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Differential strain vulnerability to binge eating behaviors in rats

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HIGHLIGHTS

- Binge eating proneness was examined in two different outbred strains of adult rats.
- Sprague–Dawley female rats were significantly more likely to be binge eating prone.
- Wistar female rats were more likely to be resistant to binge eating behaviors.
- Sprague–Dawley female rats may be a vulnerable strain for binge eating phenotypes.

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ABSTRACT

Binge eating is a significantly heritable phenotype, but efforts to detect specific risk genes have fallen short. Identification of animal strain differences in risk for binge eating could highlight genetic differences across individuals of the same species that can be exploited in future animal and molecular genetic research. The current study aimed to explore strain differences in risk for binge eating in Sprague–Dawley versus Wistar female rats using the Binge Eating Resistant/Binge Eating Prone model. A sample of male Sprague–Dawley rats, a known low-risk group for binge eating, was included as a comparison group. A total of 83 rats (23 Wistar females, 30 Sprague–Dawley females, 30 Sprague–Dawley males) completed a protocol of intermittently administered, palatable food. Binge eating prone (BEP) and binge eating resistant (BER) rats were identified using a tertile approach. Sprague–Dawley female rats consumed the highest amount of palatable food and were more likely to be classified as BEP compared to Wistar female and Sprague–Dawley male rats. Wistar female rats were not significantly different from Sprague–Dawley male rats in their palatable food intake and tendency to be classified as BER rather than BEP. Sprague–Dawley female rats appear to be a particularly vulnerable genotype for binge eating. Comparisons between this group and others could help identify specific genetic/biological factors that differentiate it from lower risk groups. The reward system, linked to binge eating in humans, is a possible candidate to explore. Strain differences in the reward system could help increase understanding of individual differences in risk for binge eating in humans.

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1. Introduction

Eating disorders are significant psychological disorders that are affected by both biological and genetic factors. Of the primary eating disorders, anorexia nervosa (AN), bulimia nervosa (BN) and binge eating disorder (BED), binge eating is a core feature of each disorder [1,2]. Binge eating involves consumption of large amounts of food in a short period of time while experiencing a loss of control during the episode [3]. In addition to eating disorders, binge eating is also associated with elevated rates of obesity [4–6] and other forms of psychopathology, such as major depression [7].

Previous research has suggested that genetic factors may contribute to binge eating. Twin studies have shown that eating disorders are significantly heritable, with 50–83% of the variation in the population resulting from genetic influences [8]. Importantly, binge eating itself is also heritable with estimates ranging from 50-82% [9]. While twin data have indicated the importance of genetic factors, molecular genetic research has been relatively inconclusive in identifying specific risk genes for binge eating. Interestingly, genes within the opioid/dopaminergic systems (e.g., mu-opioid receptor, dopamine D2 receptor) that contribute to reward processes have been associated with binge eating and the types of palatable food (e.g., high sweet, high fat foods) consumed during a binge episode [10]. Further understanding of underlying genetic factors within these systems may help to gain increased insight into the etiology of binge eating.

While initial findings from human studies have been helpful in understanding binge eating risk, animal studies offer a unique perspective

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by providing the opportunity to study behaviors in the absence of psychosocial influences found in human research [11]. For example, body dissatisfaction and peer influences are psychosocial influences that contribute to the development of binge eating in humans [6,12]. Animals likely do not experience these same risk factors for development of binge eating, and therefore, animal models can more easily isolate biological and genetic factors that influence binge eating.

To more clearly study biological and genetic differences, animal models, particularly rodent models, use genetically diverse outbred strains [13,14]. The heterogeneity within an outbred strain allows for discovery of differential phenotypes within the strain, which is similar to the diversity found in a human population. Importantly, even though an outbred strain is heterogeneous, animals within the same strain are more similar to one another than they are to animals of a different outbred strain, since rats from the same outbred strain were bred from the same initial parental animals. Taken together, these genetic features of outbred strains allow for identification of extreme phenotypes within a strain, but also the examination of strain differences in behavioral phenotypes.

