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Dyshomeostasis, obesity, addiction and chronic stress

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Abstract

When eating control is overridden by hedonic reward, a condition of obesity dyshomeostasis occurs. Appetitive hedonic reward is a natural response to an obesogenic environment containing endemic stress and easily accessible and palatable high-energy foods and beverages. Obesity dyshomeostasis is mediated by the prefrontal cortex, amygdala and hypothalamic–pituitary–adrenal axis. The ghrelin axis provides the perfect signalling system for feeding dyshomeostasis, affect control and hedonic reward. Dyshomeostasis plays a central role in obesity causation, the addictions and chronic conditions and in persons with diverse bodies. Prevention and treatment efforts that target sources of dyshomeostasis provide ways of reducing adiposity, ameliorating the health impacts of addiction and raising the quality of life in people suffering from chronic stress.

Keywords

addiction, chronic stress, Circle of Discontent, dyshomeostasis, ghrelin, hedonic reward, obesity

Homeostasis is omnipresent in nature and all living things. It occurs within individual organisms, in social milieu and in the environment. At the biochemical, physiological, psychological and social levels, smooth functioning of healthy organisms relies upon the successful operation of homeostasis. However, wherever there is homeostasis, there is the potential of dyshomeostasis. When homeostasis is disrupted, the well-being of an individual, family or population is placed in jeopardy. Recently, the principle of dyshomeostasis was applied to the explanation of obesity (Marks, 2015).

The key scientific dilemma is to understand how obesity can happen in the first place and on the global scale that exists at the present time. There has been a theoretical vacuum concerning obesity that defies logic and imagination. A phenomenon that is so pervasive cannot lie beyond explanation in science. The explanation, I believe, is a relatively simple but neglected one: obesity is a form of dyshomeostasis. In this article, I elucidate preliminary hypotheses concerning the neurobiological basis of obesity dyshomeostasis (OD) and discuss issues raised by commentators (Annunziato and Grossman, 2016; Brindal and Wittert, 2016; DiClemente and Delahanty, 2016; Markey et al., 2016; Pelletier et al., 2016; Piko and Brassai, 2016; Roosen and Mills, 2016; Rosenbaum and White, 2016).

A theoretical vacuum

The accepted explanation of overweight and obesity has been the Energy Balance Theory (EBT) in which weight gain is a consequence of energy expenditure being less than energy intake. This mechanistic approach led to the modern obsession with calorie counting and dieting (Marks et al., 2015). It is true that short-term weight loss can be achieved by any calorie-reducing diet but, in the long-term, studies show that calorie counting is not associated with significant weight loss. One reason for this outcome is that all calories are not equal (Feinman and Fine, 2004). If you eat an equal number of calories of protein, fat and carbohydrates, the metabolic processes are different, and calories from fat are more likely to end up on your waist as fewer calories are burned by the thermic effect of eating. The quality and type of foods one consumes influence diverse pathways related to weight homeostasis, such as brain reward, hunger, glucose-insulin responses, satiety, adipocyte function, metabolic expenditure and the

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microbiome. All calories are not equal: some foods impair pathways of weight homeostasis and others promote the integrity of weight regulation. In sum, the EBT is an oversimplified, descriptive approach that has promoted victim blaming and stigmatization, which has done little to reduce the prevalence of obesity (Marks et al., 2015). One might even say, it increased it.

Associated with the EBT is the view that obesity and overweight are the consequences of inactivity. This belief has been responsible for much disillusionment among people striving for significant weight loss. A 100-kg man needs to run about 20 km each week to reach a weight of 85 kg. However, this outcome would take approximately 5 years using exercise alone. That would mean running 5000 km, one-eighth of the circumference of the planet, over 5 years to lose 15 kg (Marks et al., 2015). It is perhaps unsurprising that systematic reviewers have concluded that adding physical activity (PA) to a dietary intervention for obese individuals has a marginal, if any, effect on average weight loss (Loveman et al., 2011; Swedish Council on Health Technology Assessment, 2013).

The inability of EBT to yield effective long-term interventions for obesity treatment or prevention suggests to this author that the energy balance approach is theoretically bankrupt. It is a purely descriptive theory of energy transfers in and out of the body but it fails to tell us predictively why any one individual will develop obesity rather than another. The EBT falls short, not only for lack of explanatory power but also because it has done actual harm by stigmatization of overweight individuals who have been blamed for being both 'greedy' and 'lazy'. The EBT is no longer considered helpful to the complete understanding of obesity and should be retired.

Enter homeostasis theory. The theory of the Circle of Discontent (COD) proposes that homeostatic control of eating can become disrupted under the conditions of modern living in which large sectors of the population are exposed to chronic stress and negative affect while simultaneously being offered supplies of low-cost fatty and sugary foods. Under such oppressive conditions, palliation of stress and negative affect is facilitated by the hedonic eating of high-energy, high-fat or high-sugar foods and beverages, indubitably the main cause of obesity. Over a protracted period of time, OD has a highly deleterious impact on human physical and mental health and is associated with metabolic syndrome, insulin resistance/diabetes, cardiovascular disease, cancers, fatty liver disease, polycystic ovarian disease, depression and many other conditions that are not easily reversed, or are irreversible.

The neurobiological basis of obesity

The first place to look for an explanation of OD is neurobiology. In the obese person, something has gone wrong within the psychoneuroendocrinal system. Clearly, the mechanisms responsible for feeding control have been disrupted. But what is the nature of the disruption? And why one person rather than another?

Obesity occurs as a consequence of a disruption to the homeostatic mechanisms that regulate the control of eating. When addressing the spectrum of clinical conditions that are the concern of medical and clinical science, the idea of homeostasis imbalance is quite ancient. Since classical theories of Hippocrates and Galen, the history of clinical medicine has been associated with the fundamental principle of dyshomeostasis. What is perhaps surprising is that dyshomeostasis has not previously been cited as the cause of obesity. In the following, the case for dyshomeostasis in obesity will be elaborated. Interesting parallels will become apparent between eating and other forms of consumption which rely on similar neurobiological mechanisms: the addictions to nicotine, alcohol and illicit drugs and also behavioural addictions. The following sections discuss known biological mechanisms that relate to the psychological and social issues included in the 'COD'. In doing so, the opportunity to lend 'a truly integrative biopsychosocial lens' is embraced as suggested by Rosenbaum and White (2016).

Dyshomeostasis in human feeding

In an environment that promotes widespread body dissatisfaction, angst and depression, homeostatic feedback loops are producing excessive consumption of unhealthy processed foods that over a protracted period causes obesity in large numbers of vulnerable people. Multiple clinical studies in different areas of medicine demonstrate the primary role of homeostasis in healthy functioning and the consequences of dyshomeostasis. Homeostasis can be overloaded or overridden with too strong a flow of inputs or outputs that disrupt its normal functioning: 'The homeostatic behaviour of inflow controllers breaks down when there are large uncontrolled inflows, whereas outflow controllers lose their homeostatic behaviour in the presence of large uncontrolled outflows' (Drengstig et al., 2012). Homeostasis can be disrupted anywhere, and perturbations will inevitably occur in normal functioning (Richards, 1960).

There are many examples of dyshomeostasis in clinical medicine. Well-known to psychologists, Hans Selye reported that a persistent environmental stressor (e.g. temperature extremes), together with an associated homeostatic hormonal response, leads to tissue injury that he termed a 'disease of adaptation' (Selye, 1946). Intestinal homeostasis breaks down in inflammatory bowel disease (Maloy and Powrie, 2011) and in the microbial ecology of dental plaque causing dental disease (Marsh, 1994). This form of dyshomeostasis can result from local infection and inflammation and give rise to complications that affect the nervous and endocrine systems (Maynard et al., 2012). An altered balance between the two major enteric bacterial

