Family Ecologies and Child Risk for Obesity: Focus on Regulatory Processes

Special Issue Guest Editor’s Note: In this article, the authors examine how family and social system interactions regarding food and eating can increase risk of children’s obesity. They provide practice and policy recommendations to reduce risk of obesity. This article reflects the behavioral and neurobiological mechanisms linking chronic stress with pediatric obesity presented by Pervanidou and Chrousos in their article, “Stress and Pediatric Obesity: Neurobiology and Behavior” (this issue, pp. 85–93)

Abstract
Childhood obesity is a significant public health problem. The causes of obesity are complex and extend across biological and social ecologies. This article focuses on how proximal regulatory processes connect biological risk for obesity and family systems. Response to satiety, distress during feeding, organization of family routines, and exposure to food marketing are provided as examples of the complexity of risk for obesity. The article concludes with recommendations for practice and policy to prevent childhood obesity and to empower families to take an active role in their community.

According to recent estimates, 16.9% of U.S. children between 2 and 19 years of age are obese, and 31.8% are overweight or obese (Ogden, Carroll, Kit, & Flegal, 2014). Although the rates of childhood obesity appear to have leveled in the most recent reports, they are still of concern given that childhood obesity tracks to adult obesity (McCarthy et al., 2007). The health consequences of obesity include increased risk for cardiovascular disease, diabetes, and psychosocial difficulties (Adair, 2008; Beydoun & Wang, 2010; Institute of Medicine, 2005, 2011). Although the simple explanation for overweight and obesity is the consumption of more calories than are expended, the causes of childhood obesity are complex. The family system plays an important role in understanding childhood obesity—not as a source of blame but as part of a larger ecology that may support or derail children’s health.

In this overview we provide an ecological approach that integrates family systems perspectives with variations in individual biology and response to emotions that may account, in part, for increased obesity risks. We provide examples from the Synergistic Theory and Research on Nutrition and Obesity Group (STRONG) Kids program, which uses a contextual approach to understanding how where children live and play affects their dietary habits. Families are at the center of this cells-to-society model (Fiese, Bost, McBride, & Donovan, 2013; Harrison et al., 2011), serving as both the progenitor of individual biology and as the interpreter of the environment. We conclude the article with recommendations for practitioners and policy stakeholders.
FIGURE 1. Regulatory Processes and Child Obesity Risk.

A Cells-to-Society Approach to Childhood Obesity

Several ecological approaches to childhood obesity have been proposed (Birch & Anzman, 2010; Kitzman-Ulrich et al., 2010). The STRONG Kids approach outlines 6 Cs that form multiple ecologies of childhood obesity: cell, child, clan, community, culture, and country (Fiese et al., 2013; Harrison et al., 2011; see Figure 1, this article). Relevant to this report is the developmental, transactional processes between ecologies that unfold over time to support healthy eating habits and positive regulatory behaviors.

The bioecological model originally proposed by Bronfenbrenner (1977), and then expanded by Bronfenbrenner and Evans (2000), outlined not only the layers of ecologies that affect development but also the process by which competence and dysfunction evolve. These models propose that individual biology is expressed and shaped by environmental context. A key assumption is that social interactions shape the biological potential of the developing child. Developmental outcomes are determined through interaction between the biological makeup of the child and psychosocial processes in their environment (Bronfenbrenner
& Ceci, 1994). A distinction is made between proximal and distal processes experienced directly by the child. Development occurs primarily through proximal interactions that become more complex over time as the child interacts with increasingly more complex ecologies. For example, the first social interactions around feeding are relatively simple, including eye-to-eye contact during breastfeeding and the infant crying when hungry. Over time, these signals become more complex as the child acquires language and expresses preferences for particular foods. These preferences are shaped, in part, by what is available in the home, parent preferences, and individual taste exposures. These proximal processes are considered the “engines of development” (Bronfenbrenner & Evans, 2000, p. 118). What distinguishes an adaptive from maladaptive outcome is dependent on multiple factors, including the characteristics of the child, the nature and quality of the proximal interactions, the context in which the proximal interactions occur, and the amount of exposure the child may have had to either positive or negative influences on development.

