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Purdue Ingestive Behavior Research Center Symposium 2007: Influences on Eating and Body Weight over the Lifespan – Childhood and Adolescence

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The papers in this special issue are based on presentations made at the second biannual symposium sponsored by the Ingestive Behavior Research Center of Purdue University. The topic of this symposium was: “Influences on Eating and Body Weight over the Lifespan – Childhood and Adolescence”. In the past decade, obesity has moved from our peripheral vision to front and center as we have all been taken aback by the realization that our population has become alarmingly obese or overweight, a trend which now extends globally. Maybe even more startling regarding our future has been the more recent observation that increased obesity and associated diseases are overtaking our youth. The characterization of increased obesity at the population level as being recent – occurring mainly in the last 30 or so years – has led to the argument that it must be due largely to the “obesogenic” environment that has evolved over a similar time frame, rather than to accumulation of gene mutations, which is too slow a process to account for the rapidity of this trend. Of course however, there must be some interaction of the obesogenic environment with each person’s behavior and physiology, which are partly inherited, as many people have maintained a healthy body mass index (BMI) in this environment.

Based on the suspected dominant role of environment, one strategy for dealing with the obesity epidemic/pandemic has been to identify the key environmental variables promoting weight gain and to determine how they interact with our various biologies to produce (or fail to produce) obesity and associated disorders such as the metabolic syndrome. This theme was examined fairly broadly in the first Purdue IBRC symposium in 2005, involving sessions on dietary influences, development, physiological systems and learning. Given the urgency of intervening in the rapidly increasing obesity of our youth, the goal of the present symposium was to focus on development. In particular, we examined how different environmental variables influence eating behavior and weight regulation of children and adolescents, and how these variables might alter various physiological systems to support these influences. Additionally, a session on eating disorders was included because these disorders typically involve aberrant ingestive behaviors that begin during adolescence and the predisposition to develop disordered eating may be present at earlier ages. Further, it is possible that direct comparison of symptoms, etiologies and treatments of obesity and eating disorders may provide new insights based on both their similarities and their differences. For instance, could a better understanding of the neural pathways and behaviors that support the near starvation

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of anorexia be used to oppose the mechanisms that drive the hyperphagia that contributes to obesity?

Our aim was to explore how different experimental approaches can be integrated to identify (a) environmental influences on developing controls of food intake (b) mechanisms (e.g., developmental, physiological, neural, behavioral, social, and cultural) that underlie ingestive behaviors in childhood and adolescence and (c) factors contributing to the development and maintenance of obesity and eating disorders in children and adolescents, including approaches to prevent disordered eating. Participants in the symposium were encouraged to explore links between (1) their work and the research of other participants, (2) human and animal studies, and (3) the several disciplines involved in addressing the complex and multi-factorial nature of obesity and eating disorders (e.g., psychology, psychiatry, neuroscience, physiology, nutrition and genetics). Addressing these relationships could lead to a better understanding of mechanisms underlying obesity and eating disorders in our youth and to development of treatments. The results of these explorations during the symposium are reflected in the papers collected in this issue.

Introduction. Influences on obesity and eating disorders in children and adolescents

In the introductory presentation [1], Linda Adair reviewed the epidemiological data on childhood and adolescent obesity pointing out the increased overweight (17%) and increased at risk to become overweight (16%) over the last 5 years of NHANES data (1999–2004). Potentially very disturbing was a sharp increase in weight in the 2–5 year age group, which has been reflected anecdotally in the difficulty many parents have encountered fitting their toddlers into standard car seats. Differences in obesity prevalence associated with race/ethnicity and socioeconomic status were also discussed. For example, the highest overall overweight prevalence was observed in African and Mexican American youth. Further, Linda Adair described the susceptibility to developing obesity that occurs during specific stages of development, including the prenatal period, infancy, mid-childhood and adolescence, and considered some of the influential factors unique to each of these stages. For example, prenatal exposure to over- or undernutrition, rapid growth in early infancy, an early adiposity rebound in childhood, and early pubertal development have all been implicated in the development of obesity. She also considered the importance of studying weight trajectories, as deviations in weight that occur during one stage of development can influence or interact with factors operating at subsequent stages. Finally, some new or controversial factors contributing to development of obesity at various stages were reviewed, including the protective effect of breastfeeding and the role of a child's temperament in influencing parents feeding strategies.

