesity and metabolic syndrome. These diseases are associated with increased risk for breast, colorectal, endometrial, oesophageal, kidney, pancreatic, thyroid and gallbladder cancers (National Cancer Institute, 2016).

As with tobacco and alcohol use, people who overeat do so both to increase positive affect and to reduce negative affect (Adam & Epel, 2007; Boggiano et al., 2015; Epel et al., 2014; Mason et al., 2016). Researchers have shown that consuming highly palatable and calorie-dense foods activates reward-related neural circuitry in much the same way as drugs and alcohol (Volkow, Wang, & Baler, 2011). Researchers have developed individual difference measures that tap into the upregulation of positive affect as a reason for overeating. Individuals who report higher levels of 'reward-based eating' have higher BMI and exhibit increased weight gain at follow-up (Epel et al., 2014). People who score higher on global 'sensitivity to reward' are also more likely to engage in emotional overeating, where eating is motivated more by emotion rather than hunger (Davis, Strachan, & Berkson, 2004).

Reducing negative affect is also a powerful motivation for overeating (Ganley, 1989; Oliver & Wardle, 1999; Spoor, Bekker, Van Strien, & van Heck, 2007; Torres & Nowson, 2007; Wallis & Hetherington, 2009). Cross-sectional, self-report studies have consistently demonstrated these effects, as have longitudinal and EMA studies (Boggiano et al., 2015; Serlachius, Hamer, & Wardle, 2007; Torres & Nowson, 2007). Indeed, negative affect often immediately precedes episodes of binge eating (Skinner, Haines, Austin, & Field, 2012). Experimental studies support this general pattern of findings and have shown that exposure to both acute laboratory (Epel, Lapidus, McEwen, & Brownell, 2001; Oliver, Wardle, & Gibson, 2000; Wallis & Hetherington, 2004) and chronic, environmental stressors (Groesz et al., 2012) lead to increased eating. One important caveat is that certain studies only find effects for sub-populations of eaters, such as individuals who are obese (Geliebter & Aversa, 2003; Slochower & Kaplan, 1980), binge eaters (Arnow, Kenardy, & Agras, 1992; Haedt-Matt & Keel, 2011) or emotional or restrained eaters as classified by either the Three-Factor Eating Questionnaire or the Dutch Eating Behaviour Questionnaire (Heatherton, 1991; Wallis & Hetherington, 2004). A second caveat is that some studies show a positive association not with overall eating, but with the consumption of energy dense, highly palatable foods which have the same negative weight-related effects as overeating (Adam & Epel, 2007; Epel et al., 2001; Maier, Makwana, & Hare, 2015; Torres & Nowson, 2007).

Toward an integrative model of affect regulation

It is clear that people use tobacco, alcohol and eating to regulate affect. However, there are many reasons why a person might engage in one of these behaviours. In this section, we propose a conceptual model that makes more fine-grained distinctions among

processes that give rise to maladaptive AR. In so doing, we synthesise two literatures within affective science that have studied how people regulate their affect: the stress and coping literature (Lazarus, 1993b; Lazarus and Folkman, 1984) and the emotion regulation literature (Gross, 2015). Despite the shared focus of these two research traditions, they have infrequently been brought into conversation with one another. Uniting these two perspectives will provide a more flexible framework that applies to a broader set of situations than either alone. We begin by distinguishing among terms we will use in scaffolding our model. At the broadest level, we define *affect* as any psychobiological state that entails assessment of internal or external phenomena as good or bad (Gross, 2015; Scherer, 1984). Affect is inherently multidimensional in that different instances of affect vary widely in their duration, their granularity, and their cognitive complexity. Stress responses and emotions are the two sub-types of affect that are the focus of our model.

Stress and coping

Stress responses arise when a situation 'is appraised by the person as taxing or exceeding his or her resources and endangering his or her well-being' (Lazarus and Folkman, 1984, p. 21). The first stage in the development of a stress response is awareness of the stressor. Stressors may either be physical or psychological in nature and can vary widely in their duration. The stress and coping literature focuses primarily on psychological stressors (Lazarus, 1993a). The second stage in the development of a psychological stress response is *appraisal*. There is substantial variance in different people's responses to the same stressors due to differing appraisals, which are a psychological intermediary between stressor and stress response (Lazarus, 1993a). On this view, called the cognitive-mediational approach, researchers distinguish between two overarching types of appraisals: primary and secondary (Lazarus, 1993a). Primary appraisals refer to the initial good-for-me bad-for-me categorisations of sensory input that subsequently lead to more nuanced affective states (Lazarus and Folkman, 1984). Secondary appraisals are the judgments of whether something should or can be done to change the affective state generated by the primary appraisal (Lazarus and Folkman, 1984). As described below, this something can be directed at the affect itself or at the affect-producing stimulus. In addition to involving a judgement of whether coping is necessary, secondary appraisals also involve an assessment of whether the stressor exceeds the individual's coping resources (Lazarus and Folkman, 1984). These two types of appraisals lead to the third and final stage in this trajectory which is the expression of the *stress response*.