Although previous bioecological models applied to childhood obesity have identified characteristics of the multiple levels of the ecologies that may be important in predicting unhealthy weight gain across childhood and into adolescence and adulthood, less attention has been paid to how the proximal interactions may extend across ecologies and result in either healthful eating behaviors or dysfunctional practices associated with obesity. In this article we take a process-oriented approach focusing on specific regulatory processes that connect ecologies and that may increase or decrease the child’s risk for obesity. Consistent with previous reports, we suggest there is no single cause of childhood obesity, but that the transactional processes connecting biological, interpersonal, and family systems may serve to support or disrupt positive outcomes for children’s health.

Biological Regulations

Genetic Regulations. There is no discounting that there are genetic and biological contributions to risk for obesity (see Pervanidou & Chrousos, 2016); however, these risks are typically modulated by the environment as genetic susceptibility to obesity will likely lead to different phenotypic outcomes because of differing exposure to and interactions within obesogenic and non-obesogenic environments over time (Rhee, Phelan, & McCaffrey, 2012). Genome-wide association studies in children and adolescents have identified more than 20 genes that place a child at risk for obesity (Salsberry & Reagan, 2010). An array of single-nucleotide polymorphisms has been identified in these vulnerability genes that, when combined together, cumulatively increase the child’s risk for unhealthy body mass index (BMI; Suglia, Duarte, Chambers, & Boynton-Jarrett, 2012). Many of these genes are thought to affect predispositions to obesity through dysregulation of metabolic hormones in the body and, for genes expressed in the brain, the dysregulation of energy intake and feelings of satiety (Wardle, Llewellyn, Sanderson, & Plomin, 2009).

Response to satiety may be one of the behavioral regulatory processes that mediates genetic risk for obesity and unhealthy weight gain in childhood. In a large sample of 10-year-old children, Llewellyn and colleagues found that sensitivity to satiety was negatively related to genetic risk for obesity using an array of single-nucleotide polymorphisms commonly thought to represent risk for obesity (Llewellyn, Trzaskowski, Van Jaarsveld, Plomin, & Wardle, 2014). Furthermore, response to satiety mediated the relation between genetic risk and BMI. This is one example of how a regulatory process—awareness and response to internal cues associated with being full or hungry—influences energy intake and provides a link between individual biology and risk for obesity.

Microbiota. In addition to these vulnerability genes, growing scientific attention has focused on the impact of genes and genomes of human intestinal microbes on the development of obesity (A. Cox, West, & Cripps, 2014; L. Cox & Blaser, 2013). The human gut microbiota consists of around $10^{14}$ bacteria, and microbial composition has been found to play a critical role in metabolism, digestion, as well as immunoregulation—all of which are relevant to obesity-related processes (Rook, 2013; Rook, Raison, & Lowery, 2013). Several mechanisms have been proposed for the potential links between gut microbiota and obesity risk, including the extraction of energy from diet components that are indigestible (affecting glucose homeostasis), the
effects of intestinal dysbiosis on inflammation (Bervoets et al., 2013), and the critical role of gut microbiota in early programming of the hypothalamus–pituitary–adrenal axis as well as in modulating stress reactivity and behaviors (O’Mahony et al., 2009; Sudo et al., 2004). These latter points are especially crucial to the present discussion because of the cross-talk between gut microbiota and central nervous system (CNS) functioning. Unhealthy gut microbiota profiles can stimulate inappropriate gut–brain axis signaling and affects CNS functioning, and stress triggered at the CNS level can affect gut microbiota as well. In early childhood, proximal interactions within the family are crucial for developing capacities to regulate physiological stress responses, which in turn affect inflammation states and immune system functioning (Boyce, Sokolowski, & Robinson, 2012; Repetti, Robles, & Reynolds, 2011). Indeed, obesity is widely recognized as a condition of low-grade, chronic inflammation (Johnson, Milner, & Makowski, 2012).

