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The utility of behavioral economics in expanding the free-feed model of obesity☆

Erin B. Rasmussen*, Stephen H. Robertson, Luis R. Rodriguez

Idaho State University, United States

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ABSTRACT

Animal models of obesity are numerous and diverse in terms of identifying specific neural and peripheral mechanisms related to obesity; however, they are limited when it comes to behavior. The standard behavioral measure of food intake in most animal models occurs in a free-feeding environment. While easy and cost-effective for the researcher, the free-feeding environment omits some of the most important features of obesity-related food consumption—namely, properties of food availability, such as effort and delay to obtaining food. Behavior economics expands behavioral measures of obesity animal models by identifying such behavioral mechanisms. First, economic demand analysis allows researchers to understand the role of effort in food procurement, and how physiological and neural mechanisms are related. Second, studies on delay discounting contribute to a growing literature that shows that sensitivity to delayed food- and food-related outcomes is likely a fundamental process of obesity. Together, these data expand the animal model in a manner that better characterizes how environmental factors influence food consumption.

Contents

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* Corresponding author at: Dept. of Psychology, Stop 8812, Idaho State University, Pocatello, ID, United States.
E-mail address: rasmerin@isu.edu (E.B. Rasmussen).

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1. Animal models

Laboratory researchers often use reductionist methods to simplify mechanisms involved in rather complex phenomena, such as medical and psychological disorders. The use of animal models is one manner in which more simple systems and behaviors, and their causes and influences, can be isolated and understood.

The most fundamental properties of animals models have been a topic of discussion for decades (e.g., see McKinney, 1977; Robbins and Sahakian, 1979; Robinson and Becker, 1986; Tordjman et al., 2007). Three criteria seem to be especially relevant to psychological and health-related disorders, which often incorporate behavior: behavioral similarity, physiological similarity, and treatment sensitivity. Behavioral similarity refers to the degree to which behavior characterized by the animal model is similar to that exhibited in humans. Physiological similarity refers to underlying mechanisms that accompany the behavioral change, which are generally neural or peripheral. Treatment sensitivity would indicate the degree to which a treatment (e.g., in most cases, a drug) changes both the underlying mechanism and the behavior of interest, and the degree to which the same drugs lead to amelioration of symptoms in humans and non-humans.

Few animal models meet all three of these criteria. More often, a model will satisfy one or two of the criteria. In some rare cases, all of the criteria are met, but the causes for physiological and behavioral change are mimicked or induced by other variables. For example, it is difficult to create a model of schizophrenia in non-humans in which stereotypic behavior can be examined. However, dopaminergic compounds can be used to induce stereotypies in animals that are in some ways similar, and, like humans, reflect dopaminergic involvement (see Geyer and Markou, 2002 for review). Nonetheless, the cumulative use of animal models in research can provide critical information that together can be used to first understand specific mechanisms, eventually leading to the development of effective treatments for diseases and disorders.

1.1. Animal models of obesity

There are dozens to hundreds of animal models of obesity (Speakman et al., 2007; Lutz and Woods, 2012). Given that the incidence of obesity has dramatically increased in the USA and other industrialized nations in the last two to three decades (Centers for Disease Control and Prevention, 2012; National Center for Health Statistics, 2012), and because obesity is a health-related problem that involves many of the systems in the body (e.g., pancreatic, hypothalamic), a large variety of animal models have been developed to better understand how specific systems are affected—from the molecular level to the behavioral level. This paper is not meant to review these types of models, or to comment to a great extent about the degree to which they meet the three criteria for a strong animal model. Rather, we wish to describe them to provide a general context for a central issue.

