Published in final edited form as:

J Exp Anal Behav. 2017 September ; 108(2): 171–183. doi:10.1002/jeab.275.

DELAY DISCOUNTING AS IMPAIRED VALUATION: DELAYED REWARDS IN AN ANIMAL OBESITY MODEL

David P. Jarmolowicz¹, Jennifer L. Hudnall¹, Luanne Hale¹, Stephen C. Fowler¹, Marco Bortolato², Shea M. Lemley¹, and Michael J. Sofis¹

¹University of Kansas

²University of Utah

Abstract

Obesity is a major public health problem, which, like many forms of addiction, is associated with an elevated tendency to choose smaller immediate rather than larger delayed rewards, a response pattern often referred to as excessive delay discounting. Although some accounts of delay discounting conceptualize this process as impulsivity (placing the emphasis on overvaluing the smaller immediate reward), others have conceptualized delay discounting as an executive function (placing the emphasis on delayed rewards failing to retain their value). The present experiments used a popular animal model of obesity that has been shown to discount delayed rewards at elevated rates (i.e., obese Zucker rats) to test two predictions that conceptualize delay discounting as executive function. In the first experiment, acquisition of lever pressing with delayed rewards was compared in obese versus lean Zucker rats. Contrary to predictions based on delay discounting as executive function, obese Zucker rats learned to press the lever more quickly than controls. In the second experiment, progressive ratio breakpoints (a measure of reward efficacy) with delayed rewards were compared in obese versus lean Zucker rats. Contrary to the notion that obese rats fail to value delayed rewards, the obese Zucker rats' breakpoints were (at least) as high as those of the lean Zucker rats.

Correspondence concerning this article should be addressed to David P. Jarmolowicz, Department of Applied Behavioral Science, University of Kansas, 4050 Dole Developmental Center, 1000 Sunnyside Ave., Lawrence, KS 66045 USA, dpj@ku.edu, phone: 1-785-864-2532, fax: 785-864-5202.

David P. Jarmolowicz, Department of Applied Behavioral Science and Problem Gambling Research and Education Support System, University of Kansas; Jennifer L. Hudnall, Department of Applied Behavioral Science, University of Kansas; Luanne Hale, Department of Applied Behavioral Science, University of Kansas; Stephen C. Fowler, Department of Applied Behavioral Science, University of Kansas; Marco Bortolato, Department of Pharmacology and Toxicology, University of Utah; Shea M. Lemley, Department of Applied Behavioral Science, University of Kansas; Michael J. Sofis, Department of Applied Behavioral Science, University of Kansas.

David P. Jarmolowicz conceptualized the study, assisted with data analysis, and wrote the manuscript. Jennifer L. Hudnall assisted with study conceptualization, data acquisition, and data analysis. Luanne Hale assisted with data analysis. Stephen C. Fowler and Marco Bortolato assisted with initial conceptualization of these experiments. Shea M. Lemley assisted with data acquisition and manuscript preparation. Michael J. Sofis assisted with data acquisition, data analysis, and manuscript preparation. All authors have read and approved this manuscript.

David P. Jarmolowicz declares that he has no conflicts of interest. Marco Bortolato declares that he has no conflicts of interest. Stephen C. Fowler declares that he has no conflicts of interest. Jennifer L. Hudnall declares that she has no conflicts of interest. Luanne Hale declares that she has no conflicts of interest. Shea M. Lemley declares that she has no conflicts of interest. Michael J. Sofis declares that he has no conflicts of interest.

Keywords

delayed rewards; reward valuation; progressive ratios; obesity; Zucker rats

Obesity is a worldwide public health problem due to the economic and social burdens of obesity-related health conditions (Lerma-Cabrera, Carvajal, & Lopez-Legarrea, 2016). Despite increased attention to this epidemic, obesity rates continue to rise worldwide, prompting further exploration of the underlying factors contributing to obesity (Gaetani et al., 2016). The underlying mechanisms responsible for obesity are multifaceted, and treatment efforts are often unsuccessful at producing maintained weight loss (Westerveld & Yang, 2016). Increasingly, however, the behavioral and neural underpinnings of maladaptive food intake resemble those of substance abuse; namely, dysregulated reward processing that underlies maladaptive decision making (Tomasi & Volkow, 2013; Tomasi et al., 2015). The behavioral and neural similarities between excessive food intake and substance abuse have led many to discuss this disrupted reward processing as food addiction (Volkow & Baler, 2015).

From a behavioral perspective, obesity is characterized by persistently high reward valuation and excessive preference for immediate food consumption, despite negative long-term consequences (Bickel, Jarmolowicz, Mueller, & Gatchalian, 2011). The latter of these two patterns, often referred to as excessive delay discounting (Epstein, Salvy, Carr, Dearing, & Bickel, 2010), has increasingly been linked to behavioral and neural underpinnings of obesity (Boomhower, Rasmussen, & Doherty, 2013; Rasmussen, Lawyer, & Reilly, 2010; Volkow & Baler, 2015). Specifically, obese individuals are more likely to be present-focused (Zhang & Rashad, 2008), and thus, their choice patterns might be said to reflect devaluation of delayed rewards. Further, excessive delay discounting predicts hedonic eating in humans (Rollins, Dearing, & Epstein, 2010), is linked to obesity in humans (Appelhans et al., 2011; Jarmolowicz, Cherry, et al., 2014; Weller, Cook, Avsar, & Cox, 2008) and animals (Boomhower et al., 2013), and is widely accepted as a critical feature of obesity-related decision making (Bickel, Jarmolowicz, Mueller, Koffarnus, & Gatchalian, 2012; Volkow & Baler, 2015).