Although researchers have studied many different forms of coping, two broad categories may be distinguished. In emotion-focused coping, the target of efforts to change is the stress response itself (Folkman, Lazarus, Dunkel-Schetter, DeLongis, & Gruen, 1986; Lazarus, 1993a). Under the umbrella of emotion-focused coping, researchers distinguish between cognitive strategies that alter a person's construal of a stressful situation (such as reinterpreting the significance of the stressor) and behavioural strategies that engage more with external presentations of the stress (such as taking deep breaths or drinking alcohol) (Folkman & Lazarus, 1980; Folkman et al., 1986; Lazarus and Folkman, 1984). In problem-focused coping, the target of coping is the event that elicited the affective state (Folkman et al., 1986; Lazarus and Folkman, 1984).

Problem-focused coping involves engaging in problem-solving actions that alter something about the situation, the person, or the relationship between the two (Lazarus and Folkman, 1984).

Emotion and emotion regulation

Emotions are relatively short-lived affective states and tend to have a greater degree of differentiation than stress responses (Barrett, Mesquita, Ochsner, & Gross, 2007; Gross, 2015). Emotions coordinate cognitive, subjective, behavioural and physiological responses that arise in response to environmental and psychological demands (Barrett, 2006; Barrett et al., 2007; Mauss, Levenson, McCarter, Wilhelm, & Gross, 2005). One way of delineating the unfolding of an emotion is offered by the 'modal model' of emotion (Barrett et al., 2007). In this model, a person first encounters a psychologically significant change in their *situation,* which can be an aspect either of their external environment or their internal, psychological environment. The person then directs *attention* to this aspect of their situation and gives it an *appraisal* or interpretation as helpful or harmful to that person's well-being and current goals. Finally, this appraisal launches the above-mentioned set of *responses* across subjective experience, behaviour and physiology (Gross, 2015; Moors, Ellsworth, Scherer, & Frijda, 2013).

Emotions are often adaptive, but there are contexts in which emotions can interfere with important goals (Gross, 2015). At such times, people often engage in emotion regulation. The process model of emotion regulation organises the ways people try to change either the quantity of an emotion (i.e. quantitative emotion regulation) or the quality of an emotion (i.e. qualitative emotion regulation) by influencing one or more points of the emotion-generative process. Immediately prior to and during the situation stage, two kinds of strategies a person can implement are *situation selection* and *situation modification*. *Situation selection* refers to selectively entering into environments based on the effects they might have on one's emotional state. *Situation modification* describes strategies where one constructs or alters their current environment so as to change an emotion response. At the *attention stage*, one may select among different *attentional deployment* strategies which involve directing attention towards or away from emotion-eliciting aspects of one's current internal or external situation. *Cognitive change* tactics involve deliberate reconstruals of the meaning or significance of an emotion-eliciting situation at the *appraisal stage* – a widely studied strategy in this family is cognitive reappraisal. Finally, *response modulation* describes the strategies engaged after one has already developed a full-scale emotion response.

Affect and affect regulation

Given the common interests of the stress and coping literature and the emotion regulation literature, we have found it useful to think in terms of an integrated model of AR that draws on the extended process model of emotion regulation, but applies more broadly to both stress responses and emotions (Gross, 2015). Though there are many similarities between these two areas of study, there are key differences. The stress and coping tradition tends to examine acute as well as more chronic, temporally extended stressors. However, it does not deal with the same range or specificity of affective states as emotion regulation. Work on emotion regulation examines shorter periods of affect,

but is more precise about different types of affect and different types of regulation strategies. By proposing a model that synthesises these two literatures, we capitalise on the strengths of each to create a framework that captures a diversity of ways in which people regulate a broad set of both brief and extended affective states. This model answers questions regarding how and why certain regulatory strategies are chosen over others, such as why people engage in maladaptive AR strategies rather than healthier ones. As with our description of stress responses and emotions, our AR model begins with a schematisation of affect generation.

This model begins with an abstract conceptualisation of a valuation system (see Figure 1a) that captures shared features of what are likely many valuation systems distributed across the brain (Daw & O'Doherty, 2013; Rangel, Camerer, & Montague, 2008). In this approach, an individual perceives (P) some aspects of their current internal or external world (W). The individual then evaluates (V) this world state as good, bad or neutral. This valuation entails a judgement of the gap between the current state of the world and the state in which the individual wants to be. Finally, the individual decides whether or not to take action (A) – either physical or psychological – to reduce this gap between their actual and their desired states. On this perspective, it is this valuation cycle that constitutes the ongoing affective response. However, an individual does not often sit idly by, watching the affective response unfold. Instead, the individual can modify this first-level valuation by engaging a second-order valuation which implements actions aimed at influencing the affective state (see Figure 1b).

