Published in final edited form as:

Appetite. 2010 October ; 55(2): 363–366. doi:10.1016/j.appet.2010.06.006.

Parent–child concordance of Taq1 A1 allele predicts similarity of parent–child weight loss in behavioral family-based treatment programs[☆]

Leonard H. Epstein^{a,b,*}, Kelly K. Dearing^a, and Richard W. Erbe^a

^a School of Medicine and Biomedical Sciences, University at Buffalo, United States

^b Department of Pediatrics, School of Medicine and Biomedical Sciences, University at Buffalo, Farber Hall, Room G56, 3435 Main Street, Building #26, Buffalo, NY 14214-3000, United States

Abstract

Family-based treatments show positive relationships between parent and child weight losses. One mechanism for similar parent–child changes may be a common genetic predisposition to respond similarly to a structured weight loss program. We examined whether concordance of the Taq1 A1 allele of the dopamine D2 receptor (DRD2) predicts similarities in zBMI change in 26 families with obese parents and overweight/obese 8–12-year-old children. Results showed a relationship between parent and child zBMI change over 6 and 12 months ($r_s = .69, .77, p_s < 0.001$), and concordance for the number of Taq1 A1 alleles predicted the similarity in parent and child weight loss at 6 ($p = 0.003$) and 12 ($p = 0.025$) months. These results show concordance of the Taq1 A1 allele of the DRD2 between parents and children may be one mechanism for the similar response to family-based treatments within families.

Keywords

Food reinforcement; Weight loss; Obesity; Children; Dopamine; Dopamine receptor

Family-based behavioral treatments for pediatric obesity are associated with strong positive relationships between parent and child weight losses (Epstein, Valoski, Kalarchian, & McCurley, 1995; Epstein, Wing, Steranchak, Dickson, & Michelson, 1980; Wrotniak, Epstein, Paluch, & Roemmich, 2004; Wrotniak, Epstein, Paluch, & Roemmich, 2005). Parental modeling is a behavioral factor that predicts parent and child weight change (Wrotniak et al., 2005). As parents make behavior changes necessary for their weight loss, they are modeling healthy behaviors for their children (Wrotniak et al., 2005). The focus on the shared family environment in family-based behavioral interventions may simultaneously influence both parent and child behavior change.

Parents and children not only share environments, but they have genes in common, and these common genes may be related to behavioral phenotypes that make it easy or hard to lose weight. One behavioral phenotype that is related between overweight parents and

[☆]Appreciation is expressed to Sarah J. Salvy, Brian H. Wrotniak, Colleen Kilanowski and Tinuke Oluyomi for assisting in the family-based behavioral intervention, to Paul Juzdowski and Robbert Salis for technical assistance in genotyping, and to David Allison, Myles Faith, Lara Sucheston and Rocky Paluch for statistical advice. Dr. Epstein is a consultant to Kraft foods and NuVal/ONQI. The other authors do not have any potential conflict of interests. This research was funded in part by grants from the National Institute of Drug Abuse, R01HD39778, the Foundation for Healthy Living and Kaleida Foundation, awarded to Dr. Epstein.

*Corresponding author. LHENET@acsu.buffalo.edu, lhenet@buffalo.edu (L.H. Epstein).

children is the reinforcing value of food (Epstein, Dearing, Temple, & Cavanaugh, 2008). High levels of food reinforcement are associated with increased food consumption (Epstein, Temple, et al., 2007; Epstein et al., 2004a, 2004b; Temple et al., 2009), elevated body weight (Saelens & Epstein, 1996; Temple, Leigerski, Giacomelli, Salvy, & Epstein, 2008), and risk for weight gain in young children (Hill, Saxton, Webber, Blundell, & Wardle, 2009).

One determinant of food reinforcement is dopaminergic activity (Berridge, 1996; Berridge & Robinson, 1998; Kelley & Berridge, 2002). The dopamine genotype that has been most studied in relationship to food reinforcement is the dopamine D2 receptor (DRD2) genotype (Berridge, 1996; Epstein, Leddy, Temple, & Faith, 2007). Obese individuals have a reduced density of D2 receptors (Wang et al., 2001) and an increased frequency of the Taq1 A1 allele of the DRD2 genotype (Comings et al., 1993; Epstein, Temple, et al., 2007; Tataranni et al., 2001). Presence of the Taq1 A1 allele is associated with a 30–40% reduction in DRD2 density resulting in weaker dopamine signaling (Jonsson et al., 1999; Pohjalainen et al., 1998; Ritchie & Noble, 2003). The Taq1 A1 allele is associated with activation of brain reward centers (Stice, Spoor, Bohon, & Small, 2008), increased food reinforcement (Epstein, Temple, et al., 2007), interacts with food reinforcement to influence energy intake (Epstein, Temple, et al., 2007; Epstein et al., 2004a, 2004b), and interacts with adiposity to predict food reinforcement (Epstein, Temple, et al., 2007). The primary aim of this study was to be the first to test the hypothesis that parent–child concordance of Taq1 A1 allele for the DRD2 genotype is related to similarity in weight loss in evidence-based behavioral family-based treatment (Epstein, Myers, Raynor, & Saelens, 1998).