Data support the link between gut microbiota and obesity risk. Microbial colonization has been found to differ between obese and lean adult humans as well as between obese and lean mice, primarily (but not always) showing low concentrations of intestinal Bacteroidetes and high concentrations of Firmicutes (Ley, Turnbaugh, Klein, & Gordon, 2006; Murphy et al., 2010). The data regarding children, however, are scarce, but they warrant discussion. Some findings have revealed an elevated Firmicutes:Bacteroidetes ratio in obese children when compared to lean children (Bervoets et al., 2013; Ferrer et al., 2013), and that formula-fed infants show these phyla ratios when compared to breast-fed babies (Donovan et al., 2012). In early childhood, high concentrations of the Bacteroides fragilis group during infancy have been shown to predict higher obesity risk over the first 3 years of life (Vael, Verhulst, Nelen, Goossens, & Desager, 2011). These data are promising and suggest that future studies seek to determine exactly how children’s gut microbiota change over time in response to environmental factors in ways that translate into obesity risk. This is especially important because researchers have proposed that early microbial communities play a key role in the assembly of adultlike profiles (emerging around 2 years of age) and in the epigenetic programming of stress responses and other health-related mechanisms (Li, Wang, & Donovan, 2014; Sudo et al., 2004).

Child Temperament and Self-regulation. Developmental phenomena that shape and/or are related to physiological reactivity and regulation are also becoming promising areas of inquiry in pediatric obesity. Child temperament, generally defined as inherited individual differences in reactivity and regulation across emotion, attention, and motor activity domains (Rothbart, Sheese, Rueda, & Posner, 2011), may influence eating behavior and obesity-related outcomes through its impact on stress responses, emotion, and behavioral regulation. Early and developing capacities to self-regulate eating can be influenced by distress and emotional factors because brain regions governing energy balance are also implicated in stress responses (Dallman, 2010; Herman & Polivy, 2004). Stress responses and/or emotion dysregulation, in turn, are associated with metabolic syndrome (Kyrou & Tsigos, 2009), higher energy intake in school children, and children’s BMI (Blissett, Haycraft, & Farrow, 2010; Braet & VanStrien, 1997; Nguyen-Michel, Unger, & Spruijt-Metz, 2007).

Mounting empirical data link the global temperamental dimensions of negative reactivity and self-regulation to weight-related outcomes in children. Research suggests that in early childhood, distress to limitations and negative reactivity/mood, in particular, are associated with weight gain over the first year of life (see review by Anzman-Frasca, Stifter, & Birch, 2012). Longitudinal studies have also documented relations between negative reactivity and subsequent child weight status (Slining, Adair, Goldman, Borja, & Bentley, 2009; Wells et al., 1997; Wu, Dixon, Dalton, Tudiver, & Liu, 2011), but several longitudinal cohort studies have failed to find such associations from infancy to middle childhood (Pryor et al., 2011; Wright, Cox, & Couteur, 2011). The inconsistencies in the longitudinal reports require that scholars take a more careful look at temperament, including a focus on self-regulation and potential moderating influences.

It is perhaps not surprising that more consistent findings have documented associations between the self-regulation dimension of child temperament and obesity-related outcomes (Anzman-Frasca et al., 2012). Longitudinal data reveal that several aspects of regulation (e.g.,
emotion regulation, observations of self-control, and delay of gratification) predict a wide range of child outcomes, including BMI $z$ scores, weight gain, obesity risk, and measured adiposity (Faith & Hittner, 2010; Francis & Susman, 2009; Graziano, Calkins, & Keanne, 2010; Wells et al., 1997). In one notable example, Francis and Susman (2009) analyzed child data from the National Institute of Child Health and Human Development (NICHD) Study of Early Child Care and Youth Development (SECCYD) and reported that children who had poor self-regulation at age 3 and poor delay of gratification skills at age 5 also had the highest BMI scores and the most rapid BMI gains from 3 to 12 years of age.

It is important to note that the relations between temperament dimensions and child BMI also may be moderated by parental feeding practices, maternal sensitivity, and developmental period. For example, children with low inhibitory control and whose parents have controlling feeding practices have been shown to be at a significantly higher risk for obesity than children with parents who do not have controlling feeding practices (Rollins, Loken, Savage, & Birch, 2014). Difficult infants whose mothers are insensitive have also been shown to be at a higher risk for obese or overweight status at 5 to 12 years, but not between 2 and 4 years (Wu et al., 2011). More studies clearly are needed to show how biobehavioral dispositions such as temperament interact with broader relational and environmental contexts across developmental periods to increase or decrease pediatric obesity risks. This is where we turn now.