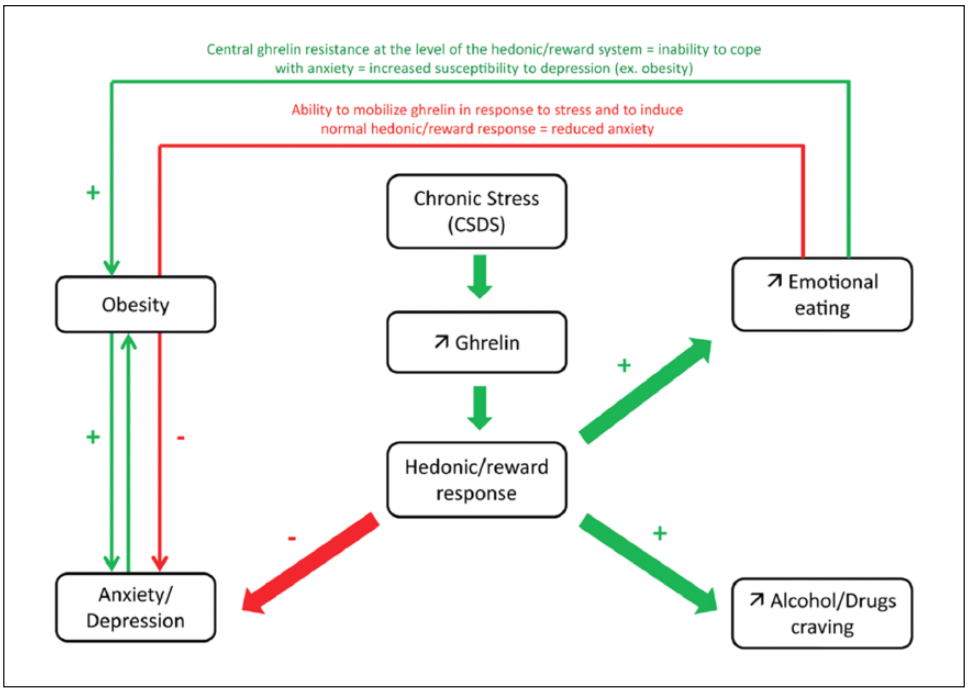

Figure 1. Model of hedonic/reward response to ghrelin after chronic stress in relation to anxiety and depression.
During chronic stress, increased ghrelin secretion induces emotional eating as hedonic reward. Ghrelin has anxiolytic actions in response to stress; this adaptive response helps to control excessive anxiety and prevent depression. In obesity, a lower ability to mobilize ghrelin in response to stress or central ghrelin resistance at the level of the hedonic/reward system may explain the inability to cope with anxiety and increased susceptibility to depression. Reciprocally, people suffering from depressed show increased susceptibility to obesity or eating disorders (due to an altered hedonic/reward response). Elevated ghrelin may also contribute to alcohol/drug craving as higher ghrelin levels correlate with greater alcohol craving. Reproduced from Labarthe et al. (2014).

phyla, the Bacteroidetes and the Firmicutes, has been associated with clinical conditions. Within the microbiota of the gut, obesity has been associated with a decreased presence of bacteroidetes and an increased presence of actinobacteria (Ley, 2010; Turnbaugh and Gordon, 2009). Kamalov et al. (2010) proposed a dyshomeostasis theory of congestive heart failure. Craddock et al. (2012) suggested a zinc dyshomeostasis hypothesis of Alzheimer's disease.

Homeostasis regulation within the endocrinal and central nervous systems has been associated with feeding control. Cortical areas conveying sensory and behavioural influences on feeding provide inputs to the nucleus accumbens (NAc) and the lateral hypothalamic area (LHA) is the site of homeostatic and circadian influences (Saper et al., 2002). Hormones such as leptin circulate in proportion to body fat mass, enter the brain and act on neurocircuits that govern food intake (Morton et al., 2006). Through direct and indirect actions, it is hypothesized that leptin diminishes the perception of food reward while enhancing the response to satiety signals generated during food consumption that inhibit feeding and lead to meal termination.

Another important hormone is ghrelin which is the only mammalian peptide hormone able to increase food intake. Interestingly, ghrelin also responds to emotional arousal and stress (Labarthe et al., 2014; Müller et al., 2015). During chronic stress, increased ghrelin secretion induces emotional eating by acting at the level of the hedonic/reward system. As ghrelin has anxiolytic action in response to stress, this adaptive response may contribute to control excessive anxiety and prevent depression (Labarthe et al., 2014). In obesity, studies have shown a reduced ability to mobilize ghrelin in response to stress or central ghrelin resistance at the level of the hedonic/reward system which may explain the inability to cope with anxiety and increased susceptibility to depression (Figure 1). Reciprocally, studies have shown that people with depression have increased susceptibility to obesity and eating disorders (Marks, 2015).

In addition to leptin and ghrelin, other lipid messengers that modulate feeding by sending messages from the gut to the brain have been identified. For example, oleoylethanolamine has been associated with control of the reward value of food in the brain (Lo Verme et al., 2005; Tellez et al., 2013). Mice fed a high-fat diet had abnormally low levels of oleoylethanolamine in their intestines and did not release as much dopamine compared to mice on low-fat diets. Thus, alterations in gastrointestinal physiology induced by excess dietary fat may be one factor responsible for excessive eating in the obese (Tellez et al., 2013).

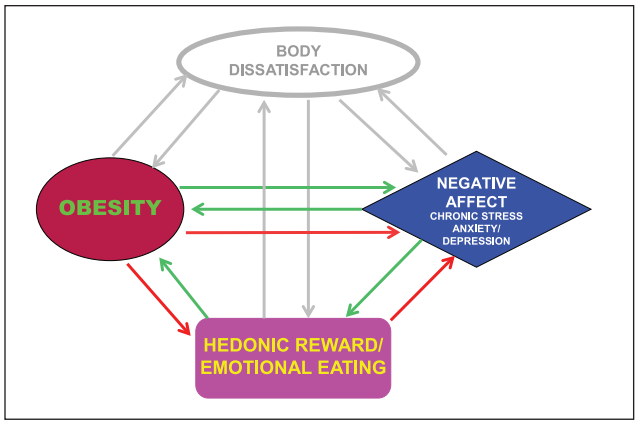

Figure 2. The potential role of ghrelin in obesity dyshomeostasis and the hedonic reward system in the amelioration of negative affect, chronic stress, anxiety and depression.
The Circle of Discontent (COD) theory is paralleled by the model of hedonic/reward response to ghrelin after chronic stress proposed by Labarthe et al. (2014) (as previously shown in Figure 1). The original version of the model by Labarthe et al. has been synced with the COD theory. Green arrows represent increased levels of ghrelin, while red arrows represent decreased ghrelin activity, as shown in Figure 1. The part of the COD shown in light grey was not included in the Labarthe model. The amended Labarthe model is a perfect fit to OD theory and lends the theory a neurochemical basis.

OD theory holds that obesity is caused by the imposition of the hedonic reward system, designed for the amelioration of chronic stress, anxiety and depression, overriding homeostasis. Within OD theory, the COD (Marks, 2015) is closely paralleled by the model of the hedonic/reward response to ghrelin (Labarthe et al., 2014; Figure 1).[1] The Labarthe et al. model contains some redundant features and duplication of constructs that can be avoided. In Figure 2, 'chronic stress' and 'anxiety/depression' are merged into the single construct, 'negative affect'. Similarly, in the context of obesity, 'hedonic reward/response' and 'emotional eating' are also operationally a single process. With these amendments, it can be seen that the simple diamond structure of the COD emerges from the Labarthe model (Figure 2). The model of Labarthe et al. (2014) provides the COD with a framework inside neurochemistry.

Convergence of OD theory with neurobiology

Traditionally, the control of feeding has been associated with the hypothalamus (Kelley et al., 2005). Circulating factors in the blood modulate the activity of energy sensing neurons in the arcuate nucleus, that modulate food-directed behaviours via their activation of outputs from lateral hypothalamic regions to thalamocortical systems, central autonomic effectors and motor pattern generators. There is a convergence of inputs from amygdala, prefrontal cortex (PFC) and NAc shell that allow direct modulation of feeding behaviours based upon cognitive and affective signalling. These avenues of influence on feeding control provide the entry point for the COD. When the environmental conditions are (a) obesogenic, due to the ready availability of highly palatable high-energy foods; (b) stressful, due to the presence of stigma, depression and anxiety; and (c) engendering body dissatisfaction, due to the omnipresent sociocultural thin-ideal, we have all the ingredients necessary for obesity formation. According to OD theory, the cognitive and affective processes of the COD override the neurobiological processes that regulate feeding and energy homeostasis breaks down.

The amygdala, PFC and NAc participate in the regulation of both affect and feeding. The amygdala consists of a group of nuclei involved in emotional learning and expression, a key element of the neural basis of emotion. Damage to the amygdala may lead to an increased threshold for emotional perception and expression, impairments in emotional learning, deficits in the perception of facially expressed emotion and impaired memory for emotional events (Cardinal et al., 2002).

Among younger adults, it has been found that the ability to wilfully regulate negative affect, enabling effective responses to stressful experiences, engages regions of PFC and the amygdala. Urry et al. (2006) tested whether PFC and amygdala responses during emotion regulation predict the diurnal pattern of salivary cortisol secretion. They also tested whether PFC and amygdala regions are involved in emotion regulation in older (62–64 years) individuals. They measured brain activity using functional magnetic resonance imaging as participants regulated (by intentionally increasing or decreasing) their affective responses or attended to negative picture stimuli. Urry et al. also collected saliva samples for 1 week at home for cortisol assay. Increasing negative affect resulted in ventral lateral, dorsolateral and dorsomedial regions of PFC and amygdala activation. The predicted link between brain function in the PFC and amygdala occurred while reducing negative affect in the laboratory and diurnal regulation of endocrine activity in the home environment (Urry et al., 2006). The authors concluded that functional coupling between the PFC and the amygdala enables effective regulation of negative emotion and the activity of PFC–amygdala circuitry during regulation of negative affect predicts longer-term regulation of endocrine activity that may be important for health and well-being.