The elemental behavioral processes that undergird delay-discounting rates, however, remain under-explored. One account associates high rates of delay discounting with impulsivity (Bickel & Marsch, 2001), emphasizing individuals' inability to inhibit their choices for smaller, immediate rewards at the loss of larger, later gains. By contrast, other accounts conceptualize delay discounting as an executive function, wherein high rates of discounting reflect an individual's inability to value future gains (Barkley, 1997; Bickel, Jarmolowicz, Mueller, Gatchalian, & McClure, 2012; Bickel & Yi, 2008; Jarmolowicz et al., 2013). Recent studies examining cross-commodity discounting, however, provided unique insight into the relative roles of the lure of the immediate reward versus the inability to value future rewards (i.e., executive function) on rates of delay discounting. Such studies examined whether the elevated rates of discounting for consumable commodities were due to those commodities failing to maintain their value when delayed, or whether high rates of discounting resulted from an inability to withhold the choice for an immediately available

reward. In a series of studies comparing delay discounting for cocaine versus money (Bickel, Landes, et al., 2011) and sex versus money (Jarmolowicz, Landes, et al., 2014), cocaine-dependent participants completed delay discounting tasks assessing choices in four conditions: immediate, consumable commodity versus delayed, consumable commodity (CC); immediate money versus delayed money (MM); immediate, consumable commodity versus delayed money (CM); and immediate money versus delayed, consumable commodity (MC). These four conditions allowed the researchers to analytically isolate the role of changes in the immediate reward (while holding the delayed reward constant) and changes in the delayed reward (while holding the immediate reward constant). Bickel, Landes, et al. (2011) found that elevated rates of discounting for cocaine relative to money were due to cocaine failing to maintain its value when delayed. Although these findings suggest elevated rates of delay discounting result from devaluation of delayed rewards, experimentally—rather than analytically—isolating the role of the immediate and delayed commodities would allow for stronger statements about the relative roles of immediate and delayed rewards in delay discounting.

Consistent with the differences seen between obese individuals and normal-weight controls (Appelhans et al., 2011; Jarmolowicz, Cherry, et al., 2014; Weller et al., 2008), delay discounting rates are predictably elevated in animal models of obesity (Boomhower et al., 2013). One popular animal model of genetic obesity, Zucker fatty rats (Zucker & Zucker, 1961) discount delayed rewards more rapidly than their genetically similar control strain (Boomhower et al., 2013). This animal model has mutated leptin receptor genes (Phillips et al., 1996), and their two homozygous *fa/fa* “fatty” alleles result in leptin insensitivity. This insensitivity is linked to a hyperphagic phenotype and increased levels of appetite (Buckley & Rasmussen, 2012; Phillips et al., 1996). These rats are hyperphagic (Kava, Greenwood, & Johnson, 1990), tend to choose diets that are higher in fat than their control strain (Castonguay, Hartmann, Fitzpatrick, & Stern, 1982), and have lower levels of locomotor activity (Towa, Kuwahara, & Tsubone, 2004). These rats’ elevated discounting rates make the Zucker fatty an ideal model to test two expected outcomes of an inability to value delayed rewards—namely, impaired learning with delayed rewards and suppressed efficacy of delayed rewards.

Experiment 1

The ability of an animal to learn a response that results in the delivery of a given reinforcer can be seen as an index of the effectiveness of that reward (Bickel, Madden, & Petry, 1988). For example, consistent with the devaluation of delayed rewards, rats’ acquisition of lever pressing is more rapid for immediate than for delayed rewards (Sgro, Dyal, & Antastasio, 1967; van Haaren, 1992). Moreover, this effect is parametric, with acquisition more significantly impaired by longer delays (Wilkenfield, Nickel, Blakely, & Poling, 1992). Potentially reflecting obese Zuckers’ sensitivity to food rewards, fatty Zucker rats acquire lever pressing with immediate rewards more rapidly than lean Zucker rats (Rasmussen & Huskinson, 2008). Obese Zucker rats, however, discount delayed rewards at higher rates than lean Zucker rats (Boomhower et al., 2013). If delay discounting is primarily driven by an inability to value future events, this impaired reward valuation should be reflected in obese Zucker rats acquiring responses less quickly than controls under conditions of delayed

reinforcement. The present experiment tested this hypothesis by comparing obese and lean Zucker rats' acquisition with delayed rewards.

Method

Subjects—Six male obese Zucker Rats (fa/fa) and six male lean Zucker Rats (fa/x) obtained from Charles River (Raleigh, NC) were maintained on 22-hr food restriction for the present experiment. Rats had access to food during the experimental sessions and for a 2-hr access period on nonexperimental days. Thus, rats had been food-restricted for 22 hr on all experimental days, but only 18 hr on the days following sessions. For this reason, sessions were run on alternating days, with the end of experimental sessions separated from the beginning of the next experimental session by 40 hr. Rats were 60 days old at the beginning of the experiment (weights; obese, $Mean = 229.62$ g, $SD = 9.39$ g; lean, $Mean = 168.42$ g, $SD = 17.66$ g; unpaired t -test with Welch's correction $t(7.617) = 7.495$, $p < .0001$) and were fed and housed in same-strain pairs. Water was freely available in the home cages, located in a colony room on a 12 hr:12 hr light–dark cycle. All sessions were conducted during the light cycle. All procedures followed the guidelines established by the University of Kansas Institutional Animal Care and Use Committee.

Apparatus—Sessions occurred in operant conditioning chambers (30.5 cm long, 24.1 cm wide, 21.0 cm high). Centered on the front wall, 1 cm above the floor grid was a pellet receptacle (3 cm x 4 cm) into which a pellet dispenser could dispense grain-based pellets (45 mg; Bio-Serv, Frenchtown, NJ). Retractable levers were positioned on either side of the pellet receptacle (11 cm apart; 5 cm from the floor). A 28-V DC cue light was positioned 2 cm above each lever, and a 28-V houselight centered on the back wall (19 cm from the floor) provided general illumination. Chambers were housed in sound-attenuating cubicles with fans to mask extraneous noise. All experimental events were programmed and recorded using MED-PC IV software (Tatham & Zurn, 1989).

Procedure—Rats began food restriction 1 week prior to the experiment. During the experiment, sessions lasted 6 hr and occurred at approximately the same time every other day. Thus, the entire experiment was conducted over the course of 7 days.

Operant pretraining: First, each rat experienced one magazine training session wherein levers remained retracted, but the houselight was lit and 60 pellets were delivered on a variable time 50-s schedule. All pellets were consumed during this pretraining.

Experimental sessions: Experimental sessions began with illumination of the houselight and insertion of the left lever into the chamber. Lever presses initiated a 17.5-s delay after which a single 45-mg grain-based pellet was delivered (accompanied by a 0.1-s tone and retraction of the lever). Lever presses during the delay interval reset the delay. After a 5-s consumption period, the left lever was reinserted into the chamber. Sessions ended after 6 hr.

Results

Figure 1 shows the mean number of lever presses per session for rats in the obese and lean groups. Although the number of lever presses increased across sessions for both obese and

lean rats, the mean was always higher in the obese rats. A repeated measures ANOVA found main effects of session ($F_{[2,20]} = 17.238, p < .001$), and group ($F_{[1,10]} = 17.09, p = .002$), but no interaction ($F_{[2,20]} = 0.772, p = 0.475$).