**Attachment Relationships.** In addition to child temperament, parent–child attachment relationships are a widely recognized contributor to emerging emotional and behavioral response and regulation patterns (Bowlby, 1980), and they are likely to influence the interpersonal context through which eating behaviors are socialized. Indeed, histories of parental responsiveness to their children’s distress (including early hunger cues) contain some of the first learning experiences of dyadic stress coping and the resulting regulation of internal states. Child security has gained increasing attention in the pediatric literature because of this well-documented impact on emotion regulation, and several studies now support the notion that attachment influences obesity-related outcomes.

Using data from the Early Childhood Longitudinal Study, Birth Cohort (Bethel, Green, Kalton, & Nord, 2005), Anderson and Whitaker (2011) reported that children who were insecure at 24 months of age were significantly more likely to be obese at 4.5 years of age when compared to secure children. They also reported longitudinal data from the NICHD SECCYD linking the quality of early maternal–child relationships (at 15, 24, and 36 months) to adolescent obesity at 15 years of age. At 24 and 36 months, having low maternal sensitivity and insecure attachment put children at a (significantly) higher risk for adolescent obesity than if either were considered alone (Anderson, Gooze, Lemeshow, & Whitaker, 2012). Finally, Rhee and colleagues (Rhee, Lumeng, Appugliese, Kaciroti, & Bradley, 2006) also used the NICHD SECCYD data set to examine maternal sensitivity and child weight status. Maternal sensitivity and expectations for self-control scores at 54 months were used to create parenting categories, and child BMI was calculated (using measured height and weight) when children were in first grade. The results revealed that children whose parents had high sensitivity/high expectations for control had the lowest incidence of overweight status (3.9%), and that children of parents with low sensitivity/high expectations for control were at the highest risk for overweight status in first grade (17.1%).

As in the literature on child temperament, moderating effects have also been reported. In addition to the Wu et al. (2011) study discussed earlier, Zeller and colleagues (Zeller, Boles, & Reiter-Purtill, 2008) examined differences...
in parenting and child temperament between obese children seeking treatment and a matched comparison group. Their analyses indicated that high temperamental difficulty coupled with low maternal warmth was the only combination that significantly discriminated between children who were obese and their non-obese counterparts (see also Boles, Reiter-Purtill, & Zeller, 2013). Taken together, these findings suggest that future studies need to consider the combined effects of early attachment and temperament on subsequent obesity risks.

We should also note that although data are accumulating that document the potential connections between attachment and child weight status, the proposed mechanisms (emotion regulation, responses to distress, emotional eating) accounting for these associations have to our knowledge not been explicitly examined. However, Bost et al. (2014), using survey data from parents of preschoolers, tested a serial mediation model of the relations among parent attachment style, parental responses to their children’s distress, and children’s food consumption. The data supported the serial mediation such that parent insecure attachment style predicted responses to child distress, which in turn predicted child consumption of unhealthful (e.g., energy-dense) foods. Combining approaches that help document these processes will help specify how the nature and quality of early relationships translate into trajectories toward or away from obesity risks.

**Feeding Styles and Parenting Practices.** Feeding children is an integral part of parenting. Parenting styles (authoritarian, authoritative, permissive, indulgent) have been found to be reliable predictors of children’s food consumption and feeding behaviors. Parents vary along the dimensions of demandingness and responsiveness; those who are high on demandingness and responsiveness (authoritative) tend to provide more nutrient-rich food to their children, and parents who are low on demandingness and high on responsiveness (indulgent) tend to have children who eat fewer fruits and vegetables and more nutrient-dense foods such as those with high sugar and high fat content (Dev et al., 2013; Hughes, Power, Fisher, Mueller, & Nicklas, 2005; Hughes, Shewchuk, Baskin, Nicklas, & Qu, 2008). In terms of self-regulation, permissive and indulgent parenting styles provide little guidance to the child in regard to attending to satiety cues. If feeding occurs in a permissive or indulgent context, then the child does not have the opportunity to develop competence in regulating healthy food preferences or age-appropriate portion sizes.