In the OD theory, negative affect causes increased feeding. This causal relationship is made possible by the fact that the system that regulates negative affect, the hypothalamic–pituitary–adrenal (HPA) axis also regulates feeding, and thus each process influences the other in eliciting increased consumption (Maniam and Morris, 2012). Negative affect induces increased comfort food intake and body weight gain in humans (Dallman et al., 2003). In rats, chronic stress produces decreases corticotropin-releasing factor (CRF) mRNA in the hypothalamus. Depressed people who overeat have

decreased cerebrospinal CRF, catecholamine concentrations, and HPA activity. In line with the COD theory, it has been proposed that people eat comfort food in an attempt to reduce the activity of the chronic stress–response network with its attendant anxiety (Dallman et al., 2003, 2004).

Obesity is associated with neuroendocrine disturbances, in which the HPA axis plays a central role. The HPA axis is stimulated by negative affect which is associated with discrete, periodical elevations of cortisol (Björntorp and Rosmond, 2000). Prolonged HPA axis stimulation is followed by a continuous degradation of the mechanisms controlling eating and affect. The net effects of neuroendocrine–endocrine perturbations in the HPA axis are insulin resistance and accumulation of body fat. These are effects of cortisol combined with diminished secretion of growth and sex hormone secretions. The outcome of these changes is hypothalamic arousal and metabolic syndrome. The feedback regulation of the HPA axis has a key position in this chain of events with control being mediated by glucocorticoid receptors (Björntorp and Rosmond, 2000).

The impact of negative affect, whether in the form of anxiety, depression or stress, is modulated by the PFC that appraises, evaluates, interprets and monitors the self and the outside world, including responses to the current appearance of the body. A person's body dissatisfaction is at once a cognitive and affective product based on cognitive appraisal and autochthonous perception of the body's attributes and one's feelings about these. In response, the HPA axis produces glucocorticoids that regulate the homeostasis of consumption.

In addition to the mediation of stress responses by the HPA axis, recent studies have observed that there is an alternative system for mediation of stress responses in circulating ghrelin, a peptide hormone, acting upon the amygdala (Meyer et al., 2014). We return to the role of ghrelin later in this article.

Based on the above review of evidence, a provisional description of the neurobiological substrate of the COD is summarized in Figure 3. The model shows feedback loops between the prefrontal cortex, the amygdala, the HPA axis and visceral adiposity as mediators of body dissatisfaction, negative affect, eating behaviour and obesity, respectively.

The evidence from neurobiology suggests that the homeostasis of eating can be overridden by the hedonic reward system acting to relieve stress through the excessive consumption of palatable foods (Figure 4). Furthermore, eating is controlled by a complex neural network including the mesocorticolimbic pathway, which consists of the ventral tegmental area, NAc, amygdala, hippocampus and PFC. These regions are the neural substrates of mood, pleasure, desire, experience of self, body satisfaction and self recognition and have a significant influence on eating patterns and can generate excessive eating. The hedonic system overrides and disrupts homeostatic control when there is chronic negative affect and accessibility to palatable energy-dense foods.

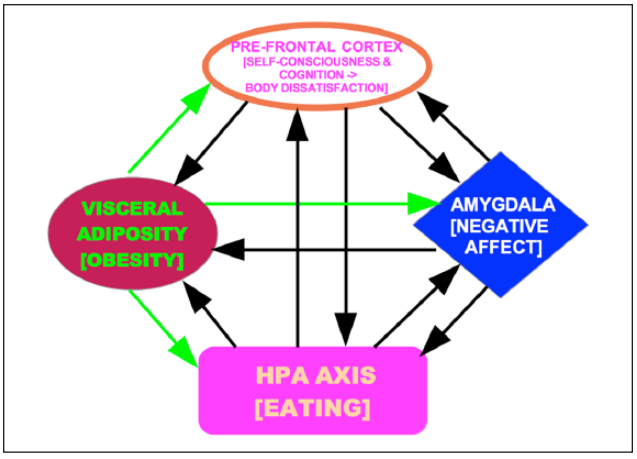

Figure 3. Model of the neurobiological basis of the Circle of Discontent.
Disruption of homeostasis in feedback loops between self-consciousness/cognition mediated by the prefrontal cortex, negative affect mediated by ghrelin signalling in amygdala, eating behaviour mediated by the hypothalamic–pituitary–adrenal (HPA) axis and increased visceral adiposity leading to increased ghrelin secretion. Ghrelin is a significant part of the signalling system throughout the systems of hedonic reward and homeostatic control of appetitive behaviour.

In obese people, eating to excess is produced by a COD, a difficult-to-control self-medication of hedonic reward to assuage stress, anxiety and depression which is paralleled by, but not identical to, the use of nicotine, alcohol and drugs among addicted users.

Entering and exiting the COD

A key question concerns entry and exit to the COD (DiClemente and Delahanty, 2016). Who enters the Circle for the first time, who stays and who leaves, and is it a revolving door? What are the prospects, once inside the Circle, of making positive change?

Important work on the issue of change in health-related behaviours has been carried out by the DiClemente group (DiClemente, 2003; DiClemente et al., 2015). If the theory is to have true explanatory value, these issues need to be addressed by the homeostasis theory. As stated by DiClemente and Delahanty (2016),

> The challenge is to understand how early problems in attachment may influence some to overeating or anorexia, others to sociopathy and drug abuse, others to depression or anxiety, and still others to being successful professionals. It depends on how the experiences, environment, knowledge, and opportunities filter the early experiences and influence movement forward in the process of change for these different outcomes.

The Homeostasis Theory of Obesity describes two main systems, the COD and the Motivation and Energy Mobilization (MEM) system (Figure 5). In the COD, the

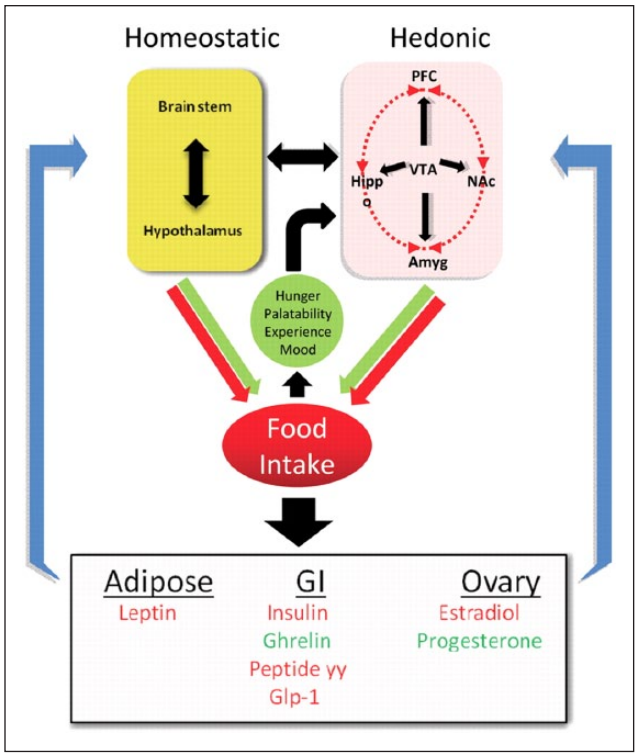

Figure 4. Interaction of homeostatic and hedonic control of food intake.
Hormones from adipose tissue, the gastrointestinal tract and, in females, the ovary influence food intake through hypothalamic and extra-hypothalamic sites of action. Activation of the corticolimbic pathway, which includes the ventral tegmental area (VTA), nucleus accumbens (NAc), prefrontal cortex (PFC), hippocampus (Hippo) and amygdala (Amyg), is associated with the hedonic pleasure of eating and is influenced by hunger level, food palatability, experience and mood. An alternative version would apply to men. Reproduced from Van Vugt (2010) with permission.

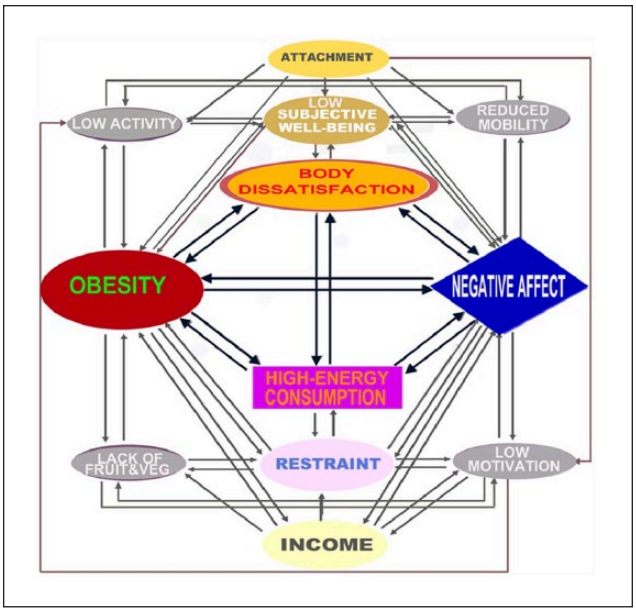

Figure 5. Obesity dyshomeostasis: causing overweight and obesity, feedback loops required for equilibrium become disrupted by the hedonic reward system.
In one system, the Circle of Discontent, the levels of body dissatisfaction, negative affect and high-energy consumption run out of control. Another system provides homeostasis for Motivation and Energy Mobilization (MEM). In the MEM system, lowered motivation causes reduced restraint, increased dietary intake and lowered activity, which cause reductions to subjective well-being, mobility, and positive affect. The whole complex establishes unhealthy eating habits, low activity levels, negative affect, overweight and obesity. Early lifetime stress which interferes with attachment can influence appetite, feeding behaviour and metabolism throughout life. People living with low levels of income will also be at high risk of negative affect, hedonic reward eating of fatty and sugary foods and, ultimately, obesity. Reproduced from Marks (2015).

levels of body dissatisfaction, negative affect and high-energy consumption run out of control. A key connection in the Circle is between chronic stress and comfort eating (Dallman et al., 2003). In the MEM system, lowered motivation causes changes in restraint, dietary intake and activity, which cause reductions to subjective well-being, mobility and positive affect. The whole complex establishes unhealthy eating habits, low activity levels, negative affect, overweight and obesity.