Methods

Participants

This study involved 26 families with obese parents (BMI ≥ 30) and overweight 8–12-year-old children (≥ 85 th BMI percentile) from a larger sample of 50 families recruited for a study on the influence of enhanced child responsibility on weight loss. Thirty-two families volunteered to provide genetic information at follow-up, with 26 families having an obese participating parent. In the larger trial, families were randomized to usual family-based treatment (FBT) (Epstein et al., 1998) or enhanced child responsibility (CRT). Inclusion criteria included a child at or above the 85th BMI percentile, one parent with a BMI of 30 or above (or above 25 with an obesity related comorbidity) willing to attend treatment meetings, and children's reading at a 3rd grade level or higher. Families were excluded if the child or parent was participating in a weight loss program, had a history of an eating disorder, had any untreated psychiatric problems, or had dietary or exercise restrictions.

Treatment procedures

Participants in the FBT condition were treated together (Epstein et al., 1998), while parents and children in the CRT condition received separate treatment. Participants in both conditions received fifteen 70-min sessions scheduled as 12 weekly sessions, 2 biweekly sessions and 1 monthly session. Treatment included the Traffic Light Diet (Epstein, 2003), a lifestyle exercise program (Epstein, Wing, Koeske, Ossip, & Beck, 1982; Epstein, Wing, Koeske, & Valoski, 1985), and behavioral therapy components that focused on self-monitoring, positive reinforcement for meeting behavior goals, stimulus control, and problem solving.

At the beginning of each treatment session, parents and children were weighed. Parents and children in both conditions met separately for 50 minutes of group behavioral treatment. Counselors held 20 minute small group sessions to identify behaviors that influenced weight

change, evaluate current goals, help problem solve behavioral challenges and establish plans for meeting that session's goals. Small groups for the FBT condition consisted of 3–4 child–parent pairs, while in the CRT condition, children or parents met in groups of 3–4. No differences in child or parent weight loss at 6 or 12 months were observed for families randomized to the treatments, or for the subset of families who provided genetic information. For purposes of analysis, the results for both treatments were combined.

Measurement

Body Mass Index was based on height and weight ($\text{BMI} = \text{kg}/\text{m}^2$). Height was measured using a Measurement Concepts & Quick Medical stadiometer (North Bend, WA), and weight was measured using a Tanita digital scale (Arlington Heights, IL). zBMI values were calculated in relationship to the population mean and standard deviation for children and adults based on age and gender (Kuczmarski et al., 2002).

DNA collection—Participants rinsed their mouth with water and spit into a plastic vial (Oragene DNA, DNA Genotek Inc., Ottawa, Canada) to provide a saliva DNA sample. If needed, participants put a small dab of sugar on the center of their tongue to increase saliva production. Parents and children each received a \$10 check for participating in the genetic testing portion of the study.

Genotyping—DNA was extracted from the samples using a commercially available genomic DNA quick preparation kit (Gentra Systems, Minneapolis, MN), yielding 20 μL of DNA at a concentration of 100–130 $\text{ng}/\mu\text{L}$. After DNA purification, each sample was stored at -20°C for later analysis. For detection of the *TaqI* A1 polymorphism in the DRD2 gene a region of 304 base pairs (bp) was amplified. The primers first described by Grandy et al. (1989) were modified to sense 5'-CCC TTC CTG AGT GTC ATC A-3' and antisense 5'-CGG CTG GCC AAG TTG TCT-3'. The presence of the amplicon was confirmed by electrophoresis on a 1% agarose gel. The restriction endonuclease *TaqI* digests the 304-bp amplicon, and subsequently the fragments are separated on a 6% polyacrylimide gel by electrophoresis. The *TaqI* A1 polymorphism in the DRD2 gene at Position 32806 T to C creates a restriction site resulting in partition of the 304 bp amplicon into fragments of 177 and 127 bp. The A1/A1 or TT variant therefore is represented by an uncut amplicon of 304 bp; the A1/A2 or TC heterozygous form digests in three fragments of 304, 177 and 127 bp; and the A2/A2 or CC variant is characterized by two fragments of 177 and 127 bp (Grandy et al., 1989). DRD2 was coded for the allele patterns of A1/A1, A1/A2 and A2/A2. Families were coded as concordant if they shared the same number of copies of the *TaqI* A1 allele (0, 1, 2), or discordant if they did not share the same number of *TaqI* A1 alleles, resulting in a dichotomous variable.

