Adolescent obesity and dietary decision making—a brain-health perspective

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Adolescence represents a key period of brain development underpinned by the ongoing maturation of the prefrontal cortex—a brain region involved in the regulation of behaviour and cognition. Given the high prevalence of obesity in adolescents worldwide, this Review examines neurobiological and neurocognitive evidence describing the adolescent propensity to consume calorie-dense foods, and the neurodevelopmental mechanisms that heighten the adverse impact of these foods on brain function. The excessive consumption of calorie-dense food can undermine self-regulatory processes through effects on brain function and behavioural control. These changes could introduce enduring maladaptive eating behaviours that underlie adult obesity and related metabolic syndromes. Better understanding of links between adolescence, dietary decision making, and brain function is essential for clinicians to develop effective intervention strategies and for reducing long-term health-care costs associated with obesity.

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Introduction

Overconsumption of nutrient-deprived, calorie-dense foods is the leading cause of obesity, preventable chronic disease,[1] and premature death among adults. Unhealthy dietary choices and obesity have adverse effects on all major organ systems of the body including the brain, with new evidence suggesting, for example, that excessive adiposity increases risk for neurodegenerative diseases such as Alzheimer's disease.[2] But what is the effect of poor diet and obesity on the developing brains and minds of youth?

Emerging evidence suggests that the adolescent brain might be particularly susceptible to the effect of obesity and overconsumption of nutrient-deprived calorie-dense foods.[3] Adolescence is the developmental period that begins with the onset of puberty and ends with the onset of adulthood. According to WHO, adolescence is the period spanning the ages of 10 to 19 years, although others argue that this period is somewhat longer because of continued physical and neurobiological development into the early 20s.[4] Regardless of the precise definition, adolescence is recognised as a period of susceptibility to health risks because of the rapid growth and heightened psychological plasticity that mark this period.

Compared with all other neurological processes, this susceptibility seems most evident in regulatory processes governing dietary behaviour and decision making. Regulatory processes are crucial for healthy dietary practice as they help to inhibit the urge to consume foods that are highly palatable and calorie dense. However, regulatory processes are underdeveloped in adolescence owing to the continued development of the prefrontal cortex, an area of the brain consistently linked to self-regulation. Less appreciated but equally important is the finding that adolescence is also a period when the adverse neurological effects of obesogenic diets could be potentiated precisely because the brain is plastic during this period. Together, these factors contribute to what might be described as a dual vulnerability of the adolescent brain to health risks associated with the overconsumption of calorie-dense foods.

In this Review, we describe how the developmental status of the prefrontal cortex during adolescence increases the risk of overconsuming palatable calorie-dense foods. Additionally, we outline preclinical animal research showing that such unhealthy dietary habits negatively affect core aspects of neurochemical signalling, reward processing, and inhibitory neurotransmission, which are essential for adaptive cognition, and that these biological and behavioural effects might be more pronounced in adolescence than adulthood.

Key messages

- During adolescence, the brain is undergoing maturation, and the prefrontal cortex—a key area for cognitive control—is the final brain region to reach maturity, remaining in a state of heightened plasticity throughout adolescence
- The developing prefrontal cortex has less capacity than the mature prefrontal cortex to exert control over reward-driven behaviours such as consuming calorie-dense foods, which are often highly palatable
- Excessive consumption of calorie-dense foods could alter functional and structural maturation trajectories in the prefrontal cortex, causing enduring cognitive and behavioural changes
- Animal models that use calorie-dense diet manipulations during adolescence have highlighted alterations to dopamine and GABAergic signalling in the prefrontal cortex and other cortical regions
- Human neuroimaging studies have shown pronounced alterations to neural systems involved in self-regulation and reward valuation in adolescents with obesity compared with adolescents with a healthy weight
- The overt negative physical effects of excessive consumption of calorie-dense foods might be masked by the rapid growth spurt during puberty
- Good nutrition is essential during adolescent neurodevelopment for optimal brain health