More specifically, during the *identification stage*, an individual perceives that they are experiencing a stress response, emotion or other affective state that is elicited by some feature of their internal or external situation. The individual then makes an assessment as to whether the affect requires regulation and, if it does, activates a goal

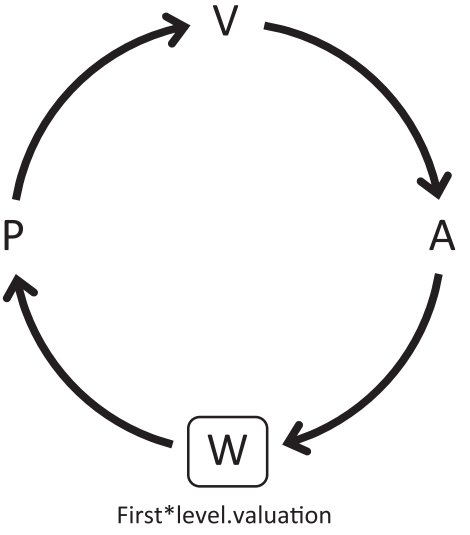

Figure 1a. A first-level valuation system generates an affective response.
Note: W stands for world, P for perception, V for valuation, and A for action, as described in the text.

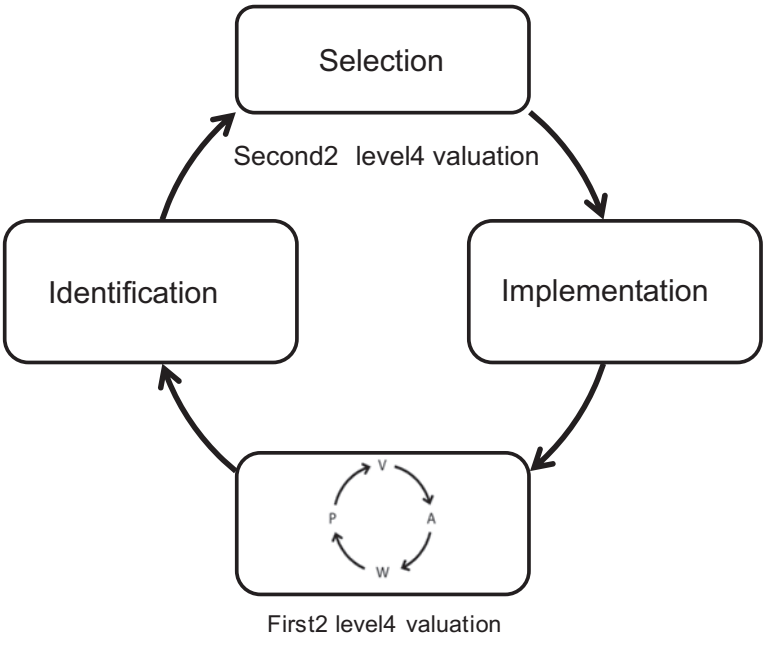

Figure 1b. The sequence of stages in affect regulation (a fourth stage – Monitoring – is not depicted).
Note: These stages represent the actions of a second-level valuation system that takes as its object a first-level valuation system that is generating an affective response.

to regulate this current state. In the *selection stage,* the affect that an individual wants to regulate serves as the input to the second-order valuation cycle. Next, an individual represents a variety of potential strategies they might use to alter their current affective state, determines which of these strategies are most likely to be successful, and subsequently selects the one which promises the greatest benefit at the lowest cost. Once a particular strategy has been selected, the individual progresses to the *implementation stage* where they enact the strategy they have selected (see Figure 2a). After implementing an AR strategy, an individual must engage in a *monitoring process* to dynamically assess their progress as they try to achieve a desired affective state. Importantly, an individual will continue to cycle through these various stages until they have successfully regulated their affect (or they have given up this goal). In so doing, an individual may realise that they have achieved success and are ready to stop regulating, or they may switch strategies if the current one is not working (for a more detailed discussion, see Gross, 2015).