It is commonly assumed in psychology that obesity is caused primarily by 'lifestyle changes'. This assumption, however, is not borne out by the evidence. Many drivers push a person towards entry of the Circle. We can use the analogy of a lottery in which people are allocated tickets. For the most part, tickets are allocated at different critical period in the life cycle starting from the moment of conception. The tickets carry percentage points according to their importance as obesity determinants. At any time, a person's body mass index (BMI) is linearly related to the total number of 'obesity points' they have been allotted. A schematic plan of obesity determinants is shown in Table 1.

The prenatal period and adolescence present critical periods for the development of obesity that persists into adulthood (Dietz, 1994). Genetic predisposition, epigenetic factors and prenatal maternal stress, including problems with the partner (Sominsky and Spencer, 2014; Talge et al., 2007) all have an influence. Socioeconomic disadvantage in the form of poverty causes chronic life stress at all stages from infancy and adolescence through to adulthood. People living with low levels of income suffer social oppression, chronic stress and multiple episodes of negative affect, restraint and hedonic reward eating of fatty and sugary foods leading to obesity (Drewnowski and Specter, 2004; McLaren, 2007; Monteiro et al., 2004). Early life stress including general parenting, parent childhood abuse and attachment style influence appetite, feeding behaviour and metabolism throughout life (Felitti, 1993; Felitti et al., 1998; Puig et al., 2013; Sleddens et al., 2011; Sominsky and Spencer, 2014).

The field of epigenetics and obesity is relatively new but early steps are being made in identifying biomarkers for obesity. Findings suggest that several epigenetic marks are

Table 1. Determination of obesity at the main entry points to the Circle of Discontent.

Period	Evidence	Estimated percentage points	Accumulated percentage
Genetic predisposition	Maes et al. (1997)	50	50
Epigenesis	Van Dijk et al. (2015)	15 (?)	65
First few months of life	Reilly et al. (2005) and Taveras et al. (2009)	15 (?)	80
Adolescence	Guo et al. (2002) and Gordon-Larsen et al. (2004)	10	90
Adult lifestyle	Multiple	10	100

Individual obesity risk accumulates over time with exposure to different periods of obesity risk. Percentage points can be allocated to each critical period to reflect its weighting as a determinant of obesity over an individual's lifetime. By the end of adolescence, the pathway to obesity has been 90 per cent determined. Only the remaining 10 per cent of the variance can be controlled by individual lifestyle components (diet, exercise).

modifiable not only by changing the exposure in utero but also by lifestyle changes in adult life, so there is the potential for interventions to reform unfavourable epigenomic profiles (Van Dijk et al., 2015).

Genetic and neurobiological factors help to explain why many people develop obesity while others head towards other consumption-related conditions such as alcoholism, nicotine or drug addiction. Once again, ghrelin helps to tell the story. Ghrelin levels in children with Prader Willi syndrome are 3- to 4-times higher compared with BMI-matched obese controls (Haqq et al., 2003). Ghrelin shows wide differences between obese and normal weight adults (Tschop et al., 2001) and among adolescents with different kinds of disorders such as anorexia nervosa and obesity. Baseline ghrelin concentrations increase and decrease, respectively, to a mixed meal in anorexic and obese female adolescents (Stock et al., 2005). Low plasma ghrelin has been independently associated with type 2 diabetes, insulin concentration, insulin resistance and elevated blood pressure (BP) (Pöykkö et al., 2003). Ghrelin levels were also found to be significantly higher in female alcohol-dependent patients than in controls, but not in male alcoholics (Wurst et al., 2007). A review of the burgeoning literature on psychoendocrinological correlates of different COD pathways across gender and population groups will be published elsewhere.

Once inside the Circle, is there any escape? As we have seen in Table 1, the majority of available 'tickets' for the obesity 'lottery' are allocated by the time a person reaches adulthood. Obesity is 90 per cent determined before early adulthood with only limited scope for change. The obesity die has been cast. If we allow for the possibility that around half of epigenetic influences on obesity might be reversible and a further 10 per cent for potentially reversible lifestyle determinants, we conclude that 80–90 per cent of obesity determinants are irreversible by treatment.

The COD is a vicious, self-sustaining one. Exit options are few. Breaking out of the vicious circle requires momentously strong motivation and a transformational change in eating habits, lifestyle and philosophy of living. Obesity is a persistent condition that is intractable to treatment. A modest average weight loss of 2–4 kg may be achievable by committed adherence to a structured diet regime (Dansinger et al., 2005) but diet systems generally do not provide the keys to a cure (Foster et al., 2003; Katz, 2002). Psychological therapies lead to disappointing outcomes, with cognitive behaviour therapy producing the highest weight loss of a few kilograms (Shaw et al., 2005). Drug treatments involve safety issues and also yield relatively low levels of weight loss. Weight loss relative to placebo ranges from 3 per cent for orlistat and lorcaserin to 9 per cent for phentermine plus topiramate-Extended Release at 1 year (Yanovski and Yanovski, 2014). The only effective treatment for long-term weight loss in patients with clinical obesity is surgery, which is costly and inaccessible for the majority of patients (Buchwald et al., 2004).

Within current knowledge, the determinants of obesity are practically irreversible; the illness is persistent and it is almost untreatable. To give any other impression to patients is unethical and misleading. A person inside the COD is likely to remain inside. The most likely exit point will be an early death. To continue to offer treatments that are known to be minimally effective and, quite possibly, harmful to physical or mental health is unethical. Until new, fully authenticated endocrinological treatments become available, all necessary resources should be directed towards prevention.

The reach of psychological homeostasis

Looking beyond the topic of obesity, evidence of dyshomeostasis occurs in several fields of health psychology and behavioural medicine. In general, these fields suffer from a theoretical vacuum. I discuss here two specific areas where dyshomeostasis is a prominent feature, addictions and body diversity.

Addictions

In obesity, it has been argued above that hedonistic reward plays a significant disrupting function in weight homeostasis. Many authors have pointed out that food and drug rewards share some common neural substrates, with opioid

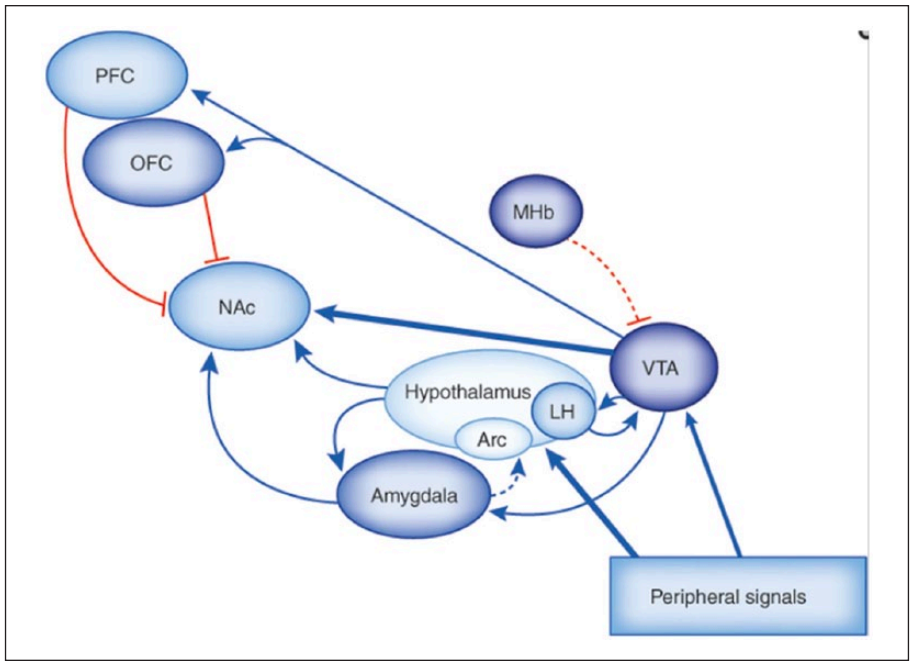

Figure 6. Areas of the brain mediating food intake and drug seeking.
Areas that are most critical for food intake are depicted in lighter shades and those areas most critical for drug reward and seeking are depicted in darker shades. Most areas have some influence on both food and drug intake, and the spectrum represents this overlap. The hypothalamus is critical for food intake and is modulated by the darker shaded areas. The ventral tegmental area (VTA) and nucleus accumbens (NAc) are critical for drug seeking and are modulated by many other brain areas. Inputs from cortex and amygdala provide control over both food- and drug-related behaviours. Arc: arcuate nucleus; LH: lateral hypothalamus; MHb: medial habenula. Red lines: inhibitory connections; dashed lines: indirect projections; thicker lines: stronger connections. Reproduced from DiLeone et al. (2012) with permission.

receptors playing a role in both feeding and reward (Kelley et al., 2005). Saper et al. (2002) stated,

> endogenous opioid systems regulate the hedonic value of food intake independently from the ongoing metabolic needs of the individual. Furthermore, food deprivation, which enhances the hedonic response to food, also increases the motivational value of non-food rewards, such as psychostimulants … intracranial self-stimulation … and heroin intake.