The young mind under construction

Because adolescence is a period of marked psychological development and continued neuroplasticity, experience exerts a larger effect on the structure and function of the brain than it does in adulthood.[5] During adolescence, the brain undergoes extensive neurobiological and functional remodelling, particularly in brain regions responsible for behavioural control and reward seeking, most notably prefrontal cortex and dopaminergic reward pathways.[6–9] Key maturational developments include dendritic pruning to refine synaptic connections, and increased axonal myelination.[7,8,10] The observed increase in myelination, and by extension white matter connectivity, increases the speed of impulse conduction throughout region-specific neurocircuitry, facilitating increased integration of brain activity essential for high-order cognitive function.[11] Brain development in adolescence facilitates the emergence of cognitive flexibility, reasoning, planning, and impulse control with the transition into adulthood.[12,13]

The maturing adolescent brain is uniquely susceptible to environmental influences and experiences that can shape the development of neurocircuits by local remodelling. This process is referred to as experience-dependent neuroplasticity,[14] an umbrella term describing dynamic reorganisation of brain structures and functions in response to environmental inputs. Experiences or environmental stimuli can strengthen the connections between presynaptic and postsynaptic neurons in a process referred to as long-term potentiation.[15] Over repeated exposures to the same experience or environmental stimuli, these connections stabilise, and the distribution of stabilised connections influences axon and dendritic growth patterns.[15] Neurotransmitters including dopamine play key roles in modulating neuroplasticity and interindividual variability in functional activation patterns and cognitive abilities.[15] Dopaminergic and cholinergic neurotransmitter systems, in particular, are thought to be important for the development of cognitive control and abilities throughout the lifespan; the activity and responsivity of these systems follow an inverted U-shaped pattern, peaking during early adolescence.[16] The development of balance in these neurotransmitter systems during adolescence is therefore crucial for obtaining optimal brain function in adulthood.

Diet quality as a dictator of brain health—a neurodevelopmental perspective

Brain development can be influenced by multiple environmental factors, of which diet quality is a major one.[17] In 2019, we outlined a neurocognitive model describing how individual differences in lateral prefrontal cortex input can drive the excessive consumption of hyperpalatable calorie-dense foods.[18] Over time, the persistent and excessive consumption of calorie-dense foods can lead to enduring changes in the structure and function of the prefrontal cortex,[19] including altered dopamine signalling[20,21] and inhibitory neurotransmitter systems within this area of the brain.[22,23] This alteration leads to impaired cognitive control, further driving the persistent and excessive intake of hyperpalatable calorie-dense foods.[18]

As the prefrontal cortex is still undergoing major developmental and maturational processes during adolescence,[24] the sustained and excessive consumption of high-fat and high-sugar foods during adolescence could have a stronger influence on neurodevelopmental trajectories than any other developmental period. Moreover, research in rodents has indicated that excessive consumption of appetitive calorie-dense foods during the rodent equivalent of adolescence could have a pervasive functional effect on the brain, leading to enduring deficits in learning and memory.[25]

Adolescence as a period of heightened reward sensitivity

Adolescence is a period of heightened emotionality,[26] during which adolescents show both increased reward drive and reduced cognitive control.[27] The prefrontal cortex is still developing throughout adolescence, whereas the limbic regions reach maturiy much earlier in development. This time difference in maturation creates an imbalance between reward-driven behaviours (limbic system) and top-down cognitive regulation (from the prefrontal cortex), which is manifested as augmented sensitivity to rewards and diminished behavioural regulation.[28,29] Low behavioural regulation has been linked to underdeveloped connectivity between the amygdala (a key node of the limbic system) and the prefrontal cortex during adolescence,[30] which is observed across species, including humans[31] and rodents.[32] This imbalance between top-down regulatory regions and subcortical regions might drive excessive consumptive behaviours, motivated by food rewards,[18] emotional eating,[33] and binge eating,[34] which are key risk factors for obesity.