Figure 2 shows cumulative rates of lever pressing for each rat. The group differences reported in Figure 1 are evident in the cumulative rates of responding for each rat shown in Figure 2. Specifically, the cumulative records of lever pressing in the obese rats were considerably steeper than those for all but one (2P1) of the lean rats. These differences were confirmed by an independent samples *t*-test of the area under the curves (AUC)¹ ($T_{[10]} = 4.194, p = 0.002$), with the AUC for obese Zucker rats (Mean = 0.27, SEM = 0.03) being higher than the AUC for lean Zucker rats (Mean = 0.07, SEM = 0.03).

Although obese rats acquired responding more quickly than lean rats, obese rats also tended to respond more inefficiently. A 17.5-s delay between each initial lever press and pellet delivery characterized maximally efficient responding. Pressing the lever during the delay, however, resulted in additional delay. Thus, additional delays indexed inefficient responding. Figure 3 shows the mean additional delay experienced by each rat (log transformed to normalize the distributions). An unpaired *t*-test with Welch's correction found the additional delays experienced by the obese group to be significantly longer than those experienced by the lean group ($t_{[5.225]} = 3.285, p = 0.02$).

Discussion

Despite our predictions based on the elevated rates of delay discounting in obese Zucker rats relative to controls (Boomhower et al., 2013), response acquisition with delayed rewards was not impaired in this animal model of obesity, but may have actually been facilitated relative to their control strain. The present data are consistent with the elevated food reward valuation seen at low response requirements in obese Zucker rats (Rasmussen, Reilly, & Hillman, 2010) and across most ratio requirements in obese clinical populations (Epstein, Dearing, Temple, & Cavanaugh, 2008; Epstein, LeDdy, Temple, & Faith, 2007; Epstein et al., 2010; Epstein, Temple, et al., 2007). These data, however, are not consistent with elevated rates of delay discounting found in obese versus lean Zucker rats (Boomhower et al., 2013) and overweight versus healthy weight individuals (Appelhans et al., 2011; Jarmolowicz, Cherry, et al., 2014; Weller et al., 2008), which suggest a failure to value delayed rewards.

Although the obese Zucker rats more robustly acquired lever pressing during the training sessions, they often responded more inefficiently than the lean Zucker rats, resetting their delays resulting in longer additional delays experienced (Fig. 2). This inefficient responding (i.e., delays longer than 17.5 s) was maladaptive, as it resulted in lower rates of reinforcement than would have been obtained with more efficient responding (i.e., 17.5 s delays). Such a pattern of disinhibited responding is consistent with the response inhibition

¹Because the cumulative records consisted of a series of x (time) – y (cumulative number of response) coordinates, area under the curve (AUC) was able to be calculated from these coordinates for each rat using the trapezoid rule. For these calculations, the y-axis was converted to the proportion of the highest cumulative number of responses obtained for any of the rats in the experiment, and the x-axis was converted to proportion of elapsed session time. The area under successive trapezoids was summed to provide a single AUC value summarizing the rat's responding across the three acquisition sessions ($AUC = \sum (x_2 - x_1) * (y_1 + y_2)/2$).

deficits seen in clinical populations (Maayan, Hoogendoorn, Sweat, & Convit, 2011; Verdejo-García et al., 2009), providing additional behavioral validation of the Zucker model.

Lastly, although Experiment 1 failed to demonstrate an impaired propensity of obese Zucker rats to learn with delayed rewards, the conclusions that can be made from this experiment are limited in at least two ways. First, although the between-group differences seen in the present experiment are arguably more robust than the differences in acquisition with immediate rewards obtained by Rasmussen and Huskinson (2008), this particular comparison was not made in the present experiment, making it difficult to disentangle the effects of food motivation in general, and motivation for delayed rewards. Second, although intact acquisition with delayed rewards may provide some evidence that delayed reinforcers impact the responding of obese Zucker rats, the processes undergirding acquisition with delayed reinforcement remain unclear (cf. Johansen et al., 2009; Killeen, 2011; Lattal, 2010) and the link to reward efficacy is somewhat indirect.

Experiment 2

Progressive ratio (PR) schedules have been widely used to examine the relative efficacy of various drug and nondrug rewards (Jarmolowicz & Lattal, 2010; Stafford, LeSage, & Glowa, 1998). Specifically, although some authors (Arnold & Roberts, 1997; Bickel, Marsch, & Carroll, 2000) question the use of PR schedules as a measure of absolute reinforcing efficacy based on their divergence from other measures of reward efficacy and the sensitivity of PR breakpoints to nonmotivational variables (e.g., breakpoint criterion, step size; Stafford & Branch, 1998), when such procedural variables are held constant, breakpoint can provide a stable and replicable index of relative reinforcing efficacy, independent of response rate (which can be confounded with motoric effects). Although there is a large literature on clinical samples of obese individuals overvaluing food rewards (Epstein et al., 2008; Epstein, LeDdy, et al., 2007; Epstein et al., 2010; Epstein, Temple, et al., 2007), obese Zucker rats only have slightly higher PR breakpoints for food than lean Zucker rats (Glass, O'Hare, Cleary, Billington, & Levine, 1999; Rasmussen & Huskinson, 2008).

The impact of delays to reinforcement on PR breakpoints in obese versus lean Zucker rats, however, remains undetermined. Consistent with the notion that delays of reinforcement reduce the value of rewards, PR breakpoints systematically decrease as delays of reinforcement increase (Jarmolowicz & Hudnall, 2014; Jarmolowicz & Lattal, 2011). If delay discounting is primarily driven by the devaluation of delayed rewards, decreases in delay-based breakpoints should be particularly pronounced in obese relative to lean Zucker rats. The present study used a novel PR-with-delayed-rewards procedure to test this hypothesis.

Method

Subjects—The same 12 Zucker rats (approximately 70 days old, weights: obese, *Mean* = 255.85 g, *SD* = 11.40 g; lean, *Mean* = 204.05 g, *SD* = 15.40 g; unpaired *t*-test with Welch's correction [$t(9.21) = 6.623, p < .0001$]) that were used in Experiment 1 were used in Experiment 2. Their care was the same as in Experiment 1, with two exceptions: Rats in

Experiment 2 were maintained on a consistent 22-hr food restriction schedule, and sessions were conducted daily.

Apparatus—The same apparatus used in Experiment 1 was used in Experiment 2.

Procedure—Because rats in this experiment were used in Experiment 1, details of their response acquisition are described above. After this initial acquisition period, rats were transferred to an FR1 schedule, and the ratio requirement was systematically extended to FR 20 over the course of several sessions (5–8). Each rat was exposed to two baseline and two delay-of-reinforcement conditions. During the experiment, sessions occurred 6–7 days per week and occurred during the light period at roughly the same time each day.