Analytic plan

One-way analysis of variance and Chi-Square tests tested for differences in the characteristics of families who participated in the genetic testing ($N = 32$) versus those who did not participate ($N = 18$), as well as to test differences in the characteristics of the 26 families who had an obese participating parent and obese child who were concordant ($N = 15$) or discordant ($N = 11$) for the *TaqI* A1 allele.

Zero order regression models assessed the relationship between parent and child zBMI change at 6 and 12 months. Hierarchical regression models determined the increase in variance accounted for by adding information about *TaqI* A1 allele concordance and the interaction of *TaqI* A1 allele concordance with parent zBMI change. Due to the effect of initial weight on weight change, and unequal distributions of racial/ethnic minority subjects, we controlled for baseline zBMI and racial/ ethnic minority status in all models, and

performed all regression models with and without racial/ethnic minorities included. One outlier (greater than 3 standard deviations from the mean) was identified in the 12-month dataset, and removed from the analysis. Hardy–Weinberg equilibrium was evaluated using a Chi-Square test (Hartl, 2000).

Results

At baseline, the average child was 10.3 ± 1.2 years of age, BMI of 28.7 ± 3.6 , and zBMI of 2.24 ± 0.28 . Ten (38.5%) of the children were male. The average participating parent was 42.0 ± 3.9 years of age, with a BMI of 38.0 ± 7.5 and zBMI of 1.98 ± 0.30 . Three (11.5%) of the parents were male. There were no significant differences in these characteristics for children or parents in families who participated in the study or in those concordant for the Taq1 A1 allele of the DRD2. Six of the children (23%) and five of the participating parents (19.2%) were racial/ethnic minorities. Ten of the children (38.5%) and eleven of the parents (42.3%) had at least one Taq1 A1 allele. There was an unequal distribution of minorities in the concordant/discordant groups, as 5/6 of the child racial/ethnic minorities were in the families discordant for the Taq1 A1 allele ($\chi^2 = 5.38$, $df = 1$, $p = 0.02$). The Chi-Square values to test for deviations from Hardy–Weinberg equation were 0.44 for children and 1.87 for adults ($ps > 0.10$).

Correlations between child and parent zBMI changes at 6 and 12 months were 0.69 ($N = 26$) and 0.77 ($N = 20$), respectively ($ps < 0.001$). Hierarchical regression models, controlling for initial zBMI and racial/ethnic minority status, showed adding information about the interaction of allele concordance $\times$ parent zBMI change increased r^2 from .502 to .659 ($p = 0.023$) at 6 months and from .597 to .745 ($p = 0.041$) at 12 months. At 6 and 12 months families discordant for the Taq1 A1 allele had parent/child zBMI correlations of 0.59 ($N = 11$) and 0.24 ($N = 9$; $\beta = 0.28$ and 0.22 , $p > 0.05$); while concordant families had relationships of 0.78 ($N = 15$) and 0.89 ($N = 11$; $\beta = 0.93$ and 1.01 , $ps < 0.001$). To further evaluate the role of racial/ethnic minority status, we redid all analyses without the racial/ethnic minority families. The improvements in variance accounted for in predicting 0–6 months ($p < 0.05$) and 0–12 months ($p < 0.01$) months remained. At 6 and 12 months discordant families showed correlations of 0.40 ($N = 6$) and 0.13 ($N = 5$; $\beta = 0.18$ and 0.07 , respectively, $ps > 0.05$) while the concordant families showed correlations of 0.78 ($N = 14$) and 0.89 ($N = 11$; $\beta = 0.90$ and 1.01 , respectively, $ps < 0.001$).