This perspective places food and addictive drugs such as nicotine and heroin into a similar category. However, while there are definitely similarities, reviews of the comparison and distinctions between mechanisms of food reward and drug addiction also indicate major differences between the two types of consumption (DiLeone et al., 2012). While eating is necessary for survival and susceptible to selection pressures during evolution, drug addiction begins as a voluntary choice and is seen as having 'piggybacked' onto pre-evolved reward pathways, engaging a subset of the circuits required for feeding (Figure 6).

The COD has relevance to a variety of conditions that are marked by compulsivity such as the addictions to tobacco, alcohol, illicit drugs and behaviours such as gambling and internet gaming. These habits/addictions involve compulsion and loss of control which can be costly to the individuals concerned in both health and monetary terms; all have been associated with chronic stress and negative affect in the form of anger, anxiety or depression (Breslau et al., 1993; Helzer and Pryzbeck, 1988; Patton et al., 1996, 1998; Swendsen et al., 1998). Diverse consumption patterns across different population groups prove that 'no size that fits all' but the causal mechanisms remain essentially the same.

Excessive consumption is a hedonic strategy to increase reward and reinforces habitual behaviour by reducing negative affect and dissatisfaction. Alcohol, drugs, gambling, gaming, shopping, Internet use, TV viewing, sports, fitness training, running, swimming, tanning and sex are all activities that have been said to be addictive or habit forming by one authority or another. It will suffice here to consider tobacco addiction.

Smoking a cigarette is a homeostatic behaviour that corrects the imbalance of the dopaminergic reward system at the biochemical and physiological levels and reduces dissatisfaction and negative affect. The different kinds of homeostasis complement each other to stabilize physiological and psychological well-being. There are many examples of unhealthy habits being strengthened by hedonic reward and palliation of negative affect in COD.

Nicotine addiction is the result of neurochemical changes to the brain. Long-term tobacco use results in

physical dependence and a compulsion to use tobacco. The cigarette is the most efficient and rapid method for delivering nicotine to the brain. Nicotine from cigarette smoke is quickly absorbed in the lungs and then rapidly passes into the brain where it binds to specialized nicotinic acetylcholine receptors (nAChRs). Stimulation of nAChRs by nicotine results in the release of a variety of neurotransmitters in the brain, of which dopamine is the most important because it produces pleasure. In an addicted smoker, nicotine therefore produces pleasure, arousal and mood modulation. However, the effects of a single cigarette are short lived, and the smoker requires frequent top-ups of nicotine to maintain a state of cognitive and affective stability. For the addicted smoker, smoking is a homeostatic process which maintains the required level of nicotine in the brain (Prochaska and Benowitz, 2016).

With chronic nicotine addiction, tolerance develops so that more nicotine is required to deliver the same neurochemical effect. Nicotine is needed to maintain normal brain functioning, and stopping smoking, or leaving a longer interval between smokes, is associated with withdrawal symptoms of irritability, anxiety, poor concentration, hunger, weight gain and problems getting along with others. Nicotine addiction is therefore a 'two-edged sword' that is sustained both by positive effects of pleasure and arousal and by the avoidance of the unpleasant effects of nicotine withdrawal. Conditioning sustains tobacco use through the reinforced association between smoking and 'triggers' in the form of specific behaviours such as drinking coffee or alcohol, talking on the phone, driving a car and/or completing a meal. Sensorimotor trigger factors associated with the act of smoking, for example, the smell, taste and feel of the cigarette smoke become cues for smoking and maintain tobacco use (Sulzberger and Marks, 1977).

In the formation of tobacco addiction, the novice inhales tobacco smoke which, in the early stages, gives a toxic and unpleasant sensation in the mouth and throat. However, with each successive inhalation, the unpleasant sensations in the throat and mouth are replaced by feelings of satisfaction as the smoker strengthens the habit. The feelings of satisfaction grow stronger as the habit is reinforced by the sensation of pleasure and the reduction in negative affect. As the habit strength increases and the addiction is established, the smoker feels withdrawal symptoms that increase in intensity the longer he/she waits before lighting the next cigarette. Symptoms of addiction appear within days or weeks after occasional smoking first begins (Russell, 1990).

The smoker is effectively using cigarette smoking as a form of mood control, as self-medication, titrating the dose to match momentary mood. Smokers are able to regulate smoke and nicotine intake on a puff-by-puff basis, an aspect of smoking control that is acquired early in the tobacco-dependence process (Collins et al., 2010). For this reason, smokers report that cigarettes help relieve their feelings of stress (Figure 7).

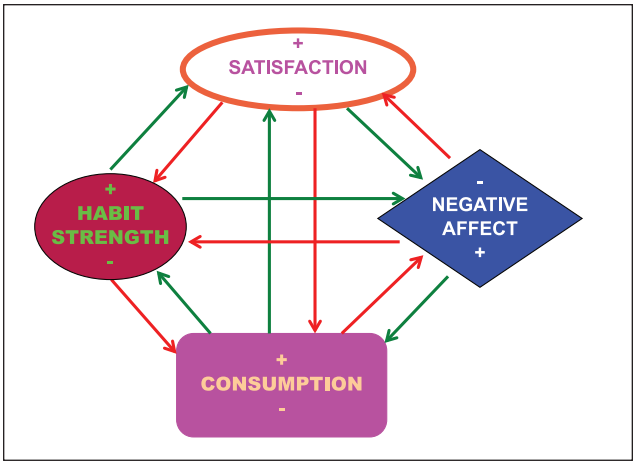

Figure 7. The Circle of Discontent in addiction: homeostatic reduction of negative affect and low satisfaction induce increased consumption, which increases habit strength through positive reinforcement by hedonic reward and negative reinforcement from palliation of negative affect.

Contrary to the subjective experience of smokers, smokers' stress levels are higher than those of nonsmokers, and adolescent smokers report increasing levels of stress as they develop regular patterns of smoking (Parrott, 1999). Nicotine consumption rapidly increases heart rate and BP (Rose et al., 2001).

Nicotine addiction exacerbates stress yet yields the delusory impression to smokers that it is stress reducing. Thus, the alleged 'relaxation effect' of smoking is a consequence of reversing the tension and irritability that develop during nicotine depletion between cigarettes. Addicted smokers need nicotine to feel normal (Parrott, 1999). Unpleasant withdrawal symptoms are often associated with increases in urges and intentions to take drugs. In addition, addicted individuals rate coping with negative affect as the prepotent motive for drug use (Baker et al., 2004). The act of smoking cessation leads to reduced stress.

One potential mechanism of nicotine addiction is increased dopamine transmission, which gives a feeling of pleasure or satisfaction. The increase in dopamine activity from nicotine results in pleasant feelings of satisfaction for the smoker, but the subsequent decrease in dopamine leaves the smoker craving for more cigarettes (Arias-Carrión et al., 2010; Gamberino and Gold, 1999).

Negative affect influences how much an individual tends to consume, whether it is food, smoking, alcohol, other drugs or behaviours and how intensely one craves and, ultimately, whether an abstinent individual will return to harmful consumption. Chronic consumption of alcohol alters the normal function of the affect system causing an increased susceptibility to stress (Adinoff et al., 1998). This increases the likelihood of progression as it produces a cycle of degeneration where exposure to stress leads to escalations in consumption, further reducing the ability to cope