Applying the AR model to behaviours that increase risk for cancer

In this section, we examine how problems at each stage of the AR model might lead a person to engage in the maladaptive forms of AR reviewed above (see Figure 2b). Throughout, we specify where existing research provides empirical support for our

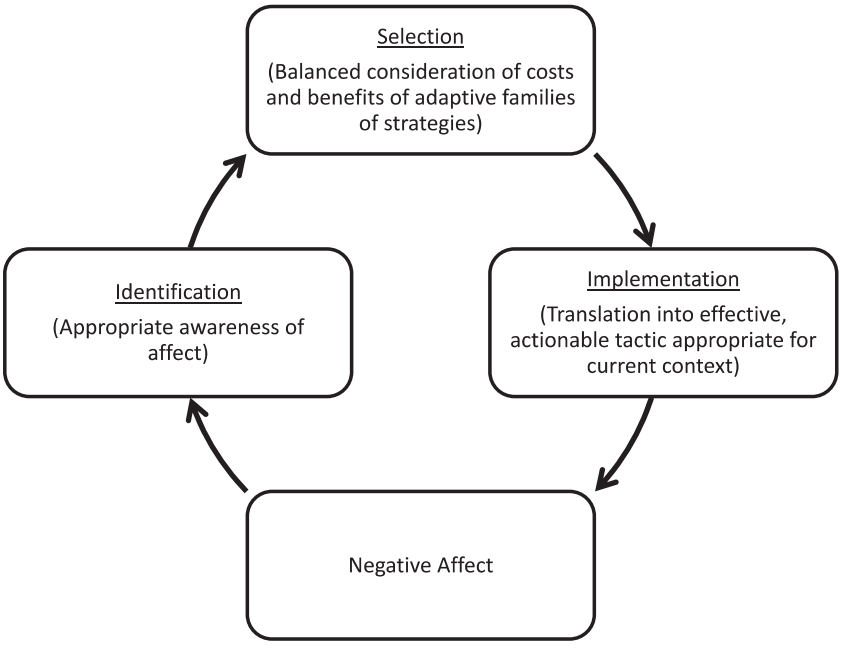

Figure 2a. Examples of healthy functioning at three out of the four stages of AR model in an instance of regulating negative affect.
Note: After an initial cycle, the fourth stage – monitoring – is set in motion and continues until successful regulation is completed.

hypotheses and highlight where additional research is needed. In so doing, we address tobacco use, alcohol use and overeating largely in concert, structured around our model rather than the behaviours themselves. It is also worth noting that, though we will address impairments at each stage separately, many people likely experience difficulties across multiple stages.

Identification stage problems

Difficulties at the *identification stage* of our AR model constitute a problem not with the execution of AR itself, but rather with the valuation process that determines whether one launches AR at all. At this stage, an individual could fail to accurately represent either their current affective state or a desired, future state. This misrepresentation comes in two forms: underrepresentation or overrepresentation (Sheppes, Suri, & Gross, 2015). Underrepresentation would manifest as awareness of only relatively coarse current and future affective states. This low-resolution sense of how one feels or how one wants to feel is likely to limit the set of options one has for regulation. There is limited evidence that people who use tobacco and alcohol have lower emotional intelligence that non-users (Fox, Hong, & Sinha, 2008; Trinidad & Johnson, 2002). Several studies have also demonstrated a positive relationship both between alexithymia – a non-clinical condition associated with impairments in emotional awareness – and alcohol use (Stasiewicz et al., 2012;

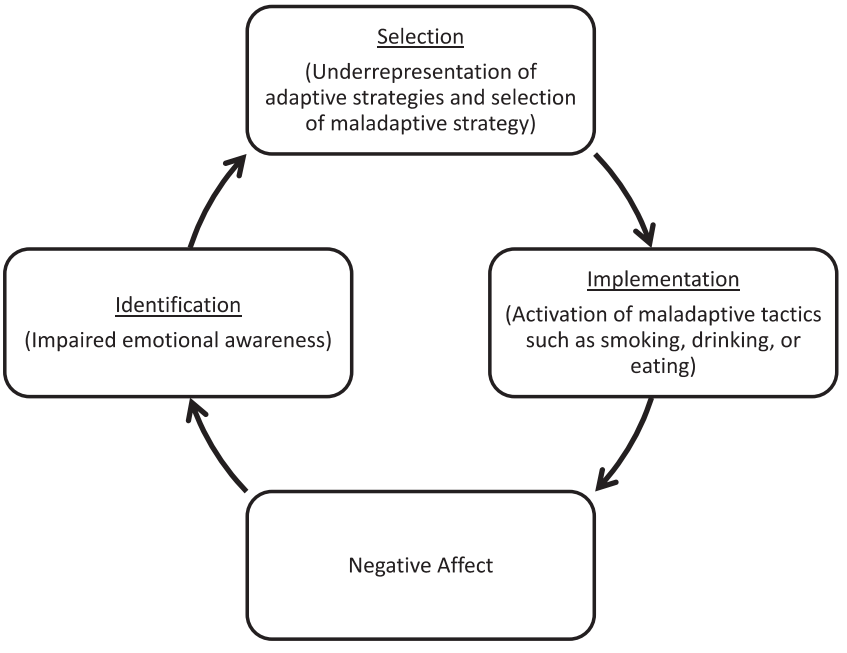

Figure 2b. Examples of unhealthy functioning at three out of the four stages of AR model in an instance of regulating negative affect.
Note: As in Figure 2a, only three out of four stages of AR model are presented here.