Based on the regression models, if parents were moderately successful in weight loss (at least 1 SD above the mean zBMI change), and the parent and child were concordant for the Taq1 A1 allele, children showed double the zBMI change at 6 months ($-.352$ vs $-.175$) and over four times the change at 12 months ($-.466$ vs $-.109$) in comparison to children who were not concordant for the Taq1 A1 allele.

Discussion

Results show concordance for the Taq1 A1 allele of the DRD2 genotype is related to similarity in parent–child zBMI change for families in a family-based weight control program. Concordance was associated with an increase in the correlation between parent and child zBMI change. Beta coefficients describing the rate of change in concordant parent and child zBMI changes were approximately 1.0, while beta coefficients for discordant families did not go above 0.30. The similarity in parent and child weight change is a reliable result in family-based treatment studies (Wrotniak et al., 2004, 2005), with similarity in parent/child weight change conceptualized as a function of the parent and child living in a shared environment, and parent modeling (Wrotniak et al., 2005). Results of this study suggest a genetic influence on parent/child similarities in weight change.

One hypothesis to explain these data is that those who have the Taq1 A1 allele experience less reward from food than those without the Taq1 A1 allele (Blum, Cull, Braverman, & Comings, 1996), and differences in food reinforcement may mediate the effects of the dopamine genes on weight loss. Obese persons find food more reinforcing than lean persons, and food reinforcement interacts with the Taq1 A1 allele to predict energy intake (Epstein, Temple, et al., 2007; Epstein et al., 2004a, 2004b), and the Taq1 A1 allele is associated with increased obesity (Comings et al., 1993; Epstein, Temple, et al., 2007; Tataranni et al., 2001). The similarity in zBMI change in parents and children if they have the same DRD2 genes may be due in part to similarities in the motivation to eat, which are related in parents and children (Epstein et al., 2008). A parent and child sharing the same genotype that is related to food intake, such as the food reinforcement phenotype, may share the same response to a reduced calorie diet. Concordance of the genotype may facilitate imitation of parent behavior if the child is predisposed to that behavior pattern due to genetics.

Food reinforcement represents a complex behavioral phenomena, and previous research has shown a relationship between the Taq1 A1 allele and food reinforcement, but not a one-to-one relationship. This is relevant since if the only pathway for food reinforcement was differences in the number of Taq1 A1 alleles of the DRD2 genotype, then it would not be necessary to assess the DRD2 genotype, since knowledge of the phenotype would in effect provide knowledge of the genotype. Since there is likely to be multiple determinants of food reinforcement, with DRD2 genes being only one pathway, research is needed that assesses the DRD2 genotype and the food reinforcement phenotype.

Given the interest in the DRD2 genotype and obesity, it would have been ideal to have a larger sample size to assess whether the Taq1 A1 allele predicts weight loss. Barnard and colleagues (Barnard et al., 2009) studied hemoglobin A1c changes as a function of a vegan diet and genotype, but did not show the Taq1 A1 allele was related to A1c change in the overall sample; however, absence of the Taq1 A1 allele was related to greater weight loss in black participants. Unpublished research from our group did not show the number of Taq1 A1 alleles predicted weight loss in obese adults seeking a behavioral alternative to bariatric surgery. It is possible that adding information about food reinforcement, or food reinforcement in combination with other genes that may be related to the motivation to eat, would be stronger predictors of weight change.

Despite the unique directions this study points to, there are limitations. One is this was a supplementary study to a randomized trial, and as such genetics were not collected at study entry, and were not available for approximately 1/3 of the sample. A second limitation is that not all families had obese parents, which limited studying parent and child weight loss. It would not be appropriate to compare weight loss of obese children with non-obese parents who either were not interested in weight loss or had minimal weight to lose. Third, the racial/ ethnic minorities were more discordant for Taq1 A1 alleles than non-minority families. Previous research has suggested that some minorities may have a greater probability of having the Taq1 A1 allele (Barnard et al., 2009; Barr & Kidd, 1993; Erblich, Lerman, Self, Diaz, & Bovbjerg, 2005), but to our knowledge there is no research that has assessed differential concordance of dopamine genes in racial/ethnic minority and non-minority families. While it would have been ideal if the same proportion of racial/ethnic minority and non-minority families were concordant for Taq1 A1 alleles, analysis of the data showed the same pattern of results with and without the racial/ethnic minority families. Fourth, we did not collect data on concordance of food reinforcement, a behavioral phenotype related to the DRD2 genotype. While we have shown concordance of the food reinforcement phenotype in previous studies (Epstein et al., 2008), it would be important to identify concordance of the behavioral phenotype and genotype in the same study before we

can be confident a potential behavioral mechanism leading to similar parent/child responses is food reinforcement.