Baseline sessions: When baseline sessions began, the house light was turned on and one lever was inserted into the chamber. Subjects responded on a PR 5 schedule wherein the ratio requirement started at 5 and increased systematically by five responses following the delivery of each reinforcer. Completing the ratio requirement resulted in a reinforcer consumption period which included a brief tone (0.1 s) followed by the delivery of a food pellet and the lever being retracted for 5 s. The house light remained on during these reinforcer consumption periods. Sessions terminated after a 300-s period without lever pressing, with the highest ratio completed being called the breakpoint. The baseline phases continued for at least 13 sessions and until the data met the stability criterion (i.e., when the final six sessions of the phase evidenced no monotonic trends, and the mean of the first three sessions and last three sessions did not vary from the grand mean by more than 6%).

Delay-of-reinforcement sessions: Once baseline breakpoints were stable, the delay-of-reinforcement conditions began. During the delay condition, the lever was retracted following ratio completion for x s (i.e., a signaled delay). After this delay, a pellet was delivered and the consumption period began. The value x started at 2 for the first session and doubled (e.g., 2 s, 4 s, 8 s, 16 s, etc.) during each subsequent session (Jarmolowicz & Lattal, 2013). This progression continued until the breakpoint dropped to 20% of the of baseline mean (i.e., mean of the final six sessions of baseline).

Results

Figure 4 shows the individual subject and mean breakpoint for obese versus lean Zucker rats during baseline (no delay) sessions. For this comparison, individual subject breakpoints were calculated by averaging the breakpoints over the final six sessions of the baseline phase. Although baseline breakpoints were somewhat higher for the obese (relative to lean) rats, a t -test found that this difference was not statistically significant ($t[10] = 1.25$, $p = 0.24$).

Figure 5 shows the mean breakpoints for obese versus lean Zucker rats at each delay experienced². A consistent pattern of decreasing breakpoints as the delays increased was

²For Figures 5–7, a value of zero (the lowest value possible) was imputed for delays or ratios not experienced because the breakpoint had been reached at a lower value. For Figure 7, a PRP value of 300 s (the longest pause possible) was imputed for any ratio not experienced because the breakpoint had been reached at a lower ratio.

obtained in most rats. Although breakpoints decreased as delays increased for both groups, breakpoints were consistently lower for the lean Zucker rats. A two-way ANOVA found main effects of delay duration ($F[10,98] = 16.79, p < .0001$) and group ($F[1,98] = 10.82, p = 0.0014$) but no significant interaction ($F[10,98] = 0.80, p = 0.6277$). Follow-up comparisons using Sidak's multiple comparisons test, however, did not find significant between-group differences at any specific delay.

Figure 6 shows the relation between breakpoint and delay duration for each individual rat. A consistent negative relation between delay duration and breakpoint was obtained for most rats. Because the longest delay experienced was a product of the rats' performance, a t -test was used to compare the longest delay duration each rat experienced across groups. No significant differences were observed ($t[10] = 0.90, p = 0.39$).

Figure 7 shows the interactive effects of ratio requirement and delay duration on overall response rate, run rate, and PRP in obese and lean rats. Increases in delay consistently decreased response rates and increased PRPs, and increases in ratio consistently decreased response rates and increased PRPs. Although response rate decreases and PRP increases were systematic across delays, these patterns did not vary systematically across groups.

Discussion

Consistent with prior studies examining relations between PR breakpoint and delay of reinforcement, breakpoints systematically decreased as delays to reinforcement increased (Jarmolowicz & Hudnall, 2014; Jarmolowicz & Lattal, 2011). Closer examination of the data revealed consistent relations between delay duration and overall rate, run rate and postreinforcement pause, consistent with prior studies using this rapid delay procedure with fixed ratio schedules (Jarmolowicz & Lattal, 2013). These delay-of-reinforcement, invoked changes in behavior interacted with the ratio requirement. Responding of obese Zucker rats, however, did not differ from that of the lean Zucker rats. This failure to find poorer valuation of delayed rewards in the obese Zucker rats is not consistent with delay discounting being primarily driven by an inability to value delayed rewards.

General Discussion

The primary aim of the present experiments was to evaluate the hypothesis that obese Zucker rats' elevated rates of delay discounting are due to their inability to value delayed reinforcers. Specifically, we tested two expected results of an inability to value delayed reinforcers. In Experiment 1, we compared obese and lean Zucker rats' acquisition of lever pressing when reinforcers were delayed. The obese Zucker rats acquired lever pressing more quickly than the lean rats, a finding inconsistent with their being unable to value delayed rewards. Specifically, it was expected that if delay discounting reflected a failure to value delayed rewards, Zucker rats—which have been shown to discount delayed rewards at elevated rates (Boomhower et al., 2013)—would have difficulty learning with delayed rewards. In Experiment 2, we compared obese and lean Zucker rats' PR breakpoints for both immediate and delayed reinforcers. Overall, obese rats' breakpoints for delayed reinforcers were significantly higher than the lean Zucker rats' breakpoints, suggesting that delayed reinforcers were significantly more efficacious for obese relative to lean Zucker rats. This

finding is also inconsistent with obese Zucker rats' elevated rates of delay discounting (Boomhower et al., 2013) resulting from their inability to value delayed reinforcers. Interestingly, however, the obese Zucker rats responded more inefficiently (i.e., experienced longer resetting delays) than the lean rats in Experiment 1, providing indirect support for response inhibition deficits often observed in obesity (Volkow & Baler, 2015). In conjunction, data from the current studies provide further support for the conceptualization of obesity as an addiction-related phenomenon and highlight the relevance of reinforcement for the study of obesity. There are seven additional points we would like to make about these data.

First, there were substantial procedural differences between the present studies and Boomhower et al. (2013) study, which may have contributed to the present findings. Most notably, the Boomhower et al. procedure entailed a choice paradigm, whereas the present experiments did not. Thus, the obese Zucker rats' elevated delay discounting rates in the Boomhower et al. study could have been due to an inability of the delayed reward to maintain its value, the reinforcing value of the immediate reinforcer, or an interaction of the two. By contrast, the present experiments isolated the role of the delayed reinforcer. Additionally, the Boomhower et al. procedure manipulated reinforcer magnitude across conditions and showed substantial differences between obese and lean Zucker rats only when rats chose between one immediate and three delayed pellets. However, because the goal of the present experiment was to isolate the role of the delay of reinforcement, reinforcer magnitude remained constant across conditions.