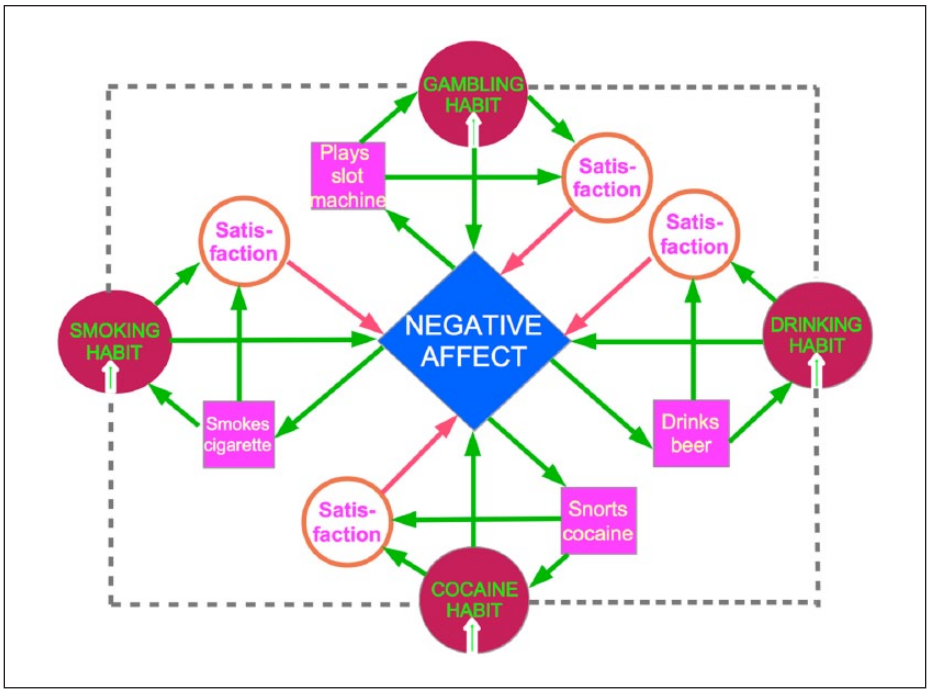

Figure 8. Multiple Circle of Discontent: a person addicted to nicotine, ethanol, cocaine and gambling.
This diagram shows a model of four concurrent addictions, each having a homeostatic system and Circle of Discontent. The green arrows indicate facilitation; the red arrows indicate temporary palliation of negative affect. The grey lines indicate associative connections between habits where one acts as a trigger for the other. The brain areas mediating appetitive drug seeking and addictive behaviours may differ between addictions, but will include the areas shown in the previous Figure 6. The four addictions reinforce one other and the four homeostatic systems become closed off from external influence. The hedonic reward system serves to palliate negative affect by appetitive behaviours and controls all four of the addictions in an identical manner. The hedonic reward system overrides homeostasis, ultimately putting the individual at significant risk of different catastrophes including life-limiting conditions, mental illness, crime and bankruptcy. Not all pathways are included to avoid cluttering the diagram. The same person could well have several other addictions (e.g. caffeine, licit or illicit drugs, gaming, sex and the Internet) and the model could be extended to include them.

with stress and shortening the length of intervals between periods of abstinence.

Many individuals in the population have multiple addictions (Anthony et al., 1994; Lorains et al., 2011). In such individuals, multiple COD operate in complementary fashion. Figure 8 illustrates a model of a person who is addicted to nicotine, ethanol, cocaine and gambling. The four concurrent addictions each has its own homeostatic system and COD. The same person could quite possibly have other addictions as well (e.g. to caffeine, other drugs and the Internet), and the already complex diagram would need to be extended to include these. The different addictions have associative connections and any one of the behaviours may act as a trigger for one or more of the others. The brain areas mediating appetitive drug seeking and addictive behaviours may differ between addictions, but include at least some of the areas shown in Figure 5. The four addictions reinforce one other and, after prolonged exposure, the addictions become closed off from external influence and compulsive in nature (Vanderschuren and Everitt, 2004; Volkow and Fowler, 2000). The total system becomes self-sustaining with all the addictions under the control of a single hedonic reward system designed to palliate negative affect by repeated appetitive behaviours. As previously mentioned, the peptide ghrelin activates reward systems, and its receptors (GHS-R1a and R1b) appear to be required for alcohol, cocaine, amphetamine and nicotine-induced reward (Jerlhag and Engel, 2011). The hedonic reward system, under the influence of ghrelin, overrides the normal functioning of homeostasis, maintaining the COD and placing the individual at significant long-term risk.

Diverse bodies

In discussing obesity stigmatization, Roosen and Mills (2016) suggest the need for a cultural shift 'not only to reduce thin valorization but also to promote social acceptance of diverse bodies, including bodies that are traditionally understood as unattractive, unhealthy, and unproductive (i.e. disabled and/or obese)'. Roosen and Mills (2016) suggest that this cultural shift is already underway, valorizing a 'fit' ideal body instead of a thin or muscular one.

In line with this perspective, homeostasis and dyshomeostasis are evident in a diverse array of life circumstances and conditions (see Table 2). Behavioural homeostasis occurs in

Table 2. The Circle of Discontent for diverse conditions.

Issue	Evidence of causal link to depression	Evidence of causal link to dissatisfaction	Evidence of homeostasis
Disfigurement	Thompson and Kent (2001)	Rumsey and Harcourt (2004) and Grogan (2006)	Rumsey and Harcourt (2004) Camouflaging
Gigantism	Whitehead et al. (1982) and Kuo et al. (2013)	Woodhouse et al. (2006)	Hurxthal (1961) Self-imposed starvation diet in a young girl
Dwarfism	Obuchowski et al. (1970)	Bjork et al. (1989)	Backstrom (2012) Positive identity advocacy
Leprosy	Tsutsumi et al. (2004)	Tsutsumi et al. (2004)	Heijnders and Van Der Meij (2006) Stigma reduction/empowerment
Amputation	Horgan and MacLachlan (2004)	Remes et al. (2010)	Gallagher and MacLachlan (1999) Wearing a prosthetic limb
Spinal cord injury	Elliott and Frank (1996)	Dijkers (1997) Kennedy et al. (2006)	Kennedy et al. (2006) Social integration

For each issue, the table lists publications providing evidence of causal links to negative affect (column 2), to dissatisfaction (column 3) and evidence of homeostasis in the form of self-medication, compensatory actions, coping or psychological adaptations (column 4).

a variety of ways including coping strategies, compensatory actions, life identity projects and an infinite array of sophisticated adaptations to illness, injury and life events. Of key significance to stigmatization is the plain visibility of obesity, gigantism, dwarfism and, in many instances, disfigurement. The degree of stigmatization may be influenced in part by the perceived self-responsibility for the condition. Gigantism, dwarfism and, in many cases, disfigurement are genetic and unavoidable. Obesity is often viewed as controllable, changeable and a matter of personal choice. The social perception that obese people can *choose* to lose weight if they want to, but fail to do so, could explain the relatively strong stigmatization of obese people in modern society (Backstrom, 2012).

Motivation

Pelletier et al. (2016) focus their review on motivation constructs from self-determination theory (SDT; Deci and Ryan, 1985). They suggest that the COD 'does not explain why some people exposed to the same conditions (e.g. abundance of unhealthy foods, negative life events) do not gain weight and become obese' and that it emphasizes strategies that do not consider individuals as active agents of their own behaviours. I clarify here the motivational aspects of my theory.

There can be no question that motivation plays a pivotal role in human behaviour change and in the aetiology of obesity. As previously stated, 'It remains necessary to explain *how* or *why* overweight or obesity can develop in a susceptible individual, and *why some individuals develop it and not others*' (Marks, 2015). The Homeostasis Theory of Health (HTO) holds that human health is regulated at all times by *multiple systems of homeostasis* that are operating in parallel and in cascades all directed towards stability of function. All of the many thousands of homeostatic systems are interconnected and complementary in maintaining the stability of the human organism. I refer the reader to Figure 5. In my earlier article, I concentrated on one only of the many homeostatic systems, the *COD*, a feedback loop that includes Physical Health, Life Satisfaction, Affect and Consumption.

Of equal importance to the COD is the *MEM system.* The MEM system incorporates Motivation, Restraint, Diet, Physical Health, Activity, Subjective Well-Being, Mobility and Affect. As the diagram of Figure 5 shows, the MEM and COD systems are both equally involved in regulating Physical Health and Affect, but only the MEM system includes individual motivation. Without any doubt, the MEM system is of great significance in the maintenance of healthy habits and behaviours and, when things go wrong, in the generation of overweight and obesity.

It is helpful to consider the regulatory styles of SDT that are differentiated along an alleged continuum that ranges from non-self-determined styles (i.e. amotivation, external regulation and introjection) to self-determined ones (i.e. identification, integration and intrinsic motivation). As suggested by Pelletier et al. (2016), there are parallels between SDT concepts concerning motivational style and the HTO. The COD is a perfect fit to the profile of 'Controlled Motivation'[2] within SDT.

Studies by Pelletier et al. (2004) have provided interesting evidence concerning the style of motivation most likely to be associated with unhealthy eating, depressive symptoms and increased BMI, namely, 'Controlled Motivation'. Pelletier et al. (2004) revealed a response pattern that is consistent with the COD, namely unsuccessful regulation of eating, a concern with quantity but not quality of food eaten, bulimic and depressive symptomatology, low self-esteem and low life satisfaction and increased BMI, all

significantly associated with controlled regulation (Table 4 in Pelletier et al., 2004). On the other hand, autonomous regulation was found to be significantly correlated with a concern for quality rather than quantity of food eaten, with successful regulation of eating, healthy eating behaviours, high self-esteem and high life satisfaction. One could not wish for a more positive confirmation of the theory, although I was unaware of it until Pelletier et al. drew my attention to it.