More specifically, the enhanced sensitivity to rewards observed during adolescence has been attributed to the age-dependent changes in the maturation of the frontostriatal circuit.[8,26] The development of the prefrontal cortex lags behind the development of subcortical reward regions, resulting in a propensity for impulsive and sensation-seeking behaviours in adolescents.[12,26] Indeed, several lines of evidence have consistently shown that, compared with adulthood, striatal responses to reward magnitudes are exaggerated during adolescence,[29] and that this pattern of activation is associated with trait impulsivity and the likelihood of engaging in risk behaviours.[35] Collectively, neurochemical, structural, and electrophysiological evidence shows that the reward-signalling dopaminergic innervation originating from the ventral tegmental area to the prefrontal cortex and nucleus accumbens matures during adolescence.[36] This process explains why rewarding behaviours, including consumption of palatable foods, are frequent in young people.[37]

Why do young people have a hard time saying no to appetitive calorie-dense foods?

Adolescents have a greater dietary intake of refined sugar and fat than do any other age group.[38] This association has been attributed to increased food consumption during this developmental period, changes in food choice independence, and an increased sensitivity to natural rewards. In the modern obesogenic food environment, dietary self-regulatory abilities are essential to control calorie-dense food consumption. At the heart of effective dietary self-regulation is the capacity to inhibit (or suppress) appetitive urges evoked by appetitive food cues and stimuli, and properly evaluate the nutritional value of available food options. Such self-regulatory abilities have been linked to the cognitive control network, particularly the lateral prefrontal cortex. Recruitment of the lateral prefrontal cortex is essential to modulate cortical activity in the reward region, thereby enabling the neurocognitive mechanisms necessary to dampen food-evoked cravings and the motivation to eat (figure, see Lowe and colleagues[18] for an overview)—abilities that are still developing in adolescence.

Adolescents have increased levels of food consumption, partly because of elevated metabolic activity driving rapid physical growth and development that comes with puberty, including gain in muscle mass in male adolescents and fat mass in female adolescents.[39,40] Rapid growth is observed across species, whereby adolescent rats have the highest caloric intake during this period relative to their bodyweight.[41] In mice, the adolescent growth spurt can partially accommodate for excessive caloric load from high fat diets without the considerable weight gain typically observed in adult animals consuming similar diets.[42] Adolescence might therefore provide partial protection against the development of obesity. However, in the absence of negative consequences (eg, excess weight gain), behavioural habits acquired during adolescence might potentiate overconsumption of calorie-dense foods into adulthood, as the health consequences might not be immediately apparent. This premise highlights an increasing need to consider diet quality, rather than weight status alone, as a key factor that can influence adolescent brain health.

Reward circuitry in the brain is activated by consumption of palatable foods.[43,44] The developmental change in caloric need that occurs in conjunction with an increased drive to engage in reward-driven behaviours can promote the consumption of palatable calorie-dense foods during adolescence.[45,46] In absence of mature top-down regulatory processes, striatal dopamine release in response to a rewarding event is exaggerated in adolescents in comparison with adults, making adolescents more sensitive to reward value than adults.[12] Moreover, the augmented striatal dopamine release in response to stimuli associated with rewarding foods might make it harder for adolescents to control consumption.