One potential pathway between feeding styles and child risk for overweight/obesity is through emotional eating and response to negative emotions. As we discussed earlier, parent response to negative emotions may be a mediator of healthy food consumption. In the context of attachment relationships, we presented evidence that insecure parents who are dismissive of negative emotions have children who are more likely to eat energy-dense foods (Bost et al., 2014). Other researchers have demonstrated that children of authoritative parents are less likely to report emotional eating, and their parents are less likely to minimize negative emotions (Goulding et al., 2014). Conversely, minimizing negative emotions was related to emotional eating. The overall emotional climate of mealtimes and family interactions surrounding food may be important to consider when examining proximal processes during family routines that predict obesity-related outcomes.

**Family Routines.** Family routines provide predictability and order to the household. Repeated over time, routines comprise several key elements: the assignment of roles, expectations for attendance, the modulation of emotion, the creation of symbolic meaning, and planning (Fiese, 2006). Family routines provide a developmental function as they become more organized over time as children become more active participants and can shape the form and substance of activities (Fiese, 2007; Spagnola & Fiese, 2007; Wildenger, McIntyre, Fiese, & Eckert, 2008). Mealtime routines have been found to be associated with children’s nutritional health, including the frequency with which families share meals together (Hammons & Fiese, 2011) and the ways in which family members interact with each other (Fiese, Hammons, & Grigsby-Toussaint, 2012; Kong et al., 2013). To best understand how the proximal process of sharing regular meals together may foster self-regulatory behaviors in children, attention to social interactions is warranted.

There are at least three primary dimensions of social interaction behaviors observed during meals that appear to be related to children’s nutritional health and well-being: (a) positive
interpersonal communication, (b) adequate response to negativity, and (c) an environment that is relatively free of habitual distractions. Positive interpersonal communication can be summarized as showing real, genuine concern about another’s activities or experience. Drawn from the McMaster Model of Family Functioning (Epstein, Ryan, Bishop, Miller, & Keitner, 2003), a focus on family communication emphasizes the necessity of exchanging information in a clear and direct manner with no hidden agendas. Direct observation of family mealtime interactions has documented that families who routinely engage in positive forms of direct communication and show genuine concern about each other’s activities also have children who are less likely to be overweight or obese or engage in unhealthy eating habits (Czaja, Hartmann, Rief, & Hilbert, 2011; Fiese et al., 2012; Jacobs & Fiese, 2007).

We have already mentioned the potentially important role that response to negative emotions may play in creating a positive family food environment. It is doubtful that all family meals progress smoothly. Indeed, mealtimes may serve as one setting where response to negative emotion is socialized. When negativity is responded to in a matter-of-fact way as part of a range of emotions, then children have an opportunity to learn how to manage distress. Conversely, when negativity is associated with struggles, in particular around food, there are fewer opportunities for positive self-regulation. In a small exploratory study, Berge and colleagues reported that families who scored lower on managing affect during meals also had children who were more likely to be obese (Berge, Jin, Hannan, & Neumark-Sztainer, 2013).

An important aspect of family routines is their organizational structure and capacity to provide a sense of order in daily life. When routines are disrupted or unsettled, then chaos becomes a distinguishing feature of the child’s proximal environment. Chaotic family environments are characterized by lack of structure, frenetic activity, and unpredictability (Evans, Gonnella, Marcynyszyn, Gentile, & Salpekar, 2005). When an environment is unpredictable and disorganized, then the smooth back-and-forth interactions essential to healthy development are disrupted and strained. Chaotic environments are also often characterized by high levels of ambient noise, which also interrupts smooth communication (Evans, 2004, 2006). Relevant to our discussion is the high prevalence of habitual distractions evident in families with children who are overweight and/or obese. Talking on a cell phone during mealtimes, watching television while eating, and being away from the table have been identified as risk factors for unhealthy weight (Coon, Goldberg, Rogers, & Tucker, 2001; Fiese et al., 2012; Kong et al., 2013; Tal, Zuckerman, & Wansink, 2014).

We conceptualize these habitual distractions as contributing to obesity risk in three ways. First, being distracted by electronic media or being away from the table prevents the positive communication and social interactions that have been found to promote healthy food consumption and attention to satiety cues. Second, being distracted during mealtime is a missed opportunity for modeling healthy eating habits such as portion control and attending to what family members are actually eating. Third, eating in front of the television exposes family members to food marketing and increases consumption of unhealthy foods (Coon et al., 2001; Harrison & Liechty, 2012). The interruption of routines by habitual distractions is an example of how ecologies intersect to influence children’s health. As social interactions among family members are replaced by distractions and inconsistent communication, electronic media become a source of regulation and information.