Models for obesity are generally separated into several categories, though the details of the grouping can differ from one review to another (cf. Kaiyala and Schwartz, 2011; Leibel, 2008; Schwartz et al., 2000; Lutz and Woods, 2012; Speakman et al., 2007; Woods, 2009). For the purposes of this paper, we will group them as follows:

1.1.1. Environmental models

A number of animal models use environmental conditions to induce obesity and these are usually dietary in nature, emphasizing exposure to palatable diets for specified time periods (see Speakman et al., 2007; West and York, 1998). Dietary models reflect strong ecological validity in terms of cause-and-effect relations observed in humans. One example is the high fat diet. In this model, rodents (though, there are others—see West and York, 1998) are chronically fed either a diet high in fat or a standard nutritionally balanced diet. Rats exposed to the high-fat diet exhibit hyperphagia, which results in excessive weight gain and adiposity, compared to controls (e.g., Warwick et al., 2000; West and York, 1998; Woods et al., 2003).

The high-fat diet model appears to meet all three properties of a strong animal model of obesity. Johnson and Kenny (2010) for example, exposed basic laboratory rats for 40 days to either standard chow, an extended high-fat diet in which a diet high in fat (e.g., frosting, cheesecake, etc.) was in place for 23-h per day, and a reduced exposure group that only received a high-fat diet for one hour per day. Rats exposed to the extended high fat diet ate 2–3 times more kcalories (kcal) than rats in the standard chow condition and gained more than twice as much weight across the 40-day period. In addition, these rats showed a 75% reduction in dopamine D_2 receptors in the striatum of the brain compared to controls and those in the reduced exposure group, both of which showed modest reductions in D_2 densities and little weight gain. Other studies have shown similar effects (Warwick et al., 2000; West and York, 1998; Woods et al., 2003).

With humans, a diet high in fat also leads to heightened caloric intake and weight gain (e.g., Lissner et al., 1987; Schrauwen and Westerterp, 2000). Moreover, imaging studies show a similar reduction of striatal dopamine D_2 receptors inversely correlated to BMI, which supports a common neurobiological mechanism (Wang et al., 2001). These data support that there is similarity between humans and rodents in terms of behavioral change by way of increased food intake and weight gain, identify at least one common neurobiological mechanism, and show that the common cause is diet related; therefore, the first two properties of a strong animal model are met. In addition, in terms of drug sensitivity, compounds that block D_2 receptors (e.g., anti-psychotic drugs like haloperidol and olazepine) are associated with increased caloric intake and weight gain in rats (e.g., Cooper et al., 2005, 2007) and humans (e.g., Moisan et al., 2005; Zipursky et al., 2005). Therefore, there is reasonable evidence that all three criteria for a strong animal model are met with high-fat diet.

1.1.2. Genetic models

Compared to environmental models, far more models that are genetic in nature have been developed to understand how expression of specific genes influence physiological and behavioral changes involved in obesity (Speakman et al., 2007). While genetic differences in humans generally account for less variability in obesity compared to environmental differences (especially diet), genetic models can isolate which genes control specific peptides, hormones, or receptors involved in obesity, which can lead to effective drug development that targets these systems. Genetic models can be divided into the following subcategories:

1.1.2.1. Single-gene loss of function. There are at least ten single-gene loss of function models that have been developed to characterize the extent to which variations in specific dominant and recessive allele combinations result in phenotypic differences that lead to obesity (Speakman et al., 2007). Three frequently used mouse models—all of which involve leptin impairment—are the *ob/ob, db/db, and s/s* strains (Lutz and Woods, 2012). The obese Zucker rat, which possesses the recessive *fa/fa* allele combination, is a single-gene rat model. Within three weeks of age, the *fa/fa* Zucker rat eats more, weighs more, and has higher adiposity than lean Zucker rats that possess one or two dominant lean alleles (see Beck, 2000; Sahu, 2004 for reviews; Zucker and Zucker, 1961). One of the main target impairments in the Zucker strain, as well as other single-gene models, is leptin regulation, though leptin also affects endocannabinoid and dopaminergic regulation (both of which are also differentially affected in the Zucker rat (Di Marzo et al., 2001;