Session I. Environmental influences on obesity and eating disorders in children and adolescents

Susanne Ozanne elaborated on Linda Adair's review of how reduced, increased, or imbalanced growth during gestation and early postnatal life due to abnormal perinatal environments (e.g., malnutrition, maternal diabetes) can result in the permanent programming of physiological systems that predisposes individuals to develop obesity and diabetes [2]. In particular, she reviewed the recent work she and her colleagues have conducted to better understand the mechanisms underlying this programming, based largely on the rodent model of undernutrition produced by feeding pregnant dams a low protein diet. A major focus has been on alterations in insulin metabolism, some of which are often associated with Type 2 diabetes, including abnormal function of pancreatic b-cells and insulin resistance. In particular, specific changes in the expression of proteins involved in pancreatic b-cell development, insulin secretion and

post- insulin receptor signaling have been found in adult rodents that had experienced low birth weights. Importantly, some of these same signaling protein alterations observed in rodent muscle and fat tissue also occur in men of low birth weight. Finally, Susan Ozanne described recent work aimed at understanding how events that occur at birth are “remembered” or expressed later in life as diabetes or predisposition to develop obesity, possibly involving epigenetic mechanisms or altered mitochondrial function.

Next, Julie Mennella began by discussing the role of environmental factors on the development of taste preferences, making some important points about [3]. For example, our initial taste preferences as infants are for sweet tasting foods, with a tendency to reject foods that are bitter such as vegetables, partly because bitter taste is often associated with toxic substances in foods. Therefore, preferences for bitter tasting foods is largely learned, which appears to require experience tasting a food. Interestingly, this experience can begin very early through exposure to substances in amniotic fluid, breast milk and formula. Thus, important early environmental factors that impact an infant’s food preferences can be influenced by parents. Julie Mennella then described several factors identified in her experiments as influencing whether dietary experience could change an infants’ acceptance of a fruit or vegetable, including the flavors of foods experienced, whether the experience occurred between- or within-meals or both, and whether the target food was a fruit or vegetable. For example, experience with the target fruit or a variety of other fruits increased intake of that fruit, but not of a green vegetable, whereas experience with that vegetable, or a variety of other vegetables did increase intake of that green vegetable. These findings demonstrate one way in which environmental influences can be harnessed to modify behavior, resulting in healthier eating habits. Moreover, the increased intake of target foods following exposure is particularly significant given the typically low consumption of these foods by children and the difficulty in enhancing acceptance beyond the toddler – child transition.

Jennifer Fisher discussed the effects of large portions of energy-dense foods on eating behaviors in young children [4]. First, she documented the increases in average portion sizes of large numbers of foods and beverages for children two years of age or older since the late 1970’s. Previous work in adults had shown that increasing portion size resulted in increased energy intake. Research conducted by Jennifer Fisher and her colleagues have extended this finding to children and shown that it occurs not only within a single meal, but also may extends across several meals. Moreover, under some experimental conditions, both energy density and portion size contributed to increased energy intake and their effects were additive. Interestingly, in single meal experiments increased intake of the food offered as a large portion size was not compensated for by reduced intake of other foods offered in fixed portions, suggesting satiation was altered by portion size. Possible mechanisms mediating the increased consumption associated with larger portions were discussed, focusing on the role of visual cues and their potential effects on feeding microstructure (e.g., bite size and frequency). In fact, Jennifer Fisher and her colleagues found that bite size is increased when portion size is increased. Therefore, they have started examining these and other microstructural variables in a natural setting and are trying to better identify the cognitive perceptual factors involved. Despite documenting the effects of portion size on intake, the relationship to weight status is not clear and needs further investigation. Finally, strategies to offset the intake promoting effects of portion size were discussed. For instance, when children self-selected their own food portion size, their consumption was reduced.