Second, the present findings suggest that rates of delay discounting likely reflect complex interactions among multiple behavioral and neurobiological processes. Although the present findings failed to demonstrate that impaired valuation of future events alone was sufficient to drive the elevated rates of delay discounting seen in obese Zucker rats (Boomhower et al., 2013), the current findings do not preclude that impaired valuation is a strong component of this interaction. The necessity of this interaction is consistent with the preponderance of neuroeconomic data (Bickel, Pitcock, Yi, & Angtuaco, 2009; McClure, Ericson, Laibson, Loewenstein, & Cohen, 2007; McClure, Laibson, Loewenstein, & Cohen, 2004; Monterosso et al., 2007) and two-system theories, such as the competing neurobehavioral decision systems hypothesis, that posit that delay discounting reflects the relative balance of activation in two neurobiological systems (Bechara, 2005; Bickel, Jarmolowicz, MacKillop, et al., 2012; Bickel, Jarmolowicz, Mueller, Gatchalian, et al., 2012; Bickel, Johnson, Koffarnus, MacKillop, & Murphy, 2014; Bickel et al., 2007; Bickel, Mueller, & Jarmolowicz, 2013; Jentsch & Taylor, 1999; Koffarnus, Jarmolowicz, Mueller, & Bickel, 2013). The present findings, however, are not consistent with single-system valuation models that posit that delay discounting simply represents the decayed value of delayed rewards (Kable & Glimcher, 2007, 2010; Monterosso & Ainslie, 2007).

Third, the data in the present experiments were orderly and consistent with a general overvaluation of rewards in obese Zucker rats. Prior studies using immediate reinforcers have shown that obese Zucker rats have both elevated breakpoints and more robust response acquisition than lean Zucker rats (Rasmussen & Huskinson, 2008). Delays of reinforcement did decrease PR breakpoints in the obese Zucker rats, but not any more rapidly than they did

in the lean Zucker rats. Although the present data suggest that the elevated discounting rates in this model may be due to an overvaluation of smaller, immediate rewards, empirical substantiation remains to be demonstrated.

Fourth, the study of delay discounting decisions for food in leptin-insensitive animals might provide relevant clues to underlying neurobehavioral mechanisms of obesity. Further evaluation of delay discounting in leptin insensitive animals (i.e., Zucker fatty rats) may be fruitful, as obese individuals have higher concentrations of leptin than their nonobese counterparts (Considine et al., 1996) and leptin levels may influence weight regain after loss (Anastasiou, Karfopoulou, & Yannakoulia, 2015). In the current studies, the Zucker fatty rats did not demonstrate impaired performance relative to their lean counterparts. In conjunction with previous findings on elevated discounting rates in Zucker fatty rats, the current data suggest that the leptin deficiency present in obese Zucker rats may be uniquely related to altered rates of discounting. Future research might evaluate, for example, whether the obese Zucker rats' elevated amounts of lever resetting observed in Experiment 1 may be specifically related to their leptin insensitivity.

Fifth, the extent to which the present findings generalize to obese humans remains to be tested. Prior findings regarding the Zucker rats' modest overvaluation of food rewards (Rasmussen & Huskinson, 2008; Rasmussen, Reilly, et al., 2010) and elevated rates of delay discounting (Booth et al., 2013) are consistent with clinical findings constituting the backbone of the reinforcer pathologies model of obesity (Carr, Daniel, Lin, & Epstein, 2011). The present findings, however, require replication in obese humans. If replicated, these results may shift the focus of neurobehavioral treatment research in obesity from increasing the value of delayed rewards (Daniel, Stanton, & Epstein, 2013) to modulating the value of immediate rewards.

Sixth, results of the present study do not support the idea that elevated rates of delay discounting are due to a failure to value delayed rewards, as suggested by prior research on cross-commodity discounting. This may be due to one of two reasons. First, prior cross-commodity discounting studies examined the role of the delayed outcome in the context of choices between immediate and delayed rewards (Bickel, Landes, et al., 2011; Jarmolowicz, Landes, et al., 2014). As such, if the role of the delayed outcome is only present interactively with the availability of a smaller, immediate outcome, the cross-commodity procedure is better suited to demonstrate that effect. Alternatively, the variables that determine elevated rates of discounting in obese individuals may differ from those that undergird the elevated rates of delay discounting in addicted individuals. Future research will surely provide additional clarity on this point.

Finally, future research can address limitations of the present study. For example, the first experiment only examined acquisition with delayed rewards. Although Rasmussen and Huskinson (2008) found that obese Zucker rats acquired responding more quickly than the lean Zucker rats, it is unclear if delay impacted acquisition more robustly in the lean relative to obese Zucker rats in the present experiment. Similarly, increasing delays of reinforcement in the second experiment also decreased the rate of reinforcement during those sessions. Future studies could control for this decreased rate of reinforcement by adding a control

condition that features an equivalent intertrial interval (ITI; cf. Jarmolowicz & Lattal, 2013). Additionally, the present experiment only examined effects of delayed reinforcement in one animal model of obesity. Replication of these findings in additional models will strengthen the obesity-related statements that could be made about the present findings. Lastly, although obesity appears related to drug addiction, the relevance of the present findings to drug addiction are yet to be tested.

Acknowledgments

This research was supported by a grant from the University of Kansas New Faculty General Research Fund to DPJ, a Strategic Initiative Grant from the University of Kansas Researcher Investment Council to DPJ, and National Institute for Mental Health grant (R01MH104603) to MB.

The authors would like to thank the undergraduate research assistants working in the Jarmolowicz Lab for their help conducting this study.