Thorberg, Young, Sullivan, & Lyvers, 2009) as well as between alexithymia and obesity (Wheeler & Broad, 1994). Overrepresenting a *current* affective state might take the form of an elevated sensitivity to what is actually low-level negative affect, which could lead an individual to launch a maladaptive strategy when AR is in fact not necessary. Overrepresenting a *desired* affective state, on the other hand, might correspond to enhanced reward sensitivity, which has in fact been shown to positively predict tobacco use, alcohol use and overeating (Davis et al., 2004; Jonker, Ostafin, Glashouwer, van Hemel-Ruiter, & de Jong, 2014; Potts, Bloom, Evans, & Drobes, 2014).

Selection stage problems

Difficulties at the *selection stage* are perhaps those most clearly related to the maladaptive cancer-causing behaviours reviewed in this article and the evidence is already quite strong that impairments at this stage lead people to use tobacco, drink alcohol and overeat. Failing to represent the various families of adaptive AR strategies that one can select among could lead one to choose a maladaptive behaviour as an AR strategy instead. For example, if a person is unaware of how to use cognitive change or how to redirect attention to lessen negative affect, smoking cigarettes, for instance, might be the only regulatory option they have. However, even if a person represents several of the more adaptive families of strategies, they might inaccurately assess the costs and benefits of each option. They might evaluate cognitive change tactics as being costlier

than they actually are or they might evaluate drinking alcohol as being more effective and less costly of an AR strategy than it actually is.

Indeed, people who smoke are more likely to believe smoking is an effective coping strategy and are less likely to think they have other, effective coping strategies at their disposal (Bindu et al., 2011; Dugan et al., 1999; Rabois & Haaga, 1997; Revell & Warburton, 1985; Wills, 1986). When examining specific coping styles, one study showed that individuals who were more likely to rely on their own resources for help, rather than turn to close friends or professionals, were more likely to smoke (Revell & Warburton, 1985). People who experience difficulties with emotion regulation are also at greater risk for relapse when trying to quit smoking (Farris, Zvolensky, & Schmidt, 2016). Though these studies do not address the causal nature of this relationship – whether reliance on smoking as an AR strategy leads to disuse of other coping strategies or whether people lack effective coping strategies to begin with – one longitudinal study found an inverse relationship between adaptive coping strategies and smoking in adolescents at follow-up (Wills, 1986).

A prominent theory of the mechanisms underlying alcohol abuse – social learning theory – posits that people with drinking problems view drinking as an effective coping strategy and are less likely to have other, more adaptive coping strategies at their disposal (Abrams & Niura, 1987; Britton, 2009). Researchers in the emotion regulation tradition have similarly shown that people drink alcohol as an emotion regulation strategy (Sher & Grekin, 2007) and impairments in emotion regulation predict relapse for people in treatment for alcohol-related disorders (Berking et al., 2011). The engagement of maladaptive AR strategies, such as rumination and avoidant-coping, is also predictive of drinking problems in longitudinal studies and is associated with increased problem-drinking in cross-sectional research as well (Cooper, Russell, & George, 1988; Nolen-Hoeksema & Harrell, 2002). Encouragingly, learning effective coping skills is one of the strongest protective factors against relapse for problem-drinkers who enter treatment (Brown, Vik, Patterson, Grant, & Schuckit, 1995). The evidence is not quite as strong for overeating, but studies have shown that people with higher levels of emotional eating are more likely to engage in emotion-focused coping than problem-focused coping and are more likely to have difficulties with emotion regulation (Gianini, White, & Masheb, 2013; Spoor et al., 2007). Several investigations have shown that people with binge eating disorders, tend to have fewer emotion regulation strategies at their disposal (Whiteside et al., 2007; Svaldi, Griepenstroh, Tuschen-Caffier, & Ehring, 2012). Additionally, in longitudinal studies researchers have found that individuals who report consuming food as a coping mechanism show increased BMI at follow-up compared to people who report eating for other reasons (Boggiano et al., 2015).

Finally, a person might have the capacity to represent a set of adaptive AR strategies and might even accurately assess which one will bring the greatest benefit at the lowest cost, but still have difficulty activating a goal to execute a particular family of strategy. One candidate cause of failure here is low AR self-efficacy. Though researchers have studied emotion regulation self-efficacy (Goldin et al., 2012), no studies have examined this construct in direct relation to the behaviours reviewed in this article. One factor that may influence AR self-efficacy could be that certain adaptive strategies, particularly within the often-studied attentional deployment and cognitive change families, require the activation of a broad set of executive functions (EF) (Sheppes et al., 2015).