Carol Boushey described various aspects of the role of television watching, videos, computer games and activity in child and adolescent obesity [5]. She pointed out that televisions and related media are present in virtually all homes and even in the bedrooms of over two-thirds of adolescents, resulting in significant time spent in sedentary behaviors. The television is on most of the time in about half of all homes and children/adolescents spend up to 4 hours a day

watching TV or related media. And in fact, relationships between the amount of time watching television and increased body fat have been established for children and adolescents, an effect which is exacerbated by a more sedentary life style or a higher fat diet. Moreover, in addition to the displacement of time spent being active, factors such as increased snacking during television viewing and thus increased total daily food intake were seen to contribute to this relationship. Snacking typically involved consumption of energy-dense foods resulting in increased fat intake. There is evidence that increased intake is at least partly due to advertising of snack foods. Snack food advertising far surpasses advertising of healthy foods such as fruits and vegetables. In support of these findings, an intervention study found that reducing television viewing decreased energy intake mainly by reducing food intake rather than by increasing activity. This result is counter to our current focus in research and policy and thus warrants further investigation.

Because obesity and eating disorders are multifactorial, understanding the causes and developing treatments will require interdisciplinary approaches. Theodore Wachs described a strategy for integrating numerous and disparate factors that contribute to the development of these diseases [6]. It involved a systems approach (where system is defined as “a complex of interacting elements”) that he and others have applied to a wide array of factors that influence nutritional deficiency. He also explored how this strategy could be utilized to better understand the complexities of obesity. Application of this systems approach identifies the factors that influence outcomes, and then determines how these factors are linked and the strength of these linkages. In this manner Theodore Wachs documented how family economic and food resources and particular caregiver resources, including maternal education, intelligence and depression contribute to children’s nutritional consumption. His newer contributions to this model were that child characteristics need to be included as a factor, especially child gender and temperament, and that this factor along with family and caregiver resources can be mediated or modified by other linked factors, including cultural characteristics, maternal input into family economic decisions and social support networks.

Shiriki Kumanyika discussed the role of cultural influences, involving attitudes, beliefs and behaviors on childhood obesity, especially as these influences apply to current differences among ethnic or racial populations [7]. This discussion was in many ways an elaboration of the type of model described by Theodore Wachs, involving numerous interacting factors all of which may need to be addressed to reduce obesity in these populations. In particular, Shiriki Kumanyika emphasized that cultural differences do not act in a vacuum, but interact with other ethnicity-related aspects of the environment or “context” within which they exist. Moreover, ethnic differences that may contribute to increased obesity may occur at each of the stages of development as discussed by Linda Adair. The ethnic differences were seen in increased obesity in adult females, maternal diabetes, parental attitudes and practices that can result in overfeeding children, increased intake of high calorie foods and drinks, and reduced physical activity. Many aspects of the context interact with the cultural contributions to these outcomes, including family structure, socioeconomic status, area of residence, community dynamics, occupations, health profiles and socio-political histories and experiences. For instance, location of residence and community dynamics can affect access to healthy food, to safe places to play sports, or to programs that permit or encourage other forms of exercise. Shiriki Kumanyika presented the concept of “embodiment of predisposition to obesity”, which integrates (1) biological context, (2) physical and economic environment and (3) cultural environment. Examples of these three factors include, respectively, (1) the physical response to stress, which may be greater in some ethnic populations that have been oppressed and can lead to increased blood pressure, deposition of abdominal fat, and psychological disorders, (2) the obesogenic environment, and (3) feasting traditions, normative obesity, social meanings of food and negative attitudes toward thinness.