References

- Anastasiou CA, Karfopoulou E, Yannakoulia M. Weight regaining: From statistics and behaviors to physiology and metabolism. *Metabolism*. 2015; 64(11):1395–1407. doi:<http://dx.doi.org/10.1016/j.metabol.2015.08.006>. [PubMed: 26362728]
- Appelhans BM, Woolf K, Pagoto SL, Schneider KL, Whited MC, Liebman R. Inhibiting food reward: Delay discounting, food reward sensitivity, and palatable food intake in overweight and obese women. *Obesity*. 2011; 19(11):2175–2182. DOI: 10.1038/oby.2011.57 [PubMed: 21475139]
- Arnold JM, Roberts DCS. A critique of fixed and progressive ratio schedules used to examine the neural substrates of drug reinforcement. *Pharmacology Biochemistry and Behavior*. 1997; 57:441–447.
- Barkley RA. Behavioral inhibition, sustained attention, and executive functions: Constructing a unifying theory of ADHD. *Psychological Bulletin*. 1997; 121(1):65–94. DOI: 10.1037/0033-2909.121.1.65 [PubMed: 9000892]
- Bechara A. Decision making, impulse control and loss of willpower to resist drugs: A neurocognitive perspective. *Nature Neuroscience*. 2005; 8(11):1458–1463. DOI: 10.1038/nn1584 [PubMed: 16251988]
- Bickel WK, Jarmolowicz DP, MacKillop J, Epstein LH, Carr K, Mueller ET, Waltz T. The behavioral economics of reinforcement pathologies: Novel approaches to addictive disorders. In: Shaffer, HJ., editor. *Addiction syndrome handbook*. Vol. 2. Washington, DC: American Psychological Association; 2012. p. 333–363.
- Bickel WK, Jarmolowicz DP, Mueller ET, Gatchalian KM. The behavioral economics and neuroeconomics of reinforcer pathologies: Implications for etiology and treatment of addiction. *Current Psychiatry Reports*. 2011; 13(5):406–415. DOI: 10.1007/s11920-011-0215-1 [PubMed: 21732213]
- Bickel WK, Jarmolowicz DP, Mueller ET, Gatchalian KM, McClure SM. Are executive function and impulsivity antipodes? A conceptual reconstruction with special reference to addiction. *Psychopharmacology*. 2012; 221(3):361–387. DOI: 10.1007/s00213-012-2689-x [PubMed: 22441659]
- Bickel WK, Jarmolowicz DP, Mueller ET, Koffarnus MN, Gatchalian KM. Excessive discounting of delayed reinforcers as a trans-disease process contributing to addiction and other disease-related vulnerabilities: Emerging evidence. *Pharmacology and Therapeutics*. 2012; 134(3):287–297. DOI: 10.1016/j.pharmthera.2012.02.004 [PubMed: 22387232]
- Bickel WK, Johnson MW, Koffarnus MN, MacKillop J, Murphy JG. The behavioral economics of substance use disorders: Reinforcement pathologies and their repair. *Annual Review of Clinical Psychology*. 2014; 10:641–677. DOI: 10.1146/annurev-clinpsy-032813-153724
- Bickel WK, Landes RD, Christensen DR, Jackson L, Jones BA, Kurth-Nelson Z, Redish AD. Single- and cross-commodity discounting among cocaine addicts: The commodity and its temporal

location determine discounting rate. *Psychopharmacology*. 2011; 217(2):177–187. DOI: 10.1007/s00213-011-2272-x [PubMed: 21487658]

- Bickel WK, Madden GJ, Petry NM. The price of change: The behavioral economics of drug dependence. *Behavior Therapy*. 1988; 29(4):545–565. DOI: 10.1016/S0005-7894(98)80050-6
- Bickel WK, Marsch LA. Toward a behavioral economic understanding of drug dependence: Delay discounting processes. *Addiction*. 2001; 96(1):73–86. DOI: 10.1046/j.1360-0443.2001.961736.x [PubMed: 11177521]
- Bickel WK, Marsch LA, Carroll ME. Deconstructing relative reinforcing efficacy and situating the measures of pharmacological reinforcement with behavioral economics: A theoretical proposal. *Psychopharmacology*. 2000; 153(1):44–56. [PubMed: 11255928]
- Bickel WK, Miller ML, Yi R, Kowal BP, Lindquist DM, Pitcock JA. Behavioral and neuroeconomics of drug addiction: Competing neural systems and temporal discounting processes. *Drug and Alcohol Dependence*. 2007; 90S:S85–S91. DOI: 10.1016/j.drugalcdep.2006.09.016
- Bickel, WK., Mueller, ET., Jarmolowicz, DP. What is addiction?. In: McCrady, B., Epstein, E., editors. *Addictions: A comprehensive guidebook*, Second edition. New York: Oxford University Press; 2013. p. 3-16.
- Bickel WK, Pitcock JA, Yi R, Angtuaco EJ. Equivalent neural correlates across intertemporal choice conditions. *NeuroImage*. 2009; 47(Supplement 1):S39–S41. DOI: 10.1016/s1053-8119(09)70766-9
- Bickel, WK., Yi, R. Temporal discounting as a measure of executive function: Insights from the competing neuro-behavioral decision system hypothesis of addiction. In: Houser, D., McCabe, K., editors. *Neuroeconomics: Advances in health services research*. Vol. 20. Bingley, UK: Emerald Group Publishing; 2008. p. 289-309.
- Boomhower SR, Rasmussen EB, Doherty TS. Impulsive-choice patterns for food in genetically lean and obese Zucker rats. *Behavioural Brain Research*. 2013; 241:214–221. DOI: 10.1016/j.bbr.2012.12.013 [PubMed: 23261877]
- Buckley J, Rasmussen E. Obese and lean Zucker rats demonstrate differential sensitivity to rates of food reinforcement in a choice procedure. *Physiology & Behavior*. 2012; 108:19–27. DOI: 10.1016/j.physbeh.2012.10.001 [PubMed: 23046726]
- Carr KA, Daniel TO, Lin H, Epstein LH. Reinforcement pathology and obesity. *Current Drug Abuse Reviews*. 2011; 4(3):190–196. [PubMed: 21999693]
- Castonguay TW, Hartmann WJ, Fitzpatrick EA, Stern JA. Dietary self-selection and the Zucker rats. *Journal of Nutrition*. 1982; 112:796–800. [PubMed: 7069514]
- Considine RV, Sinha MK, Heiman ML, Kriauciunas A, Stephens TW, Nyce MR, ... Caro JF. Serum immunoreactive-leptin concentrations in normal-weight and obese humans. *New England Journal of Medicine*. 1996; 334:292–295. DOI: 10.1056/NEJM199602013340503 [PubMed: 8532024]
- Daniel TO, Stanton CM, Epstein LH. The future is now: Reducing impulsivity and energy intake using episodic future thinking. *Psychological Science*. 2013; 24(11):2339–2342. DOI: 10.1177/0956797613488780 [PubMed: 24022653]
- Epstein LH, Dearing KK, Temple JL, Cavanaugh MD. Food reinforcement and impulsivity in overweight children and their parents. *Eating Behaviors*. 2008; 9(3):319–327. DOI: 10.1016/j.eatbeh.2007.10.007 [PubMed: 18549991]
- Epstein LH, Leddy JJ, Temple JL, Faith MS. Food reinforcement and eating: A multilevel analysis. *Psychological Bulletin*. 2007; 133(5):884–906. [PubMed: 17723034]
- Epstein LH, Salvy SJ, Carr KA, Dearing KK, Bickel WK. Food reinforcement, delay discounting and obesity. *Physiology and Behavior*. 2010; 100(5):438–445. DOI: 10.1016/j.physbeh.2010.04.029 [PubMed: 20435052]
- Epstein LH, Temple LJ, Neaderhiser BJ, Salis RJ, Erbe RW, Leddy JJ. Food reinforcement, the dopamine D2 receptor genotype, and energy intake in obese and nonobese humans. *Behavioral Neuroscience*. 2007; 121:877–886. [PubMed: 17907820]
- Gaetani S, Romano A, Provensi G, Ricca V, Lutz TM, Passani B. Eating disorders: From bench to bedside and back. *Journal of Neurochemistry*. 2016; 139:691–699. DOI: 10.1111/jnc.13848 [PubMed: 27649625]