If these requirements seem beyond a person's ability, they might choose a less cognitively taxing and potentially maladaptive AR strategy.

Implementation stage problems

Failures at the *implementation stage* consist of difficulties in translating the strategy chosen at the *selection stage* into a more specific and actionable tactic. Here, an individual might misrepresent the option space of a family of strategies either by underrepresenting certain tactics or overrepresenting others. There is broad agreement that behaviours like smoking, drinking and overeating come from the *response modulation* family (Gross, 2015; Khantzian, 1985). Tactics that intervene at the *response stage* of the modal model tend to be somewhat less effective than strategies engaged at earlier stages. Still, there are more adaptive ways of engaging in response modulation (Gross, 2015), and future work should test whether people who engage in maladaptive AR behaviours fail to represent these options.

Relatedly, inaccurate assessment of costs and benefits could lead individuals overvalue tactics within a certain family of strategy that promise immediate relief, but have severe long-term costs. One commonly studied form of this phenomenon is temporal discounting, which describes the fact that many people overvalue immediate rewards and undervalue future ones. Studies indicate that smoking tobacco, drinking alcohol, overeating and other forms of substance abuse are all associated with higher levels of temporal discounting (Epstein, Salvy, Carr, Dearing, & Bickel, 2010; Vuchinich & Simpson, 1998; Yi & Landes, 2012). Though not studied as often, temporal discounting could presumably also apply to costs of these behaviours, such as cancer.

Finally, an individual could fail to enact their chosen tactic due to a lack of resources. Several of the canonically adaptive AR strategies, such as attention deployment and cognitive change, tend to require more self-regulatory resources than using tobacco, drinking alcohol and overeating. Indeed, engagement in these behaviours is often seen as a failure of self-control. Lower levels of self-control and EF predict increased tobacco use and higher levels of alcohol use (Moffitt, Poulton, & Caspi, 2013; Moffitt et al., 2011; Wills & Stoolmiller, 2002). Unlike tobacco, alcohol use is also known to impair EF (Houston et al., 2014). There is evidence from decision-making studies that people who more often choose highly palatable foods are less likely to exhibit the activity in brain regions associated with cognitive control that is often displayed when individuals make healthier food choices (Hare, Camerer, & Rangel, 2009). In other words, engagement in these unhealthy behaviours may signal a lack of EF, a capacity crucial for implementing more resource intensive strategies.

Monitoring process problems

Though it is less likely that individuals who engage in the maladaptive health behaviours reviewed above experience problems exclusively in the *monitoring process*, it seems likely that problems could exist here as well. There is evidence that people who engage in each of the three preventable behaviours we reviewed may lack awareness that these misguided AR strategies do not actually reduce negative affect in the long run. Indeed, an important point of contention in the smoking literature is that although many smokers claim they smoke to reduce negative affect, smokers often also report

higher levels of negative affect than non-smokers (Kassel, 2000; Kassel et al., 2003; Parrott, 1995, 2000). An obvious potential cause for this increase in negative affect is nicotine dependence (Parrott, 1995, 2000). Relatedly, apart from the sedative effect of alcohol, there is conflicting evidence as to whether drinking alcohol is an effective AR method and does in fact reduce stress (Brown et al., 1995; Swendsen et al., 2000). As with smoking, a complicating factor here is the development of alcohol dependence (Heilig, 2007). The same logic could apply to overeating, but to date there is no evidence that overeating increases stress in the long run. Whether or not these behaviours do in fact reduce negative affect, the fact remains that people engage in them with the expectation that they will work as AR strategies. If an individual fails to properly monitor their affect, they may continue using these maladaptive strategies indefinitely.

Future directions

We have proposed ways in which difficulties at each of the different stages of our AR model might lead one to smoke tobacco, drink alcohol or overeat as a means of regulating affect. The existing research supports several of our propositions, but much work remains to be done. In this section, we describe several related areas that follow naturally from our review which could be particularly beneficial to cancer control efforts.

Measuring underlying psychological mechanisms

One pressing issue is developing measures to assess functioning at each of the stages of the AR model. For the *identification stage,* researchers can use psychometrically validated scales to measure alexithymia (Bagby, Parker, & Taylor, 1994) and emotional awareness (Lane, Quinlan, Schwartz, Walker, & Zeitlin, 1990), as well as sensitivity to positive and negative affect (Tapper, Baker, Jiga-Boy, Haddock, & Maio, 2015). Research on ideal affect, specifically the Affect Valuation Inventory (Tsai, Knutson, & Fung, 2006), could be adapted to measure valuations of current and desired affective states at this stage. An extensive body of work has also examined emotion and emotion regulation goals, which could be expanded to include additional categories of affect and to assess activation of AR goals (Mauss & Tamir, 2014).