Session II. Biological influences on obesity and eating disorders in children and adolescents

Tim Moran began the session on biological influences by describing several experiments aimed at understanding the development of obesity and diabetes in the Otsuka Long Evans Tokushima Fatty (OLETF) rat [8]. These rats have a chromosome deletion that results in loss of the CCK-1 (or CCK-A) receptor, which leads to consumption of large meals and results in obesity. Peripheral CCK-1 receptors normally activate vagal afferents that signal satiation, and thus are involved in the control of meal size. The effect of the loss of afferent activation in OLETF rats, which results in hyperphagia and obesity was reviewed. One of the newer findings presented was that CCK-1 receptors are normally also present in the dorsomedial nucleus of the hypothalamus (DMH) and are required to maintain normal levels of DMH neuropeptide Y (NPY). Consequently, in OLETF rats, the absence of the CCK-1 receptors leads to increased DMH NPY levels, which drive increased food intake. Thus, lack of CCK regulation of both peripheral (vagal sensory) and central (DMH NPY) neural pathways regulating food intake contribute to OLETF rat obesity. Recent developmental studies in OLETF rats were also described. Shortly after birth OLETF rats show satiation deficits (increased meal size), increased suckling efficiency (even when cross-fostered to a control dam), and increased weight gain. OLETF pups showed increased DMH NPY expression as early as postnatal day 15. Taken together, these results suggest that CCK regulation of meal size and food intake may be active at birth or shortly thereafter. The last experiment described examined the effects of exercise on OLETF rats. Age-dependent effects were observed for food intake, weight gain and peptide expression in the DMH. Exercise produced a fairly rapid decrease in food intake in both young and mature OLETF rats, which eventually resulted in normalization of body weight. Interestingly, when exercise was stopped the normalization was partially maintained in young rats, but not in older ones. Moreover, blood glucose regulation was also normalized in exercising OLETF rats, preventing the development of Type II diabetes. In the short-term, reduced food intake was associated with increased DMH expression of corticotrophin releasing hormone. In contrast, long-term exercise prevented increases in DMH NPY, which contributed to the long-term reduction in food intake. These studies demonstrate the utility of the OLETF model in developing interventions at critical time points that can alter the course of genetic influences on food intake and body weight.

Richard Simerly began his presentation with a review of studies with his colleagues demonstrating a role for leptin in development of hypothalamic arcuate nucleus pathways that regulate food intake and metabolism [9]. The development of some of these pathways was disrupted in leptin-deficient mice, an effect probably due to the absence of an early postnatal leptin surge. *In vitro* and gene knockout experiments suggested the leptin surge influences development of the arcuate pathways. By binding to the long form of the leptin receptor on arcuate neurons, leptin activates the ERK, rather than the STAT3 signaling cascade. Consistent with this interpretation, leptin injections in postnatal, but not in mature mice restored normal development of hypothalamic arcuate pathways. Next Richard Simerly described a more recent direction of his research, which has involved looking at the effects of early postnatal nutritional manipulations, in particular over- and undernutrition produced by varying litter size, on leptin levels and development of hypothalamic arcuate feeding pathways. Overfed neonatal mice had increased leptin levels, whereas underfed ones had reduced levels. Moreover, arcuate hypothalamic projections appeared abnormal in mice with reduced neonatal leptin levels. These findings suggest that one mechanism mediating the undesirable changes in long-term energy balance associated with abnormal perinatal nutrition involves changes in leptin secretion that result in central defects in neural systems controlling behavior and autonomic regulation.