- Glass MJ, O'Hare E, Cleary JP, Billington CJ, Levine AS. The effect of naloxone on food-motivated behavior in the obese Zucker rat. *Psychopharmacology*. 1999; 141:378–384. [PubMed: 10090645]
- Jarmolowicz DP, Cherry JB, Reed DD, Bruce JM, Crespi JM, Lusk JL, Bruce AS. Robust relation between temporal discounting rates and body mass. *Appetite*. 2014; 78:63–67. DOI: 10.1016/j.appet.2014.02.013 [PubMed: 24650831]
- Jarmolowicz DP, Hudnall JL. Concurrent progressive-ratio schedules: Built-in controls in the study of delayed reward efficacy. *Journal of Neuroscience Methods*. 2014; 233:172–176. DOI: 10.1016/j.jneumeth.2014.06.024 [PubMed: 24979725]
- Jarmolowicz DP, Landes RD, Christensen DR, Jones BA, Jackson L, Yi R, Bickel WK. Discounting of money and sex: Effects of commodity and temporal position in stimulant dependent men and women. *Addictive Behaviors*. 2014; 39:1652–1657. DOI: 10.1016/j.addbeh.2014.04.026 [PubMed: 24857686]
- Jarmolowicz DP, Lattal KA. On distinguishing progressively increasing response requirements for reinforcement. *The Behavior Analyst*. 2010; 33(1):119–125. [PubMed: 22479130]
- Jarmolowicz DP, Lattal KA. Reinforcer efficacy, response persistence, and delay of reinforcement. *Acta de Investigación Psicología*. 2011; 1:1–13.
- Jarmolowicz DP, Lattal KA. Delayed reinforcement and fixed-ratio performance. *Journal of the Experimental Analysis of Behavior*. 2013; 100(3):370–395. DOI: 10.1002/jeab.48 [PubMed: 24037852]
- Jarmolowicz DP, Mueller ET, Koffarnus MN, Carter AE, Gatchalian KM, Bickel WK. Executive dysfunction in addiction. In: MacKillop J, de Wit H, editors. *The Wiley-Blackwell handbook of addiction psychopharmacology*. Oxford: Wiley-Blackwell; 2013. p. 27–62.
- Jentsch JD, Taylor JR. Impulsivity resulting from frontostriatal dysfunction in drug abuse: Implications for the control of behavior by reward-related stimuli. *Psychopharmacology*. 1999; 146(4):373–390. DOI: 10.1007/PL00005483 [PubMed: 10550488]
- Johansen EB, Killeen PR, Russell VA, Tripp G, Wickens JR, Tannock R, ... Sagvolden T. Origins of altered reinforcement effects in ADHD. *Behavioral and Brain Functions*. 2009; 5:7. <https://doi.org/10.1186/1744-9081-5-7>. [PubMed: 19226460]
- Kable JW, Glimcher PW. The neural correlates of subjective value during intertemporal choice. *Nature Neuroscience*. 2007; 10(12):1625–1633. DOI: 10.1038/nn2007 [PubMed: 17982449]
- Kable JW, Glimcher PW. An “as soon as possible” effect in human intertemporal decision making: Behavioral evidence and neural mechanisms. *Journal of Neurophysiology*. 2010; 103(5):2513–2531. DOI: 10.1152/jn.00177.2009 [PubMed: 20181737]
- Kava R, Greenwood MRC, Johnson PR. Zucker (fa/fa) rat. *ILAR News*. 1990; 32:4–8.
- Killeen PR. Models of trace decay, eligibility for reinforcement, and delay of reinforcement gradients, from exponential to hyperboloid. *Behavioural Processes*. 2011; 87:57–63. [PubMed: 21215304]
- Koffarnus MN, Jarmolowicz DP, Mueller ET, Bickel WK. Changing discounting in the light of the competing neurobehavioral decision systems theory. *Journal of the Experimental Analysis of Behavior*. 2013; 99(1):32–57. DOI: 10.1002/jeab.2 [PubMed: 23344987]
- Lattal KA. Delayed reinforcement of operant behavior. *Journal of the Experimental Analysis of Behavior*. 2010; 93(1):129–139. [PubMed: 20676272]
- Lerma-Cabrera J, Carvajal F, Lopez-Legarrea P. Food addiction as a new piece of the obesity framework. *Nutrition Journal*. 2016; 15(5):1–5. doi:10.1186/s12937-016-0124-6. [PubMed: 26728196]
- Maayan L, Hoogendoorn C, Sweat V, Convit A. Disinhibited eating in obese adolescents is associated with orbitofrontal volume reductions and executive dysfunction. *Obesity*. 2011; 19(7):1382–1387. DOI: 10.1038/oby.2011.15 [PubMed: 21350433]
- McClure SM, Ericson KM, Laibson DI, Loewenstein G, Cohen JD. Time discounting for primary rewards. *Journal of Neuroscience*. 2007; 27(21):5796–5804. <http://dx.doi.org/10.1523/JNEUROSCI.4246-06.2007>. [PubMed: 17522323]
- McClure SM, Laibson DI, Loewenstein G, Cohen JD. Separate neural systems value immediate and delayed monetary rewards. *Science*. 2004; 306(5695):503–507. DOI: 10.1126/science.1100907 [PubMed: 15486304]