Research is needed with larger sample sizes, and more racial/ ethnic minority families with the same distribution of Taq1 A1 alleles as non-minority families to be confident that similarity in parent–child weight loss can be attributed to concordance of the DRD2 genotype. Future research should identify whether the current observations are due to the food reinforcement phenotype or other behavioral phenotypes that are shared within families. Other behavioral phenotypes may be shared within families, and other genotypes can be identified that are related to the shared behavioral phenotypes. As this type of research progresses, it will be important to determine whether these additional genotypes add to the prediction of the similarity in parent and child weight loss in family-based treatment programs.

References

- Barnard ND, Noble EP, Ritchie T, Cohen J, Jenkins DJ, Turner-McGrievy G, et al. D2 dopamine receptor Taq1A polymorphism, body weight, and dietary intake in type 2 diabetes. *Nutrition* 2009;25:58–65. [PubMed: 18834717]
- Barr CL, Kidd KK. Population frequencies of the A1 allele at the dopamine D2 receptor locus. *Biological Psychiatry* 1993;34:204–209. [PubMed: 8399816]
- Berridge KC. Food reward: brain substrates of wanting and liking. *Neuroscience and Biobehavioral Reviews* 1996;20:1–25. [PubMed: 8622814]
- Berridge KC, Robinson TE. What is the role of dopamine in reward: hedonic impact, reward learning, or incentive salience? *Brain Research. Brain Research Reviews* 1998;28:309–369. [PubMed: 9858756]
- Blum K, Cull JG, Braverman ER, Comings DE. Reward deficiency syndrome. *American Scientist* 1996;84:132–145.
- Comings DE, Flanagan SD, Dietz G, Muhleman D, Knell E, Gysin R. The dopamine D2 receptor (DRD2) as a major gene in obesity and height. *Biochemical Medicine and Metabolic Biology* 1993;50:176–185. [PubMed: 8260195]
- Epstein, LH. Development of evidence-based treatments for pediatric obesity. In: Kazdin, AE.; Weisz, JR., editors. *Evidence-based psychotherapies for children and adolescents*. New York: Guilford Publications, Inc; 2003. p. 374–388.
- Epstein LH, Dearing KK, Temple JL, Cavanaugh MD. Food reinforcement and impulsivity in overweight children and their parents. *Eating Behaviors* 2008;9:319–327. [PubMed: 18549991]
- Epstein LH, Leddy JJ, Temple JL, Faith MS. Food reinforcement and eating: a multilevel analysis. *Psychological Bulletin* 2007;133:884–906. [PubMed: 17723034]
- Epstein LH, Myers MD, Raynor HA, Saelens BE. Treatment of pediatric obesity. *Pediatrics* 1998;101:554–570. [PubMed: 12224662]
- Epstein LH, Temple JL, Neaderhiser BJ, Salis RJ, Erbe RW, Leddy JJ. Food reinforcement, the dopamine D2 receptor genotype and energy intake in obese and non-obese humans. *Behavioral Neuroscience* 2007;121:877–886. [PubMed: 17907820]
- Epstein LH, Valoski AM, Kalarchian MA, McCurley J. Do children lose and maintain weight easier than adults: a comparison of child and parent weight changes from six months to ten years. *Obesity Research* 1995;3:411–417. [PubMed: 8521160]
- Epstein LH, Wing RR, Koeske R, Ossip DJ, Beck S. A comparison of lifestyle change and programmed aerobic exercise on weight and fitness changes in obese children. *Behavior Therapy* 1982;13:651–665.
- Epstein LH, Wing RR, Koeske R, Valoski A. A comparison of lifestyle exercise, aerobic exercise and calisthenics on weight loss in obese children. *Behavior Therapy* 1985;16:345–356.
- Epstein LH, Wing RR, Steranchak L, Dickson B, Michelson J. Comparison of family based behavior modification and nutrition education for childhood obesity. *Journal of Pediatric Psychology* 1980;5:25–36. [PubMed: 7452420]