Ed Fox presented next [10], and began by reinforcing Timothy Moran's discussion of the vagal afferent role in determining meal size by signaling satiation and regulating digestive reflexes. Next he reviewed the role of altered meal size in obesity and eating disorders, including increased meal size associated with the predisposition to develop obesity in adult rats as a consequence of perinatal overnutrition. Based on this knowledge he postulated that one way abnormal perinatal nutrition could increase meal size and lead to a predisposition to develop obesity would be through altering vagal afferent development. Such an effect might be mediated by mediated by loss of neurotrophic effects of leptin, or other molecules –similar to the mechanism underlying altered development of hypothalamic arcuate feeding pathways as discussed by Richard Simerly. As a first step toward testing this hypothesis, recent experiments that investigated the time course of vagal sensory innervation of the stomach were described. Vagal innervation first reached the stomach at mid-embryonic ages, and importantly, contrary to the common assumption that development of this innervation is largely complete at birth, key events, including mechanoreceptor formation were found to occur mainly after birth. Thus, vagal development in the GI tract could be impacted by abnormal environmental influences present postnatally as well as prenatally. Next, results from an ongoing study examining the expression of brain-derived neurotrophic factor (BDNF) were described. BDNF is a neurotrophin important for vagal sensory development. Its expression in the stomach wall exhibited spatial and temporal overlap with developing vagal sensory elements, a pattern consistent with a role in development of vagal afferent stomach innervation. Finally, results of an ongoing experiment examining the role of BDNF in vagal development were described. Vagal afferent innervation of the stomach was altered in neonatal BDNF-deficient mice. The pattern of changes included loss of one population of vagal mechanoreceptors and abnormal morphology of a second population of vagal mechanoreceptors. These alterations were consistent with reduced satiation signaling and increased meal size that are observed in mice subjected to perinatal over- or undernutrition. Future experiments will examine the effects of altered perinatal environments on development of vagal sensory innervation of the upper GI tract and on the expression of BDNF, which contributes to regulation of this development.

In the final presentation of this session, Jose Fernandez discussed the genetics of obesity [11]. First he reviewed the numerous environmental factors that influence development of obesity. He also emphasized that it is the interaction between an individual and the environment that influences obesity and related diseases and therefore an important goal should be to understand mechanisms of these interactions, especially for the diverse races and ethnicities that could have unique cultures and behaviors that differentially influence obesity. Further, Jose Fernandez suggested that the large differences in the eating behavior and weight gain of individuals that experience the same environment suggests that non-environmental factors, including genes play a role. Moreover these differences have suggested that a contribution of genetic background independent of social and financial factors underlies racial and ethnic differences in obesity and metabolic risk factors. He then described the approaches that have been employed to identify genes associated with obesity, and pointed out that although a large number of genes have been identified, only a small proportion replicated, which raises questions about their general significance. It was then argued that one major factor contributing to lack of replication may have been the heterogeneity of human populations. He and his colleagues have taken a genetic admixture approach to overcome some of the contributions to heterogeneity. This approach involved grouping of individuals according to genetic markers that reflect their ancestry, and thus reflect the gene pools and the associated gene frequencies that their genotypes were derived from. This tool will be particularly important for studies involving individuals of diverse racial and ethnic backgrounds.