**Food Marketing and Food Consumption**

Children are exposed to a large amount of food advertisements every day. In 2009, children between 2 and 11 years old viewed more than 10 food advertisements on television per day (Powell, Szczypka, & Chaloupka, 2010). Other forms of media, such as adver-games, are also replete with food advertisements, much of it for high-fat, high-sugar foods. Disturbingly, children and adolescents perceive these products to have positive nutritional value if they view the advertisement multiple times or see it in multiple forms, such as billboards, web pages, or social media (Staiano & Calvert, 2012).

Parents are concerned about the amount of exposure their children have to food marketing, responding that television commercials have a large impact on children between ages 2 and 11 years (Harris, Milici, Sarda, & Schwartz, 2012). In one survey study, over 40% of children asked their parents to purchase a food product while shopping that they had seen on television,
and over 8% cried or had a tantrum when told they could not have the item (Arnas, 2006). Qualitative reports suggest that it is important for parents to “shield” children from the effects of food marketing, especially if food marketers are targeting ethnic minorities (Baskin et al., 2013).

The Institute of Medicine (2013) and the World Health Organization (2013) have identified the marketing of foods high in fat, sugar, and salt as an important element in the causes of childhood obesity. Although recognized as an important concern, the solution is less straightforward. Voluntary self-regulation through the Children’s Food and Beverage Initiative (Institute of Medicine, 2013) has made some progress in limiting food advertising directed at children and including healthier products. However, even when healthier products are included in advertisements, young children do not recall seeing them (e.g., milk), but they do recall seeing items such as french fries when they were not present (Grow & Schwartz, 2014). The strong pull toward food brands starts early. By 5 years of age, most children can name common food brands and more accurately name unhealthy brands than healthy brands (Tatlow-Golden, Hennessy, Dean, & Hollywood, 2014). Federal regulation is unlikely to be effective in the United States because concerns about paternalism and freedom of speech outweigh concerns about children’s health. There are, however, other actionable steps that can be taken to create a healthier media environment for children and families.

**Summary and Recommendations for Policy and Practice**

The proximal processes that regulate the development of healthy eating behaviors extend across multiple ecologies, ranging from cell to community. Across each ecology, regulatory processes transact to organize the complex function of maintaining homeostatic balance. The research agenda for this multifaceted problem cannot be accomplished by a single investigator or even a single team of investigators; instead, it requires a transdisciplinary approach involving the integration and synthesis of multiple disciplines to create new knowledge, methods of inquiry, and translation (Fiese et al., 2013).

First, translating biological and genetic risk within the context of developmental and family regulatory systems requires attention not only to between-person variability but also to myriad factors that affect interpersonal processes, such as child temperament, attachment relationships, response to negativity, and the organization of family routines. Second, family processes intersect with individual emotion-regulatory systems that can vary over time. More attention needs to be paid to the intersection of emotion-regulatory systems, such as temperament and attachment, and family systems regulatory systems, such as routines. This intersection may provide valuable insight into how food choices and weight trajectories change over time. Third, family processes are ultimately embedded in larger cultural and sociopolitical contexts. Although it may not be immediately obvious how momentary interactions are influenced by this larger context, the personal interpretation of what is considered to be healthy and the influence of the media on purchasing and food preferences cannot be ignored.

Because we are proposing a complex and multifaceted approach to understanding childhood obesity, a multipronged approach to prevention, intervention, and policy are likewise warranted. Several influential government and private foundations have made a host of recommendations over the past several years to reduce the prevalence of childhood obesity (Robert Wood Johnson Foundation, 2010; Institute of Medicine, 2011, 2013). We broaden our recommendations to include family members either as key stakeholders or as recipients of information. Too often families are left out of the conversation for fear that they are being blamed as the cause for childhood obesity. From a cell-to-society framework, families are at the nexus as a means for change rather than being at fault.