In this light, the two profiles of Controlled Motivation and Autonomous Motivation represent the opposite ends of the homeostasis continuum. Autonomous Motivation brings satisfactory internalized control of eating behaviours, relatively high life satisfaction and positive affect, a state of positive homeostasis. Controlled Motivation, on the other hand, is a component of homeostatic imbalance in which the individual fails to enjoy, or internalize, the desired goals of eating behaviour (Ryan and Deci, 2006). The COD is perfectly represented by the 'Controlled Regulator', a person whose eating habits are running out of control and whose life satisfaction, and affect levels have deteriorated. In SDT, controlled regulation occurs in three forms:

1. The Introjected Regulator, not wanting to be ashamed of how they look and eat, feeling that they must absolutely be thin, feeling they would be humiliated if they were not in control of their eating behaviours.
2. The External Regulator, other people close to them insist that they do things a certain way, other people close to them will be upset if they do not eat well, people around them will nag them to do it, or it is expected of them.
3. The Amotivated Regulator, the worst case scenario, feeling helpless and hopeless, not really knowing what to do, having the impression that they are wasting their time trying to regulate their eating behaviours, not seeing how their efforts can ever lead to eating healthily or helping to improve their health.

In SDT, motivation is king, with a commanding role in need satisfaction for autonomy, competence and relatedness (Ryan and Deci, 2000). In the HTO, motivation is more a courtier than a king, but a key player, nevertheless, in the MEM system. From the perspective of HTO, the role of motivation in real behaviour change should be evaluated on the basis of the hard-won findings of systematic reviews and meta-analyses. Ng et al.'s (2012) meta-analysis of SDT studies in health care found only low correlations: between autonomous self-regulation and mental and physical health of .06 and .11, respectively; between controlled regulation and mental and physical health of −.19 and .09, respectively; and between amotivation and mental and physical health of −.05 and −.15, respectively. These findings suggest that motivational style controls, at most, 3–4 per cent of variance in mental and physical health.

These modest empirical associations between SDT constructs and health outcomes may, in part, be explained by methodological problems concerning the scoring of self-determination motivation. The validity of the assumed self-determination continuum, forming the basis of the measures employed, has not been supported by robust state-of-the-art psychometric analyses. In a Rasch analysis of the continuum concept, Chemolli and Gagne (2014) found strong evidence of a multidimensional factor structure rather than evidence of a continuum. This significant issue places a serious limitation on the use of SDT in prevention of obesity. Until these methodological issues are solved, the status of SDT remains uncertain and unclear. Unless psychological theories and interventions can be cashed out in objective benefits to health outcomes, they tend to lead only to false hopes and disappointment.

A randomized controlled trial (RCT) with SDT-based exercise motivation variables evaluated a behavioural weight control intervention on 3-year weight change (Silva et al., 2011). The 1-year SDT-based intervention was immediately followed up and then again 2 years later with 221 female participants. The intervention group attended 30 sessions, targeted at increasing PA and energy expenditure, adopting a diet consistent with a moderate energy deficit and integrating exercise and eating patterns that would support weight maintenance. The control group received 29 sessions of general health education on the basis of several educational courses covering various topics, for example, preventive nutrition, stress management, self-care and effective communication skills.

Treatment had significant effects on 1- and 2-year autonomous regulations, 2-year PA and 3-year weight change. Average weight loss at 12 months was −7.29 per cent versus −1.74 per cent in the control group, but the intervention effect tapered off over time showing only −3.9 per cent versus −1.9 per cent in the control at 36 months. The intervention produced a 2.0 per cent greater average weight loss at 36 months than the control condition. Autonomous style motivation correlated −.31 with 3-year weight change, explaining only 10 per cent of the variance in weight change.

Unfortunately, the abstract, theoretical importance of motivation in SDT has not yet been established in the form of concrete health outcomes. The role of individual motivation appears to be a quite modest one, one process within a complex system, as explicated in the HTO.

Upstream versus downstream intervention

To have any significant impact on the obesity epidemic, effective interventions must be delivered. Any long-term strategy to curtail the obesity epidemic needs to be based on

effectiveness and cost-effectiveness. In this regard, upstream interventions (primary prevention) have been shown to be more effective and more cost-effective than downstream (secondary prevention) ones. A recent economic analysis of the obesity epidemic concluded:

> Education and personal responsibility are critical elements of any program to reduce obesity, but not sufficient on their own. Additional interventions are needed that rely less on conscious choices by individuals and more on changes to the environment and societal norms. (Dobbs et al., 2014)

There are 1 billion plus sufferers alive today. The infrastructure required for individual-level psychological interventions on a universal basis for these 1 billion people far outstrips available resources. To make any real impact on the obesity epidemic, it is essential to combine prevention efforts with individuals with upstream policies to change the context that is currently promoting the spread of obesity at all levels of society.

Pelletier et al. (2016) argue that 'Environmental changes … may be slow to implement, can be very expensive, and could be stalled by industries with competing interests'. However, to give just two examples, environmental changes in the form of regulations on sugar or advertizing can generate significant revenue. Both the sugar-sweetened beverage excise tax and the elimination of the tax subsidy for advertising unhealthy food to children would lead to substantial yearly tax revenues (US$12.5 billion and US$80 million, respectively; see Gortmaker et al., 2015b). The analyses of Gortmaker et al. (2015a, 2015b) have shown that the cost effectiveness of these preventive interventions is greater than that obtained from published clinical interventions to treat obesity. Individual approaches using social cognition models have been tried and tested for many years, and outcomes have been disappointing (Marks et al., 2015). Lehnert et al. (2012) reviewed long-term economic findings (at least 40 years) for 41 obesity prevention interventions. Interventions were grouped according to their method of delivery, setting and risk factors targeted into behavioural ($n=21$), community ($n=12$) and environmental interventions (n=8). Interventions that modified a target population's environment, that is, fiscal and regulatory measures, reported the most favourable cost-effectiveness. There can be little doubt that obesity prevention requires the use of cost-effective interventions at all levels of society.

For the 1 billion plus individuals living today with obesity, these words will not be very welcome. But it is better to face the truth than live in a dream world with impossible hopes and expectations. For the vast majority of obese people alive today, there will be no significant reversal. Current treatments are disappointingly weak, expensive, and, often, have unwanted side effects, especially drugs and surgery (Marks et al., 2015). The only way forward that makes sense is prevention – to prevent new cases, as many as possible. The emphasis should be placed on upstream approaches, preventing new floods of cases before they arrive at the point of no return.

Spirituality homeostasis?

Piko and Brassai (2015) make a case for *spiritual balance* as a form of homeostasis. They contend, correctly I believe, that existential attitudes are closely related to 'identity formation, moral development, value-related attitudes, personal goals and lifestyle choices'. Having meaning in life encourages engagement in health-promoting behaviours and the avoidance of health-risking behaviours, such as obesity and eating disorders. Along with physical, cultural, psychosocial and economic needs, a definition of health can also include spiritual needs, not simply the absence of illness (Marks et al., 2015: 5).

Piko and Brassai (2016) discuss the meaning-making model of Park (2010), which proposes that people's perceptions may contribute to content/discontent with life, body and the world. Park (2013) states,

> According to the Meaning Making Model, the degree to which one perceives one's illness as discrepant from one's global beliefs, such as those regarding identity (e.g., I live a healthy life style) and health (e.g., living a healthy lifestyle protects people from illness), and global goals (e.g., desire to live a long time with robust health) determines the extent to which the illness is distressing. (p. 43)

The Meaning Making Model of Park (2013) assumes that a discrepancy between global beliefs and identity produces distress. In some cases, those beliefs are spiritual in nature. However, primary sources of research on spirituality are not generally supportive of the model proposed by Park (2013).

The central role of meaning and purpose in life was previously advocated by Viktor E Frankl (1959) and, later, in the Salutogenic Theory of Aaron Antonovsky (1979, 1987). Neither Frankl's (1959) study nor Antonovsy's theory of salutogenesis is discussed by Park (2010, 2013). We must never forget what Frankl (1959) said about the prisoners living in concentration camps: 'Every man was controlled by one thought only: to keep himself alive for the family waiting for him at home, and to save his friends'. In describing the inmates' dream life, he stated, 'What did the prisoner dream about most frequently? Of bread, cake, cigarettes, and nice warm baths. The lack of having these simple desires satisfied led him to seek wish-fulfilment in dreams'. In another place, Frankl describes his ultimate realization that it is love that satisfies a person's needs for meaning:

> A thought transfixed me: for the first time in my life I saw the truth as it is set into song by so many poets, proclaimed as the final wisdom by so many thinkers. The truth – that love is the ultimate

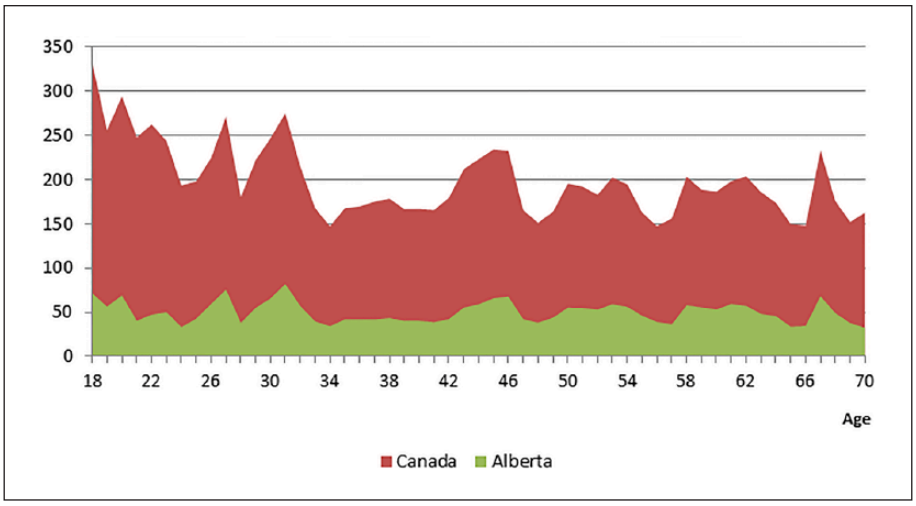

Figure 9. Life course projections of avoided health care costs for Canada and the province of Alberta (in million dollars) in light of a school-based obesity prevention programme (reproduced from Tran et al. (2014), Figure 6).