In addition to self-report instruments, experimental assessments will also play a role. At the *identification stage*, a recently published study employed a novel task where participants identified specific emotions either with or without the help of verbal labels. Performance in the condition without verbal labels was negatively associated with alexithymia suggesting this task could be used to measure affect identification processes (Nook, Lindquist, & Zaki, 2015). In the affect valuation literature, researchers recently used neuroimaging in combination with a behavioural task to show that activity in reward-related brain regions was positively associated with types of affect that participants evaluated more favourably (Park, Tsai, Chim, Blevins, & Knutson, 2015). A similar type of task could presumably be used to measure affect valuation processes in individuals who engage in maladaptive AR behaviours to see whether certain valuation profiles predict higher levels of unhealthy AR.

At the *selection stage,* measures are needed that evaluate the different types of strategies people have available in different contexts. There are widely used measures that assess trait-level strategy selection, such as the emotion regulation questionnaire,

which could be expanded to measure other families of regulation strategies (Gross & John, 2003). Such measures could also be adapted to assess the costs and benefits that people associate with particular families of strategies. Additionally, some work has used self-report to assess emotion regulation self-efficacy (Goldin et al., 2012) which could be used to measure AR self-efficacy. To assess *selection stage* functioning experimentally, uninstructed AR tasks followed by in-depth debriefings could be used to assess both the broader families and the more specific within-family strategies that people tend represent when faced different types of affect (Opitz, Cavanagh, & Urry, 2015). Recent work on emotion regulation choice could be adapted so as to assess both valuation and selection of particular families of strategies or tactics within families (Sheppes, Scheibe, Suri, & Gross, 2011).

For the *implementation stage,* we imagine that a structured interview procedure could also be used to measures the tactics people represent within a particular family. The Difficulties with Emotion Regulation Scale (Gratz & Roemer, 2004) measures people's beliefs in their ability to implement selected strategies, but also contains items about responses to affect more generally. This questionnaire could be adapted to exclusively assess the difficulties people experience in implementing concrete strategies. What does not exist for this stage are instruments that measure valuation, the costs and benefits people associate with particular tactics. Measuring functioning at the *implementation stage* may prove difficult, but one potentially fruitful line of research could leverage the use of psychophysiological, electroencephalography or other known neuroimaging indices of emotional arousal to index success at implementing particular strategies (Foti & Hajcak, 2008; Ochsner & Gross, 2014). Also at the *implementation stage*, future research could administer EF and AR tasks in the same session to examine whether performance on standard cognitive control tasks predicts AR effectiveness and self-reported engagement in maladaptive AR strategies.

Examining individual and group differences

Many important unanswered questions regarding our AR model concern individual and group differences. Do people experience impairments in particular processes that predict engagement in specific types of maladaptive AR behaviours? Do certain people experience impairments at a certain stages as more incapacitating than others? Given that AR abilities develop over time (McRae et al., 2012), which stages do people experience more difficulties with early on? In the section on overeating, we identified specific types of individuals who are particularly likely to use overeating as a coping strategy – emotional eaters, restrained eaters and individuals who binge eat. Are there similar sub-groups among those who use tobacco and use alcohol? We also noted that people who use tobacco, abuse alcohol and overeat tend to have more global coping difficulties. One crucial unanswered question concerns temporal precedence. Though there is one study on this topic (Wills, 1986), it is not well understood whether certain individuals who use maladaptive AR strategies do so because of pre-existing AR difficulties or whether problems with AR develop as a result of an overreliance on these maladaptive strategies.

One important type of individual difference that we have not yet discussed concerns socioeconomic status (SES). People of low SES disproportionately engage in the maladaptive forms of AR reviewed above (Pampel, Krueger, & Denney, 2010). SES is a highly complex, multidimensional construct and there are likely many factors that

influence this link. Some research has already shown that exposure to the kinds of chaotic environments and uncontrollable stress common in low SES areas leads to AR deficits (Evans & Kim, 2013; Troy, Shallcross, & Mauss, 2013) and lower SES is associated with alexithymia (Lane, Sechrest, & Riedel, 1998). One study has shown that more efficacious use of certain AR strategies correlates positively with SES (Côté, Gyurak, & Levenson, 2010) and there is also evidence that people of lower SES are less likely to use cognitive reappraisal (Troy, Ford, McRae, Zarolia, & Mauss, 2016), but no evidence as to how this relates to the selection of alternate AR strategies. Encouragingly, if low SES individuals employ cognitive reappraisal, however, they appear to receive greater benefits than their high SES counterparts (Troy et al., 2016). The outsize prevalence in these lower social strata of the unhealthy behaviours we reviewed merit special attention to determine whether there are types of AR deficits specific to low SES individuals.