We have proposed that biological and child self-regulatory factors transact with the social family environment to either increase or reduce risk for obesity. However, we have very little evidence about parental understanding of the ways in which individual biology can be shaped by the environment. In a recent review of qualitative studies that examined parents’ beliefs about healthy behaviors for preventing overweight in children, Pocock and colleagues reported that parents identified child characteristics such as “picky eating” and a tendency toward sedentary behavior (Pocock, Trivedi, Wills, Bunn, & Magnusson, 2010). It is not clear whether parents ascribed these characteristics to inborn biological traits or to behaviors that can be shaped.
through family involvement. As broader prevention and intervention programs are created and implemented, it may be important to develop educational materials about the “new biology” of health. With emerging evidence linking diet, physical activity, and brain health (Khan, Raine, Donovan, & Hillman, 2014), parents may be receptive to educational materials that emphasize the importance of a healthy diet and physical activity in effecting positive changes in biological functioning, including brain health and reducing disease risk.

Key aspects of self-regulation include the modulation of negative emotions. This includes the child’s response to negative emotions and response to satiety cues as well as parents’ response to child negativity, in particular during meals. Innovative interventions designed to promote children’s attention to satiety cues and reduce parental controlling and restrictive feeding dynamics are now underway (Eneli et al., 2015). Similar approaches have been proposed for child care settings (Dev, McBride, Speirs, Donovan, & Cho, 2014). It is important to note, however, that an emphasis on self-regulation has been limited primarily to the preschool years. This makes sense given that the preschool years are critical for the formation of self-regulation. However, other developmental periods with known risk for unhealthy weight gain, such as early adolescence, may also be prime targets for interventions that include a focus on self-regulation (e.g., mindfulness; Olson & Emery, 2015).

Social interactions that have been found to be meaningful and health promoting during family meals should be included in prevention and intervention efforts. Families across the economic spectrum report that they value the time spent during meals but often experience barriers to having a calm and pleasant mealtime with their children (Fulkerson, Story, Neumark-Sztainer, & Rydell, 2008; Malhotra et al., 2013; Quick, Fiese, Anderson, Koester, & Marlin, 2011). Interventions that include parent–child communication as a key element in health promotion have demonstrated positive effects in decreasing sedentary behavior (St. George, Wilson, Schneider, & Alia, 2013). Staff at the Family Resiliency Center at the University of Illinois have developed a series of public service announcements that address common mealtime barriers, such as work/life stress, use of media during meals, and cooking together as a family. These are publically available and can be used for educational programming (http://familyresiliency.illinois.edu/mealtimeminutes.htm). Educating families about the need to have a healthy diet and increase physical activity may not be enough; it is also important to provide them with the social tools to put this knowledge into action.

Involving families as key stakeholders in the conversation about targeted food marketing is essential. It is important to raise families’ awareness about the amount and variety of exposure to food marketing and its impact on young children. It is possible to create discussion boards at child care settings, schools, libraries, and other venues where parents of young children gather to discuss how food marketing affects choice, family dynamics, and young children’s inability to distinguish persuasion from a friendly request. Too often, families are left out of the planning phases of campaigns to promote healthy living. Building on parent-led communication strategies, including blogs and online parent groups, can have transformative effects at the community level (Institute of Medicine, 2013). For example, as noted by the Institute of Medicine (2013), a group called MomsRising has successfully collected thousands of personal stories that have influenced health policy. They have developed a powerful model that builds on partnerships among busy moms, advocates, and trusted experts with access to a knowledge base about specific topics. This organizational model can be adapted to reduce exposure to food marketing targeted at children and empower families to take control of media in their households.

There is cause for optimism in what may seem like an overwhelming agenda. Researchers are increasingly working on transdisciplinary teams to address grand challenges such as childhood obesity (Gortmaker et al., 2011). The next step is to translate scientific advances to families who can take charge of their food environment and encourage healthy household habits. Families may be agents of change through partnerships with child care settings, schools, parks, and nonprofit organizations such the YMCA/YWCA, Boys and Girls Clubs, and community organizations. A multisector approach that includes families as partners will encourage policy changes that promote the health and well-being of the community at large and reduce the tendency to blame parents. Working together, we have many opportunities
to address one of the nation’s major public health problems and provide a brighter future for children.

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