> and the highest goal to which man can aspire. Then I grasped the meaning of the greatest secret that human poetry and human thought and belief have to impart: The salvation of man is through love and in love. I understood how a man who has nothing left in this world still may know bliss, be it only for a brief moment, in the contemplation of his beloved … 'Set me like a seal upon thy heart, love is as strong as death'.

No mention is made in Frankl's account of the search for meaning to find spirituality. Frankl (1959) asserted what he called 'the will to meaning': man's search for meaning as the primary motivation in his life.

The HTO is a particular case of the General Theory of Well-Being, which posits causal reciprocal relationships between subjective well-being and life satisfaction (Marks, 2015; Marks et al., 2015). Empirical studies suggest the existence of a strong and stable relationship between meaning in life and subjective well-being (Zika and Chamberlain, 1992). People who experience their lives as meaningful tend to be more optimistic and self-actualized (Compton et al., 1996), experience more self-esteem (Steger et al., 2006) and positive affect (King et al., 2006), as well as suffering less depression and anxiety (Steger et al., 2006) and less suicidal ideation (Harlow et al., 1986). The Salutogenic Theory of Antonovsky emphasized the relationship between meaning, purpose in life and positive health outcomes (Eriksson and Lindström, 2006).

For many people, spiritual experience is a source of great meaning to their lives. However, spiritual beliefs and experiences are far from universal. To quote one statistic, in the region of 500–750 million people worldwide have no religious or spiritual beliefs and are living as declared atheists (Zuckerman, 2009). In homeostasis, the organism actively strives to reduce the discrepancy between an optimum level of a quantity or quality and its current state. While many people certainly strive for meaning and may feel that they lead 'empty lives', there is no evidence of an optimum level or a homeostatic mechanism for spirituality.

Issues requiring more research

Homeostasis theory proposes that weight gain is fostered by a COD consisting of body dissatisfaction, negative affect and overconsumption. Drawing on this framework, Annunziato and Grossman (2016) describe research in two domains, victim blaming and devalorizing the thin ideal. They suggest that university-based clinical health psychologists are uniquely positioned to implement large-scale approaches that have shown promise in addressing core issues in the HTO. Annunziato and Grossman cite examples of research that include a 'Social and Emotional Learning' curriculum in Sweden that demonstrated decreases in victimization (Kimber et al., 2008) and the 'Body Project' which produced reductions in eating disorders (Stice et al., 2013), in thin-ideal internalization, body image dissatisfaction and negative affect in female students (Stice et al., 2011) and an Internet-based programme that demonstrated large weight gain prevention effects (Stice et al., 2014). Annunziato and Grossman (2016) propose more extensive use of both systemic and individual interventions with adolescents and young adults in school settings. For example, large-scale programmes in secondary schools and universities could be designed to bring about culture change.

A school-based programme is described by Tran et al. (2014) based in the Canadian province of Alberta. Fung et al. (2012) demonstrated the feasibility and effectiveness of a school-based programme in preventing childhood obesity, the Alberta Project Promoting active Living and healthy Eating in Schools (APPLE Schools). The

intervention involved a full-time School Health Facilitator in each of 10 schools for implementing healthy eating and active living policies, practices and strategies while engaging stakeholders, including parents, staff and the community. The facilitators contributed to the schools' health curriculum and organized activities such as cooking clubs and healthy breakfast, lunch and snack programmes, after school PA programmes, walk-to-school days, community gardens, weekend events and circulated newsletters. By 2010, the students' eating habits and PA levels at APPLE Schools had improved significantly while obesity prevalence had declined relative to their peers attending other Albertan schools (Figure 9). Other comprehensive school-based programmes have achieved similarly positive results (Greening et al., 2011; Khambalia et al., 2012; Verstraeten et al., 2012; Veugelers and Fitzgerald, 2005). Ideally, education about and training of healthy eating habits and regular PA will become a part of every school curriculum universally.

Markey et al. (2016) referred to the role of social relationships in eating patterns and romantic partners who appear to be especially important and an understudied factor in eating behaviours, body image, and obesity risk. In line with Markey et al.'s (2016) ideas, the influence of the quality of marital relationships in appetite regulation was investigated in a double-blind randomized crossover trial (Jaremka et al., 2015). Both members in 43 couples ate a standardized meal at the beginning of two visits. Observational recordings of marital conflict were employed to assess marital distress. Ghrelin and leptin were sampled premeal and postmeal at 2, 4 and 7 hours. People in more distressed marriages were found to have higher postmeal ghrelin and a poorer quality diet than those in less distressed marriages, but only among participants with lower BMI. Ghrelin and diet quality therefore may be the links between marital distress and its negative health effects (Jaremka et al., 2015).

Children growing up in a disharmonious environment, whether the disharmony is caused by socioeconomic disadvantage or other factors, are exposed to parental frustrations, relationship discord, a lack of support and cohesion, negative belief systems, unmet emotional needs and general insecurity. These stressful experiences increase the risk of psychological and emotional distress, including low self-esteem and self-worth, negative emotions, negative self-belief, powerlessness, depression, anxiety, insecurity and a heightened sensitivity to stress (Hemmingsson, 2014).

Brindal and Wittert (2016) suggest consideration of allostasis, coping style and habituation in addition to the COD model. They argue that the incorporation of these elements into the Homeostatic Theory of Obesity may help to 'expand its explanatory power and associated avenues of intervention'. Furthermore, they suggest that a meaningfully approach to the obesity epidemic and associated chronic disease will require 'policy and regulation as well as targeted behavioural strategies aiming to reduce allostatic load'. However, in this author's opinion, the concept of allostasis does not add anything new to the COD model, which is founded on the concept of homeostasis described by Cannon (1932). The concepts of 'allostasis' and 'allostatic load' appear to be based on a misunderstanding of the original concept of homeostasis, which covers all the functions that the proponents wish to attribute to allostasis (Day, 2005). In addition, the construct of allostasis does not help us to better define stress. I concur with Day (2005), who provided a helpful précis of the 'allostasis theory' as follows: McEwen and Wingfield (2003) wrote:

> '(the term) stress will be used to describe events that are threatening to the individual and which elicit physiological and behavioural responses as part of allostasis *in addition to that imposed by the normal life cycle*' (my italics). They propose, in effect, that stress is just one type of challenge that can activate … allostatic (or, as I prefer, homeostatic) responses. Accordingly, we can summarise their position as follows: life is a series of challenges; some are part of the normal life cycle; some can be described as stressors; all of these challenges must be met, i.e. homeostasis must be maintained; the process of maintaining homeostasis (a process they would refer to as allostasis) involves wear and tear (which they refer to as allostatic load) which can impact adversely on health. This re-statement of McEwen and Wingfield's thesis may seem banal but reading it with the bracketed words eliminated will demonstrate that understanding their thesis does not require the adoption of allostasis terminology. The critical question that remains then is this: does the concept of allostasis help us to better define stress? I suggest that the answer is 'no'. (Day, 2005: 1198)

Conclusion

Homeostasis is an omnipresent process that has been neglected in theoretical psychology. Homeostasis is the primary process for the maintenance of healthy organisms. The breakdown of homeostasis causes disorders including obesity, the addictions and chronic conditions including stress in persons with diverse bodies. All such conditions entail the self-reinforcing activity of a vicious COD. Hedonic reward overrides weight homeostasis to produce OD. A preliminary model suggests that OD is mediated by the PFC, amygdala and HPA axis with signalling by the peptide hormone ghrelin which simultaneously controls feeding, affect and hedonic reward. The totality of evidence within current knowledge suggests that obesity is a persistent, intractable condition. Prevention and treatment efforts targeting sources of dyshomeostasis provide ways of reducing adiposity, ameliorating addiction and raising the quality of life in people suffering chronic stress.

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Notes

1. A similar model, recently published by Hemmingsson (2014), discusses emotional distress in causing obesity:

   > … inner disturbances eventually cause a psycho-emotional overload, triggering a cascade of weight gain-inducing effects including maladaptive coping strategies such as eating to suppress negative emotions, chronic stress, appetite up-regulation, low-grade inflammation and possibly reduced basal metabolism. Over time, this causes obesity, circular causality and further weight gain. (p. 770)

2. In self-determination theory, the term for non-autonomous motivation is 'Controlled Motivation'. Perhaps, a more apposite term would be '*Un*controlled Motivation'.

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