Session III. Eating disorders in children and adolescents

Angela Guarda opened the session on eating disorders with a discussion of the characteristic features of anorexia [12]. She reviewed the research examining the success of different treatment approaches, and finished by describing some promising areas of research that attempt to better understand the pathophysiological mechanisms that maintain anorexia, which may lead to improved treatments. Although anorexia occurs much less frequently than obesity, there are similarities between them. These include increasing prevalence in adolescents and young adults, heterogeneity, and heritability in the form of multigenic effects modified by the environment, which may be reflected in an inherited predisposition that is unmasked by stressors. Heritable personality traits that may signify a predisposition for developing anorexia include perfectionism, harm avoidance and early neuroticism. Cognitive components of anorexia include motivated self-starvation, or an obsession with fear of fatness that is beyond reason, denial of illness and ambivalence toward treatment. As a consequence, anorexia is difficult to treat, a process which requires conversion from seeing dieting as the patient's goal to realizing it is the problem. Moreover, patients with anorexia exhibit a high drop out rate from treatment, which has, along with other methodological constraints, limited both the number of studies that have examined the efficacy of different treatments and the ability to draw conclusions from these studies. The results of this body of research have suggested that family therapy (e.g., Maudesly) has been more successful than individual therapy for young patients that have been anorexic for less than three years. In contrast, inpatient therapy involving a specialized eating disorders treatment facility has been most successful for older or more chronic patients. Such facilities are better able to help patients achieve weight restoration – a factor associated with a reduction in relapse rate – and to utilize peer pressure through group therapy. Regarding the pathophysiology of anorexia, Angela Guarda reviewed the classic findings, for example, the starvation associated with anorexia alters hunger and satiety as well as the underlying physiological processes such as gastric emptying. One aspect that recent research has focused on is the role of pancreatic polypeptide (PP) hormone that reduces appetite and food intake. This hormone is maintained at high levels in anorexic patients and normalizing PP levels might be especially important for preventing relapse. Improved understanding of these and other hormonal and physiological alterations that occur in anorexic patients may provide targets for treatment.

Walter Kaye began his presentation by expanding on Angela Guarda's description of the traits associated with anorexia, which aided understanding their relationship to various brain circuits and neurotransmitter systems [13]. For instance, he added that dieting is rewarding for anorexic patients, but othewise they do not respond well to reward or pleasure. Moreover, they are driven in terms of exercise and achievement, they exhibit dysphoric mood, obsessive-compulsiveness, conformity and a failure to learn from mistakes. He also distinguished anorexia and bulimia, pointing out that while they appear to be related disorders sharing many features, there are differences in changes in brain function associated with each, suggesting they might have a different neurological basis. Walter Kaye also discussed the importance of focusing on brain imaging data from recovered patients because the malnutrition associated with anorexia affects the function of many neurotransmitter systems. He reviewed several imaging studies from his group and other labs that indicated altered functioning of neural circuits and transmitter systems in association with anorexia and bulimia, in particular the serotonergic and dopaminergic systems. These transmitters had been implicated in eating disorders because their metabolites are found at abnormal levels in cerebrospinal fluid of ill and recovered patients that correlate with some of the symptoms. For example, the anxiety-reducing effects of dieting were related to a reduction in 5-HT neurotransmission, an abnormality that may be present prior to disease onset. Striatal dopaminergic deficits that were identified in patients may relate to several symptoms including reduced food ingestion and alterations in both reward and affect. An example of one of the unifying concepts to emerge from this body of work is that several regions

of the ventral limbic system, which are important for identifying the emotional significance of a stimulus and generating the affective response to it, exhibit signs of altered dopamine and serotonin regulation. Moreover, in some of the first studies examining brain activation in response to food consumption, reduced activity was observed in limbic regions directly related to evaluation of food stimuli, including the primary taste cortex in the insula, adjacent frontal operculum, orbitofrontal cortex and basal ganglia regions connected to the insula that are involved in evaluating food reward. These studies demonstrate that it may be possible to identify brain regions and neurotransmitter functions altered in association with anorexia and bulimia, to single out those that predate disorder onset and to identify possible sites of treatment.