- Monterosso J, Ainslie G. The behavioral economics of will in recovery from addiction. *Drug and Alcohol Dependence*. 2007; 90(Suppl 1):S100–111. DOI: 10.1016/j.drugalcdep.2006.09.004 [PubMed: 17034958]
- Monterosso JR, Ainslie G, Xu J, Cordova X, Domier CP, London ED. Frontoparietal cortical activity of methamphetamine-dependent and comparison subjects performing a delay discounting task. *Human Brain Mapping*. 2007; 28(5):383–393. DOI: 10.1002/hbm.20281 [PubMed: 16944492]
- Phillips MS, Liu Q, Hammond HA, Dugan V, Hey PJ, Caskey CT, Hess F. Leptin receptor missense mutation in the fatty Zucker rat. *Nature Genetics*. 1996; 13:18–19. DOI: 10.1038/ng0596-18 [PubMed: 8673096]
- Rasmussen EB, Huskinson SL. Effects of rimonabant on behavior maintained by progressive ratio schedules of sucrose reinforcement in obese Zucker (fa/fa) rats. *Behavioural Pharmacology*. 2008; 19:735–742. DOI: 10.1097/FBP.0b013e3283123cc2 [PubMed: 18797250]
- Rasmussen EB, Lawyer SR, Reilly W. Percent body fat is related to delay and probability discounting for food in humans. *Behavioural Processes*. 2010; 83(1):23–30. DOI: 10.1016/j.beproc.2009.09.001 [PubMed: 19744547]
- Rasmussen EB, Reilly W, Hillman C. Demand for sucrose in the genetically obese Zucker (fa/fa) rat. *Behavioural Processes*. 2010; 85:191–197. <http://dx.doi.org/10.1016/j.beproc.2010.07.008>. [PubMed: 20674704]
- Rollins BY, Dearing KK, Epstein LH. Delay discounting moderates the effect of food reinforcement on energy intake among non-obese women. *Appetite*. 2010; 55(3):420–425. DOI: 10.1016/j.appet.2010.07.014 [PubMed: 20678532]
- Sgro JA, Dyal JA, Antastasio EJ. Effects of constant delay of reinforcement on acquisition asymptote and resistance to extinction. *Journal of Experimental Psychology*. 1967; 73:634–636. [PubMed: 6034024]
- Stafford D, Branch MN. Effects of step size and break-point criterion on progressive-ratio performance. *Journal of the Experimental Analysis of Behavior*. 1998; 70:123–138. [PubMed: 9768504]
- Stafford D, LeSage MG, Glowa JR. Progressive-ratio schedules of drug delivery in the analysis of drug self-administration: A review. *Psychopharmacology*. 1998; 139(3):169–184. DOI: 10.1007/s002130050702 [PubMed: 9784071]
- Tatham TA, Zurn KR. The Med-PC experimental apparatus programming system. *Behavior Research Methods, Instruments, and Computers*. 1989; 21:294–302.
- Tomasi D, Volkow ND. Striatocortical pathway dysfunction in addiction and obesity: Differences and similarities. *Critical Reviews in Biochemistry and Molecular Biology*. 2013; 48(1):1–19. DOI: 10.3109/10409238.2012.735642 [PubMed: 23173916]
- Tomasi D, Wang GJ, Wang R, Caparelli EC, Logan J, Volkow ND. Overlapping patterns of brain activation to food and cocaine cues in cocaine abusers: Association to striatal D2/D3 receptors. *Human Brain Mapping*. 2015; 36(1):120–136. DOI: 10.1002/hbm.22617 [PubMed: 25142207]
- Towa S, Kuwahara M, Tsubone H. Characteristics of autonomic nervous function in Zucker-fatty rats: Investigation by power spectral analysis of heart rate variability. *Experimental Animals*. 2004; 53:137–144. [PubMed: 15153676]
- van Haaren F. Response acquisition with fixed and variable delays of reinforcement in male and female Wistar rats. *Physiology and Behavior*. 1992; 52(4):767–772. DOI: 10.1016/0031-9384(92)90412-U [PubMed: 1409951]
- Verdejo-García A, Pérez-Expósito M, Schmidt-Río-Valle J, Fernández-Serrano M, Cruz F, Pérez-García M, ... Campoy C. Selective alterations within executive functions in adolescents with excess weight. *Behavior and Psychology*. 2009; 18(8):1572–1578. DOI: 10.1038/oby.2009.475
- Volkow N, Baler RD. NOW vs LATER brain circuits: Implications for obesity and addiction. *Trends in Neuroscience*. 2015; 38(6):345–352. <http://dx.doi.org/10.1016/j.tins.2015.04.002>.
- Weller RE, Cook EW III, Avsar KB, Cox JE. Obese women show greater delay discounting than healthy-weight women. *Appetite*. 2008; 51(3):563–569. <http://dx.doi.org/10.1016/j.appet.2008.04.010>. [PubMed: 18513828]

- Westerveld D, Yang D. Through thick and thin: Identifying barriers to bariatric surgery, weight loss maintenance, and tailoring obesity treatment for the future. *Surgery Research and Practice*. 2016; 1:1–7. <http://dx.doi.org/10.1155/2016/8616581>.
- Wilkenfield J, Nickel M, Blakely E, Poling A. Acquisition of lever-press responding in rats with delayed reinforcement: A comparison of three procedures. *Journal of the Experimental Analysis of Behavior*. 1992; 58(3):431–443. [PubMed: 16812674]
- Zhang L, Rashad I. Obesity and time preference: The health consequences of discounting the future. *Journal of Biosocial Science*. 2008; 40(1):97–113. DOI: 10.1017/S0021932007002039 [PubMed: 17517169]
- Zucker LM, Zucker TF. Fatty, a new mutation in the rat. *The Journal of Heredity*. 1961; 52:275–278.

Fig. 1.

Total lever presses (y-axis) during each of the three acquisition sessions (x-axis) in obese (closed circle) and lean (open circle) rats. Error bars show one *SEM*.

Fig. 2. Cumulative records of lever pressing (y axis) over time (x-axis) for each rat (rows) in the obese (left column) and lean (right column) Zucker rats. Resets show the beginning of each new session.

Fig. 3.

Duration of additional delays. Duration of the delay beyond the programmed 17.5 s (y-axis; note natural log scale) for each rat (circles) in the obese and lean groups (x-axis). Horizontal lines indicate the mean, and error bars show one *SEM*.

Fig. 4.

Progressive ratio breakpoints for immediate reinforcement. Data points show each individual's mean breakpoint (over the final six sessions of the baseline phase), horizontal lines show the group mean baseline breakpoints, and error bars show one *SEM*.

Fig. 5. Mean breakpoints across delays by groups. Mean breakpoint (y-axis) at each delay (x-axis) for the obese (closed circles) versus lean (open circles) rats. Error bars show one *SEM*.

Fig. 6.

Breakpoints across delays for individual subjects. Breakpoint (y-axis) at each delay (x-axis) for rats in the obese (left two columns) and lean (right two columns) groups. Error bars show one standard deviation.

Fig. 7.

Interactive effects of delay and ratio requirement on mean overall rate. Overall rate is shown in the top row, run rate is shown in the middle row, and postreinforcement pause (PRP) is shown in the bottom row for rats in the obese (left column) and lean (right column) groups.