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Editorial

Obesity: A Deadly Consequence of the Modern Addiction to Over Nutrition and Under Exertion

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This editorial discusses the problem of the growing obesity epidemic with a focus on the addictive roots of the disease. Addiction is a chronic, relapsing disorder with three phases, a binge intoxication phase characterized by the rewarding properties of the drug, a withdrawal phase accompanied by a negative emotional state as the rewarding properties of the drug wear off, and a preoccupation and anticipation phase that precedes renewed drug intake. Food can indeed be the "drug of choice" in certain at risk individuals. Palatable food consumption has positive reinforcing effects from inducing pleasure, and negative reinforcing from comforting effects that can change the individual's reaction to stress. Recently the Yale Food Addiction Scale (YFAS) has been introduced as an index of addictive-like eating behaviors that mimic the diagnostic criteria for substance dependence in the DSM-IV-TR. [1,2] Food addictive behavior, in particular to sugar and high caloric palatable food has been demonstrated in human and animal studies [3,4].

One may consider obesity as a consequence of modern-day addictions associated with specific risk factors. As in any addictive disorder, individuals display different susceptibility levels to over nutrition and under exertion. Individual variations in risk of addiction can exist at a neuroendocrine, psychological, or psychosocial level. While some obese patients visiting obesity clinics are inherently at a higher risk for obesity owing to genetics and underlying medical diseases a vast number of our patients are at a higher risk from neuroendocrine hormonal dysregulation, psychological imbalance, and psychosocial stressors that promote disordered eating.

The latest guidelines on obesity suggest dietary interventions, exercise programs, commercial weight loss programs, pharmacological agents, and bariatric surgery as tools in the management of obesity. They mention the use of behavioral strategies to facilitate patients' adherence to diet and activity recommendations. Below we discuss the epidemic of obesity in a different light, as a disease of addiction, associated with stress-related triggers for food intake, increased vulnerability to addictive eating, and disordered neuroendocrine responses and abnormal feedback mechanisms following food intake. Several groups have presented data to indicate that obese people (and animals) still have robust feedback mechanisms for defending body weight and adiposity – but at a higher set-point – and in the obese, the set-point rises with age. Thus, many of the feedback mechanisms must still work. Why the set-point is elevated remains a topic of considerable debate. Evidence supporting the role of neuroendocrine dysregulation affecting appetite and satiety is accumulating. The appetite stimulant hormone ghrelin has been shown to rise in response to a stressor [5]. Exposure to dopamine has been shown to reduce leptin in a dose-related fashion in adipocytes from obese subjects [6].

Patients develop obesity from increased caloric intake over expenditure. This imbalance can result from increased vulnerability to addictive eating behavior and emotional eating. The individual response to stress in relation to food is variable with stress- and cue-induced feeding possibly playing important roles as causes for the obesity. Stressful emotions can trigger excessive eating in some individuals, the emotional overeaters and suppress appetite in others, the emotional under-eaters. Animal models have shown that stress-induced anorexia was associated with reduced standard diet consumption and weight loss. However in the presence of palatable high caloric food, animals consumed an increased proportion of comfort food relative to the standard diet leading to obesity [7]. During a stressful condition, hunger has been associated with greater food intake suggesting that emotional eating may be contributing to increase eating in response to stressors. Stress-induced elevation of hormones, such as cortisol or appetite-stimulating hormones such as ghrelin, promote food intake and may subdue the gratification response from normal caloric intake. A disturbed ghrelin response might be a risk factor for emotional eating and therefore a risk factor obesity [8]. Blocking the activity of ghrelin receptors has been associated with reducing the consumption of both food and drugs of abuse [9]. A type of addictive eating behavior commonly seen among obese patients with emotional eating is the night eating syndrome. It is characterized by increased evening or nocturnal food intake, insomnia, morning anorexia, and depressed mood. Night eating severity has been shown to be associated with frequent binge episodes and higher BMI at high levels of emotional eating [10].

Emotional eating has been under diagnosed and is a culprit in obese patients who do not display other risk factors for obesity. We are still discovering methods to best identify and treat these obese patients with an excessive drive to eat. A recent study used naltrexone-induced cortisol release and nausea to identify individuals who have greater underlying food reward dependence [11]. However why some individuals react differently to triggers such as stress with decreased appetite and anorexia is yet to be fully understood. The mesolimbic dopamine reward system plays a crucial role in the individual's drive to seek out and consume in excess, especially fatty and high caloric food, A history of binge eating on fat and chronic sleep deprivation has been shown to predict a higher vulnerability to addictive behavior in animal models [12]. Stress induced increase in serum corticosterone has been shown to be attenuated when animals had access to comfort food [7]. We postulate possible risk factors for

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obesity include elevated circulating corticosterone levels and blunted dopamine levels [7,12].

Binge eaters are more likely to relapse following bariatric intervention and initial weight loss [13]. These patients are presumably more likely to fail dietary restriction techniques and would benefit from psychological evaluation before dietary restriction or pharmacological intervention [14]. A distorted or possibly adaptive response to stress and other cues for eating should be treated before expensive and irreversible surgical interventions. One may argue that obesity treatments, other than bariatric surgery, have inconsistent outcomes and therefore extensive evaluations to distinguish stress responses before surgery are not warranted. However as we develop new markers for diagnosis and tools for treating this class of obese patients it may soon be a worth wile investment. An investigational technique possibly used in the future is repetitive transcranial magnetic stimulation, a brain stimulation technique used in the treatment of psychoactive substance addiction which has been evaluated in the treatment of bulimia nervosa. Brain stimulation exerts a beneficial effect via the reduction of the level of craving for food [15]. This technique has been successfully utilized in cases of refractory obesity following failed bariatric surgery [16]. Emotional eaters should be treated differently from non-emotional eaters in any stepwise weight-loss program. Obese patients with a strong component of emotional eating, addictive personality traits, night eating disorder, and stress related binge eating should be identified and receive differential treatment. Identifying these patients would be one step closer to conquering the battle against this epidemic of obesity.

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