In relation to binge eating, identifying specific outbred strains that exhibit more or less binge eating behavior may help narrow the search for potential genetic and biological risk factors. Follow-up research could then use information on phenotypic and genetic strain differences to identify potential risk genes for binge eating. To date, however, no studies have examined strain differences in binge eating in animal models; however, several animal models of binge eating exist that would be appropriate for such investigations. The Binge Eating Resistant/Binge Eating Prone (BER/BEP) model is a well-established animal model of binge eating that has been successfully utilized in previous research [11,15,16]. The BER/BEP model has high face validity for binge eating behaviors as they appear in humans, and there are several features of the model that make it ideal for studying strain differences in binge eating. The BER/BEP model identifies binge eating prone rats based on amount of palatable food consistently consumed during the first four hours of a 24-hour feeding test. For example, animals classified as BEP are required to consistently consume high amounts of palatable food that are higher than palatable food consumption of BER rats. Four-hour intakes have been shown to be a reliable time frame in which to observe differences in palatable food intake [15], and is similar to binge eating patterns observed in humans which occurs over a short period of time. The feeding tests are administered every few days, which models the intermittent pattern typically seen in binge eating patterns in humans. Importantly, food consumed during binge episodes in binge eating prone rats is typically palatable food that is high in sweetness and fat, as opposed to standard rat chow [11,15–17]. Consumption of highly palatable food during binge eating episodes in BEP rats is similar to the binge eating that is present in humans where a large proportion of the food consumed during binge eating episodes is comprised of palatable food [18]. While cognitive symptoms of binge eating are more difficult to model, research has also provided evidence that binge eating prone rats will endure painful foot-shock for the opportunity to consume palatable food [16]. Therefore, similar to binge episodes in humans, rats may exhibit behaviors resembling a loss of control over binge eating [3,11]. Furthermore, binge eating prone animals do not differ in body weight when compared to binge eating resistant animals [15]. This is similar to data showing that women with BN typically are not overweight but are of average weight [15,16]. In sum, while it is difficult to model all aspects of human binge eating in an animal model, the BER/BEP model has strong face validity, as it is able to model many key features of binge eating behaviors as they appear in humans.

To date, all studies of the BER/BEP model have used rats from the outbred Sprague–Dawley strain, yet other outbred strains have the possibility to show increased or decreased vulnerability towards binge eating. Although no studies have specifically examined strain differences in binge eating, one study examined strain differences in taste preferences of 17 liquid taste compounds [19]. This study used 14 different rat strains, three of which were outbred strains (including Sprague–Dawley and Wistar), and found that the Sprague–Dawley strain had a stronger preference for the sweet compounds. These findings provide indirect evidence that the Sprague–Dawley strain may indeed be particularly vulnerable to consumption of highly palatable food. Notably, the Wistar rat strain had a lower preference for sweet solutions, suggesting that it might represent a particularly low risk strain for palatable food consumption. As previously mentioned, preference for sweetness is present in binge eating, as binge foods tend to be highly palatable, high-sweet foods. This suggests that the Sprague–Dawley strain may be a particularly high-risk strain for binge eating, while the Wistar strain may be a low-risk strain.

The aim of the current study was to examine this possibility by directly examining group differences in the number of rats classified as binge eating prone in 30 Sprague–Dawley female rats and 23 Wistar female rats. First, we examined differences in palatable food intake between Sprague–Dawley and Wistar rats. These analyses were used to examine general differences in palatable food consumption across the Sprague–Dawley and Wistar strains. Second, strain differences in binge eating proneness were examined using a tertile approach. This method identifies binge eating prone and binge eating resistant rats based on the 4-hour palatable food intakes across feeding tests. Importantly, in both analyses, female rats were examined, but a group of previously phenotyped, Sprague–Dawley male rats (see Klump et al. [20]) was also included as a comparison group. Previous research has shown that Sprague–Dawley male rats consistently consume smaller amounts of palatable food across feeding tests as compared to Sprague–Dawley female rats. Therefore, the number of male rats classified as binge eating prone is smaller than the number of Sprague–Dawley female rats classified as binge eating prone. The inclusion of a male comparison group will help determine the degree to which binge eating proneness observed in the Wistar female rats matches that of a known low-risk group (i.e., the Sprague–Dawley male rats) or a high-risk binge eating prone group (i.e., the Sprague–Dawley female rats). Interestingly, if the binge eating proneness observed in the Wistar female rats is similar to that of a known low-risk strain (i.e., Sprague–Dawley male rats), it would indicate that differences in binge eating proneness expand beyond just sex differences (i.e., females are more likely to be binge eating prone compared to males) to strain differences as well.

2. Methods

2.1. Animals

A sample of 83 previously BER/BEP phenotyped animals (female Sprague–Dawley n = 30, initial body weight $M(SD)$ = 184.96 g (6.44); male Sprague–Dawley n = 30, initial body weight $M(SD)$ = 260.23 g (6.25); female Wistar n = 23, initial body weight $M(SD)$ = 165.74 g (21.74)) were examined in this study. All of these animals were part of a larger study examining sex differences in binge eating phenotypes in Sprague–Dawley male and female rats (already published in Klump et al. [20]) as well as a study investigating differences in binge eating between wild-type (n = 12) and serotonin transporter knock-out (n = 11) female Wistar rats. Notably, comparisons of wild-type versus knock-out rats showed no significant differences in palatable food intake or binge eating proneness between groups (all p's > .10), suggesting that removal of the serotonin transporter gene did not influence binge eating behavior in Wistar rats. Therefore, all Wistar rats were combined into one group for the analyses reported herein. Importantly, the same pattern of results was obtained when only Wistar wild-type rats were included in analyses (data not shown).

All Sprague–Dawley rats were obtained on approximately postnatal day 60 from Harlan (Madison, Wisconsin), while the 23 female Wistar rats were obtained from Dr. James Galligan (Department of Pharmacology

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