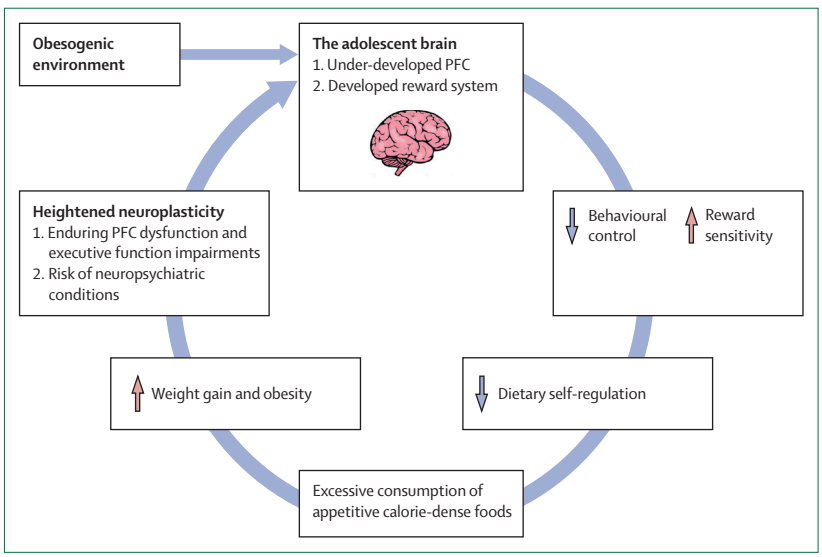

Figure: **Impact of the obesogenic environment on the adolescent brain**
The maturing adolescent prefrontal cortex does not have full top-down control over behaviour. Consequently, excessive consumption of calorie-dense food, weight gain, and the neurobiological impact of such food is heightened. PFC=prefrontal cortex.

The increased neural responsivity to rewarding events in conjunction with the ongoing maturation of the prefrontal cortex could potentiate adolescent susceptibility to the overconsumption of foods that are nutritionally deprived and calorie dense. Many neuroimaging studies signify the importance of prefrontal cortex regions in the regulation of appetitive food cravings and consumption. Areas in the prefrontal cortex, such as the dorsolateral prefrontal cortex, play a crucial role in the volitional regulation of appetitive short-term rewards, enabling individuals to select the more beneficial, but less palatable, long-term rewards.[18] Indeed, the motivation to consume appetitive calorie-dense foods is negatively associated with brain activity (measured by the haemodynamic blood oxygen level-dependent response using functional MRI) in the dorsolateral prefrontal cortex and medial prefrontal cortex in individuals reporting strong impulses to consume these foods.[47]

Furthermore, lower activation of superior and middle frontal gyrus, ventrolateral prefrontal cortex, and medial prefrontal cortex in response to food rewards is associated with increased body-mass index in adolescents.[48] Correspondingly, evidence from prospective imaging studies suggests that elevated striatal activity in response to palatable food cues is associated with an increase in bodyweight and fat mass in adolescents.[49] Conversely, successful weight loss in adolescents was associated with increased brain activity in the dorsolateral prefrontal cortex in response to images of appetitive calorie-dense food, supporting the contention that increased activity in the prefrontal cortex in response to food cues might play a crucial role in modulating food choices.[50] Together, these data indicate that food-seeking and consummatory

behaviours might be especially pronounced in young people, and that this association is related to a reduced prefrontal regulation of food reward cues.

The industrialisation of food production has led to the mass development of cheap and appetitive food items that are heavily marketed in a way that exploits hedonic responses, thereby driving excessive purchasing and consumption. More than 84% of media advertisements seen by children and adolescents are for hypercaloric foods and beverages.[51] These advertisements are present in television, social media websites, and apps.[52] In Canada alone, adolescents are exposed to over 14·4 million food advertisements a year on their favourite websites.[53] This pervasive marketing of calorie-dense foods in combination with their ubiquitous presence in the modern environment can potentiate overconsumption. Indeed, there is increasing evidence that these advertisements can influence children and adolescents' preferences and attitudes toward calorie-dense foods, leading to an increased risk of excessive consumption and overall unhealthy diets.[54]

Food for thought

As described previously, the brain adapts to the environment and experiences via neuroplasticity processes. The foods consumed as part of the modern diet are considered a powerful environmental influence on adolescent neurodevelopment. Rodent studies, in which the rodents were fed diets that replicate human diets that are high in saturated fats and refined sugars, indicate that excessive consumption of these foods underpins alterations to a range of neurotransmitter systems in cortical brain regions responsible for behavioural control. Chronic stimulation of the still maturing mesocorticolimbic dopamine reward system during adolescence by excessive consumption of palatable foods might drive enduring neurobiological changes to neurotransmitter and endocrine systems.[9,55] Adaptive changes in neurotransmission could recalibrate brain reward systems to promote heightened food reward preference and evoke cognitive dysregulation.[56]