Patricia Faris concluded this final session by describing studies performed with her colleagues, developing and evaluating their theories about the involvement of sensory vagus nerve transmission in bulimia nervosa [14]. In particular, they have hypothesized that the binge-eating and vomiting used by some women as a means for controlling their weight produces an increase in vagal tone or firing rate. They suggest that with continued experience the vagus nerve spontaneously fires at an increased rate in cycles that correspond to - and may underlie - the bulimic episodes. A number of studies were reviewed that support this hypothesis. For instance, bulimic patients did in fact become satiated during a meal, but their threshold for achieving satiation was greater-than-normal, which could be explained by increased receptive relaxation reflex of the stomach, a reflex activated by vagal afferents. Also, they found that somatosensory pain thresholds were increased in bulimic patients, suggesting increased activity of the vagal afferents may mediate this effect. Further, employing imaging studies they found that expanding the stomach by inflating an intragastric balloon led to greater increases in activity of the parabranchial nucleus (a group of neurons in the brainstem that receives input from vagal afferents) in bulimic patients as compared with controls. They further proposed that if cyclic changes in vagal tone drive the bulimic feeding behavior, then normalizing vagal sensory activity could provide a means for reducing the behavior. This hypothesis was initially tested in humans using ondansetron, a drug that blocks serotonin receptors ($5\text{-}HT_3$ subtype) that normally activate vagal satiation signals. With encouraging results from these experiments they recently employed stimulating electrodes implanted on the cervical vagus nerve unilaterally to “pace” its activity. Vagal stimulation improved both bulimic eating behaviors as well as sensations of satiety in response to a test meal. These studies thus provide “proof of concept” data in support of the hypothesis that abnormal vagal tone is involved in bulimia and that abstinence is associated with recovery of normal meal related satiety.

Summary and Conclusions

The presentations made at this symposium along with the accompanying discussions highlighted some of the environmental factors that may be influencing food intake and weight regulation of our youth. In some instances the interactions of these factors with physiological and neural systems that mediate or maintain obesity and eating disorders were also examined. Environmental factors included meal portion size, perinatal nutrition, snacking associated with television viewing and advertising, and social/cultural influences in context. Combating the increase in number of affected youth may require addressing many if not all of these factors, as well as others that were not the focus of presentations. Further, our discussion of a systems approach made it clear that many environmental factors interact with one another and with biological factors to varying degrees, and a systems approach or other strategy that takes this into account will be necessary to fully understand the impact of each factor on development of obesity or eating disorders. Several presentations addressed the role of perinatal nutrition and maternal diabetes in permanent programming of feeding behavior and metabolism. A strong case was made for a role of perinatal influences in development of a predisposition to become obese and diabetic. Significant progress has been made in understanding the physiological mechanisms mediating obesities of developmental origin, especially those

involving insulin metabolism and formation of hypothalamic pathways regulating feeding and metabolism. Early, but apparently powerful nutritional and hormonal influences on subsequent obesity and metabolic disease reinforce the need to insure that adequate education programs on nutrition and health for pregnant women are in place and easily accessible, especially for high-risk populations. Similarly, government programs should insure that pregnant women and mothers who recently delivered and whose resources are limited have access to adequate nutrition. Recent genetic models were described that have aided our understanding of the effects of abnormal perinatal environments on neural development. For example, leptin and BDNF knockout mice exhibited alterations of development of central and peripheral feeding regulatory pathways, respectively, suggesting changes in leptin or BDNF produced by abnormal perinatal nutrition or hormone levels could similarly alter their development. Also described were the benefits of a rat genetic obesity model lacking CCK-1 receptors in understanding of the roles peripheral vagal and central hypothalamic pathways in both young and adult rats, as well as for examining interactions between genes and environment. For example, it was found that exercise limited to an early age, but not a later age, had lasting preventative effects on weight gain – a finding that could have important implications for treating obesity in children. Other promising approaches for developing therapies were also presented. These approaches ranged from research on strategies to encourage infants to increase consumption of fruits and vegetables to use of a pharmacological agent or electrical stimulation for regulating activity of the sensory vagus nerve to control bulimia. We also saw how imaging studies are being utilized to identify brain pathways and neurotransmitter systems involved in anorexia and bulimia, which might lead to new pharmacological treatments. Thus, the body of research discussed at this symposium was promising. Many avenues of research into understanding obesity and eating disorders in children and adolescents, as well as treatment approaches, are making significant progress. As research progress continues it will benefit from increased collaborative efforts and greater integration as we continue to address the multifactorial nature of obesity and eating disorders and ultimately improve our success in prevention and treatment.

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