A distinct, but closely related group difference which has significant implications for engagement in maladaptive AR is race. Racial minorities are more often socioeconomically disadvantaged and research has consistently shown that racial minorities, black Americans in particular, tend to experience higher levels of stress and negative affect than non-Hispanic whites (Turner & Avison, 2003). With the exception of alcohol use, black Americans are more likely to engage in the unhealthy forms of AR described above and, correspondingly, are more likely to experience negative physical health outcomes that are associated with increased risk of developing cancer (Mezuk et al., 2010). Paradoxically, though black Americans experience increased stress and negative affect compared to other racial groups, studies have repeatedly shown that their mental health is either the same or better than non-Hispanic whites (Riolo, Nguyen, Greden, & King, 2005). One research group has shown that for black Americans experiencing high levels of stress, the more often they engage in unhealthy AR behaviours, the less likely they are to be depressed – an interaction not observed among non-Hispanic whites (Jackson, Knight, & Rafferty, 2010; Mezuk et al., 2010). Indeed, it seems as though black Americans successfully use maladaptive forms of AR to more successfully preserve their mental health, but at a significant cost to their physical health (Jackson et al., 2010; Mezuk et al., 2010). Thus, in addition to addressing societal sources of increased stress, future work must address how to provide racial minorities with healthy means of AR.

Developing enhanced interventions

Using the proposed AR model to scaffold intervention development is one the areas we think most urgently needs attention in future research. A more fine-grained understanding of which specific AR impairments lead to the selection of maladaptive behaviours as regulatory strategies will undoubtedly direct intervention development, but there are already a number of useful starting points to guide this research. Certain interventions might involve not simply replacing maladaptive AR strategies with more adaptive ones, but rather intervening prior to the *selection stage* altogether. Interventions or trainings designed to enhance processes at the *identification stage,* such as emotional awareness, might facilitate the selection of more adaptive AR strategies. One promising effort in this area is mindfulness interventions, which is now widely applied in behaviour change as well as in treating various forms of psychopathology (Daubenmier et al., 2016;

Goldin, Ziv, Jazaieri, & Gross, 2012). In the past decade, researchers have also begun to develop emotional intelligence trainings that could be used to help people with difficulties at the *identification stage* (Slaski & Cartwright, 2003). If our hypothesis that people who have low levels of emotional awareness are more likely to engage in maladaptive AR behaviours due to their inability to fully elaborate current and desired affect states that set the entire AR process in motion, such trainings might be of use to them.

Given the existing research on *selection stage* difficulties, interventions that teach people new AR strategies will clearly benefit people who engage in maladaptive AR behaviours. Many forms of therapy, including cognitive behavioural therapy, dialectical behaviour therapy, and mindfulness-based treatments, all incorporate forms of AR training (Goldin et al., 2012). Armed with more precise information regarding specific AR-stage deficits, researchers could fine-tune these therapies to teach individuals specific AR strategies. Strategies that intervene at the *response stage* of the modal model – such as smoking, drinking and overeating – tend to be somewhat less effective than strategies engaged at earlier stages (Gross, 2015). Put simply, it is easier to regulate affect before it has developed into a full-blown response than afterwards. Thus, teaching people strategies that target earlier points in the affect generation trajectory could reduce people's use of unhealthy behaviours as AR tactics and would therefore be not only healthier, but could potentially also be more effective. Additionally, we know that high intensity affect leads people to choose less cognitively intensive AR strategies over others (Sheppes et al., 2011). Perhaps interventions could train people how to choose adaptive AR strategies under conditions of duress, even if they require more cognitive resources.

Concluding comment

Cancer is the second most common cause of death in the United States (American Cancer Society, 2016). The notion that nearly half of all cancer deaths are due to preventable behaviours is at once disheartening and encouraging. It is disheartening because these deaths could be avoided. It is encouraging because if researchers continue to develop effective ways to intervene and help people avoid these behaviours, many lives could be markedly improved and extended. In proposing the model of AR outlined in this article, we hope to have furthered a growing area of research in which findings in affective science can be used to guide cancer control interventions. We reviewed several unhealthy, but preventable, AR behaviours that are major contributors to the incidence of cancer in the United States; there are of course other behaviours that play a role as well. Additionally, we have outlined several areas of future research where we think empirical studies would produce particularly useful results. We look forward to seeing how findings, methods, and theoretical frameworks drawn from affective science, such as the one developed in this article, can be applied to help cancer control efforts.

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