- Epstein LH, Wright SM, Paluch RA, Leddy J, Hawk LW, Jaroni JL, et al. Food hedonics and reinforcement as determinants of laboratory food intake in smokers. *Physiology and Behavior* 2004a;81:511–517. [PubMed: 15135024]
- Epstein LH, Wright SM, Paluch RA, Leddy JJ, Hawk LW Jr, Jaroni JL, et al. Relation between food reinforcement and dopamine genotypes and its effect on food intake in smokers. *American Journal of Clinical Nutrition* 2004b;80:82–88. [PubMed: 15213032]
- Erblich J, Lerman C, Self DW, Diaz GA, Bovbjerg DH. Effects of dopamine D2 receptor (DRD2) and transporter (SLC6A3) polymorphisms on smoking cue-induced cigarette craving among African-American smokers. *Molecular Psychiatry* 2005;10:407–414. [PubMed: 15381926]
- Grandy DK, Litt M, Allen L, Bunzow JR, Marchionni M, Makam H, et al. The human dopamine D2 receptor gene is located on chromosome 11 at q22-q23 and identifies a TaqI RFLP. *American Journal of Human Genetics* 1989;45:778–785. [PubMed: 2573278]
- Hartl, DL. A primer of population genetics. Sunderland, MA: Sinauer Associates; 2000.
- Hill C, Saxton J, Webber L, Blundell J, Wardle J. The relative reinforcing value of food predicts weight gain in a longitudinal study of 7–10-y-old children. *American Journal of Clinical Nutrition* 2009;90:276–281. [PubMed: 19535428]
- Jonsson EG, Nothen MM, Grunhage F, Farde L, Nakashima Y, Propping P, et al. Polymorphisms in the dopamine D2 receptor gene and their relationships to striatal dopamine receptor density of healthy volunteers. *Molecular Psychiatry* 1999;4:290–296. [PubMed: 10395223]
- Kelley AE, Berridge KC. The neuroscience of natural rewards: relevance to addictive drugs. *Journal of Neuroscience* 2002;22:3306–3311. [PubMed: 11978804]
- Kuczmarski, RJ.; Ogden, CL.; Guo, SS.; Grummer-Strawn, LM.; Flegal, KM.; Mei, Z., et al. CDC growth charts for the United States: methods and development. *Vital Health Statistics. Series 11. Vol. 246.* Hyattsville, MD: National Center for Health Statistics; 2002. p. 1-90.
- Pohjalainen T, Rinne JO, Nagren K, Lehtikainen P, Anttila K, Syvalahti EK, et al. The A1 allele of the human D2 dopamine receptor gene predicts low D2 receptor availability in healthy volunteers. *Molecular Psychiatry* 1998;3:256–260. [PubMed: 9672901]
- Ritchie T, Noble EP. Association of seven polymorphisms of the D2 dopamine receptor gene with brain receptor-binding characteristics. *Neurochemistry Research* 2003;28:73–82.
- Saelens BE, Epstein LH. The reinforcing value of food in obese and non-obese women. *Appetite* 1996;27:41–50. [PubMed: 8879418]
- Stice E, Spoor S, Bohon C, Small DM. Relation between obesity and blunted striatal response to food is moderated by TaqIA A1 allele. *Science* 2008;322:449–452. [PubMed: 18927395]
- Tataranni PA, Baier L, Jenkinson C, Harper I, Del Parigi A, Bogardus C. A Ser311Cys mutation in the human dopamine receptor D2 gene is associated with reduced energy expenditure. *Diabetes* 2001;50:901–904. [PubMed: 11289060]
- Temple JL, Bulkley AM, Badawy RL, Krause N, McCann S, Epstein LH. Differential effects of daily snack food intake on the reinforcing value of food in obese and nonobese women. *American Journal of Clinical Nutrition* 2009;90:304–313. [PubMed: 19458018]
- Temple JL, Legerski CM, Giacomelli AM, Salvy SJ, Epstein LH. Overweight children find food more reinforcing and consume more energy than do nonoverweight children. *American Journal of Clinical Nutrition* 2008;87:1121–1127. [PubMed: 18469229]
- Wang GJ, Volkow ND, Logan J, Pappas NR, Wong CT, Zhu W, et al. Brain dopamine and obesity. *Lancet* 2001;357:354–357. [PubMed: 11210998]
- Wrotniak BH, Epstein LH, Paluch RA, Roemmich JN. Parent weight change as a predictor of child weight change in family-based behavioral obesity treatment. *Archives of Pediatric and Adolescent Medicine* 2004;158:342–347.
- Wrotniak BH, Epstein LH, Paluch RA, Roemmich JN. The relationship between parent and child self-reported adherence and weight loss. *Obesity Research* 2005;13:1089–1096. [PubMed: 15976152]