Addiction and the Eating Disorders

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Although comprehensive theories of addiction recognize the etiological importance of environmental and cognitive factors, it has been widely accepted for many years that addiction is also a brain disease and that individuals differ in their susceptibility to this condition (Leshner, 1997; Wise and Bozarth, 1987). Explanations of the eating disorders have tended to eschew biological models in favor of those that focus on psychosocial and family influences—the most prominent models arising from psychoanalytic, feminist and cultural theory. It is not surprising, therefore, that although clear parallels exist between the abuse of substances and disturbances in eating, there has been a reluctance to accept that the two may share a common etiology. It is also probable that their similarities were obscured by dramatic differences in the social profile of the stereotypic drug addict and the patient with an eating disorder—the former typically associated with male criminality and social deviance and the latter with female submissiveness and social conformity.

In the past decade, however, there has been a growing paradigmatic shift in eating disorder research, with a movement away from explanations that rely solely on psychosocial factors, to a belief that disturbances in the function of brain neurotransmitter pathways are also highly relevant (Kaye, 1999). One outcome of this change in orientation has been an emerging and increasing interest in the links between eating disorders and substance abuse disorders.

Clinical and Biological Traits

It is generally agreed that the commencement of addictive behaviors can take two motivational routes: either the seeking of positive sensations or the self-medicating of painful affective states. While current research documents a substantial lifetime comorbidity between the eating disorders and other forms of addiction, there is less agreement on the reasons for this link (Holderness et al., 1994; Wiederman and Pryor, 1996). Some researchers have suggested that a common set of personality traits predispose an individual to a range of behaviors that have the potential to become excessive (Koob and Le Moal, 1997; Leshner, 1997). Support for this idea comes from evidence that anxiety and depression are frequent premorbid characteristics both of addicts (Grant and Harford, 1995; Kessler et al., 1997) and of patients with eating disorders (Deep et al., 1995; Vitousek and Manke, 1994). Our own research has also found that among eating-disordered patients, irrespective of diagnostic category, scores on a measure of addictive personality characteristics were comparable to those reported for drug addicts and alcoholics (Davis and Claridge, 1998). Complementary to this viewpoint, an addiction to one behavior reinforces a certain style of coping pattern that leaves the individual vulnerable to developing another type of addiction (Holderness et al., 1994).
Others have suggested that the eating disorders are, themselves, a form of drug addiction since their characteristics satisfy all the clinical and biological criteria for conventional addictions such as smoking, alcoholism and cocaine abuse (Davis and Claridge, 1998; Davis et al., 1999; Marrazzi and Luby, 1986). Foremost among these is the progressively compulsive nature of the behavior, even in the face of adverse consequences to health and safety (Heyman, 1996; Robinson and Berridge, 1993). Moreover, with continual exposure, individuals typically require more of the behavior to produce the same reinforcing effect (Berridge and Robinson, 1995). They also tend to experience an obsessively increasing craving for the behavior that can persist even after a long period of abstinence. Presumably that accounts, at least in part, for the fact that addicts have a strong tendency to resume the addictive behavior after treatment and for the chronic relapsing nature of addiction (Robinson and Berridge, 1993). These characteristics find direct parallels in the core eating-disorder behaviors such as dieting, over-exercising and binge eating, all of which tend to become increasingly excessive over time. Patients also report a strong compulsion to continue these behaviors despite serious medical complications, which is reflected in their prolonged morbidity and the high rate of relapse (Herzog et al., 1999; Strober et al., 1999).

At the biological level, similarities are also evident. We know, for instance, that strenuous exercise and starvation activate the dopaminergic (DA) reward pathway of the brain (Bergh and Sodersten, 1996; Casper, 1998). The resulting biological events underlie the auto-addiction opioid theory, which proposes that a chronic eating disorder is an addiction to the body's production of endogenous opioids and therefore is identical to the physiology and psychology of substance abuse in general (Huebner, 1993; Marrazzi and Luby, 1986). In other words, starving, bingeing and exercise all serve as drug delivery devices since they increase circulating levels of β-endorphins that are chemically identical to exogenous opiates, and these endorphins are as potentially addictive because of their ability to stimulate DA in the brain's mesolimbic reward centers.

Via a different route, self-starving may have other biologically rewarding properties, albeit as a negative reinforcer. For example, in certain individuals, food restriction is reported to reduce anxiety. It has been suggested this might occur because of reduced serotonin activity in those with overactivity in this neurotransmitter system (Kaye, 1999).

**Vulnerability**

A key concept in current formulations about risk for addiction concerns individual differences in sensitivity to reward or the ability to experience pleasure—a concept firmly rooted in neurobiology. In recent years, theories of addiction have moved from an emphasis on physical dependence to a broader focus on motivational dependence and the importance of anhedonia and dysphoria as powerful incentives for the continuation of addictive behaviors (Di Chiara, 1999).
A large body of research has demonstrated that a relatively long-lasting anhedonic state can be induced by prolonged drug administration (Gamberino and Gold, 1999) and by exposure to chronic mild stress (Zacharko, 1994) and that this process is primarily mediated by DA receptor downregulation. Furthermore, anhedonia can also be an innate characterological trait associated with low DA availability; a factor that has consistently been associated with greater risk for a variety of addictions (Volkow et al., 1999).

Recently, we proposed that this concept has great utility for understanding certain differences between patients with anorexia nervosa (AN) and patients with bulimia nervosa (BN), viz that the former display a facility for self-starving and report a decreasing interest in food, while the latter have increasing difficulty resisting food and become compulsive overeaters (Davis et al., 2000). There is no doubt that these differences are influenced by a variety of cognitive factors relating to personal identity and a need for control as well as social reinforcement. As with addiction, there is good reason to believe that biologically based motivational effects relating to capacity for reward and the regulation of affective states are also influential. Given that food is the most basic natural reinforcer, our findings that patients with AN were significantly more anhedonic than those with BN offer support for the premise that individual differences in this characteristic contribute to the avoidance and approach relationships to food found in these two groups, respectively. The cross-sectional nature of our data raises the inevitable question of whether these differences reflect premorbid-and, therefore, potentially causal-characteristics, whether the stress of starvation experienced most acutely in the patients with AN induced their anhedonic state, or both. Recently it has been suggested that differences in the ability to experience the positive-incentive value of food may be diminished in response to an extended period of food deprivation, making it easier for patients with severe anorexia to starve themselves (Pinel et al., 2000).

Summary and Conclusions

There is a strong argument that the eating disorders are a form of addiction. Clinically, the behaviors that define eating disorders and substance abuse are very similar. Similar biological mechanisms account for the compulsively progressive nature of both disorders; in the case of alcohol and other drugs, from recreational use to a state of pathological dependence; in AN, from casual dieting to a life-threatening refusal to eat; and, in BN, the increasing inability to resist large quantities of food. There is also support for a common psychobiological vulnerability and for the notion that individuals use a variety of rewarding behaviors to self-medicate their affective disturbances depending on the specific effects of each. But why do some individuals choose nicotine, others alcohol and others food (or its absence)? Environment, sociocultural factors and personality must play an important role. For example, one who is obsessional, anxious, conforming and female is more likely to be attracted to highly sanctioned behaviors like dieting and exercise, and to the enormous rewards associated with the pursuit of thinness in our culture, than to illicit drug use. In order to understand the motivation to continue these behaviors
beyond the point of body-image improvements, we must look beyond psychosocial explanations. I suggest that the great strides which have occurred in our understanding of brain mechanisms in addiction also provide excellent insight into the processes that occur in the eating disorders. As such, they also offer a useful approach to improved methods of treatment.

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References


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