In rodent studies, excessive consumption of hyperpalatable foods leads to adaptive changes in dopamine signalling pathways within brain regions controlling reward processing and decision making, including the prefrontal cortex,[57] nucleus accumbens,[58,59] and hippocampus.[60,61] Although this Review focuses on the prefrontal cortex as a driver of food choice, the hippocampus plays an integral role in regulating dietary choices and calorie-dense food consumption (see Hargrave and colleagues[62] for an overview). The consumption of hyperpalatable foods evokes release of dopamine in the mesocorticolimbic system, such that frequent consumption of these foods can lead to overstimulation of this pathway.[63] Adaptive changes then occur in receptor expression to compensate for this overstimulation, including downregulation of dopamine D_2 receptors in the striatum, resulting in a neurochemical blunting of responses to typically rewarding stimuli.[63] It has been proposed that adaptive changes to the reward systems can lead to deficient reward responses and further drive food-seeking behaviours to compensate for the reduced experience of reward.[64] Furthermore, rodent studies indicate that effects on the dopaminergic reward signalling neurocircuitry can be persistent.[65] As dopamine is a modulator of cortical plasticity, these adaptive responses to diet quality can potentially influence plasticity processes during this malleable period, leading to the observed structural and functional changes observed in people with obesity (panel).

The susceptibility of the adolescent brain to alterations evoked by a hyperpalatable diet might emerge as altered motivated behaviours. Experimentally, motivation in rodents is measured through progressive ratio tasks that require increasing numbers of responses, such as lever presses, to gain a reward. Male rats that consumed a high-sugar diet during adolescence, but not in adulthood, were less motivated to press a lever for palatable food rewards as adults than rats who had not consumed a high-sugar diet during adolescents.[77] Conversely, adolescent female rats that consumed high-sugar diets showed heightened motivation for rewards, indicative of craving.[77] These studies showed that high-sugar diets can alter reward processing neurocircuitry in a sex-dependent and age-dependent manner, with heightened susceptibility in adolescent males.

Developing balance in the brain

During brain development, dopamine plays a key role in controlling excitatory and inhibitory neurotransmission in the prefrontal cortex.[78] In the postnatal brain, γ-aminobutyric acid (GABA) is the main inhibitory neurotransmitter and glutamate is the main excitatory neurotransmitter. Balance between excitation and inhibition is essential for brain network function and cognitive control. Disrupted balance between excitation and inhibition is linked with neuropsychiatric conditions, including autism and schizophrenia.[79,80] Neurochemical data indicate that inhibitory GABAergic neurotransmission, particularly within the prefrontal cortex, is still developing throughout adolescence,[80] and might promote impulsive and risk-taking behaviours characteristic of adolescence.[29]

The late maturation of inhibitory GABAergic signalling in the brain also results in a prolonged period of susceptibility within the highly plastic adolescent brain. The consumption of poor quality, hypercaloric foods during adolescence is proposed to trigger functional changes in dopaminergic signalling,[59] which in turn might alter the developing inhibitory GABAergic signalling in the prefrontal cortex.[77,81] As the balance of excitatory and inhibitory neurotransmission in the mature prefrontal cortex is important for high-order cognition and the control of behaviour,[82] these changes in dopaminergic, and subsequently GABAergic signalling, triggered by junk

***Panel*: Candidate brain mechanisms that might be influenced by obesity during childhood and adolescence**

Local remodelling of neural circuitry

Alterations to neurotransmitter systems within the brain (including dopamine, γ-aminobutyric-acid, cannabinoid, and serotonin) by obesity can promote local remodelling of neural circuitry through receptor changes. For example, increased striatal D_2 receptor activity alters the firing pattern of midbrain dopamine neurons in the ventral tegmental area.[66] This process results in changes to the reward system, and overexpression of striatal D_2 receptors can alter inhibitory neurotransmission in the prefrontal cortex.[67] Moreover, local remodelling of neural circuitry occurs within brain regions controlling food intake and energy balance, including the hypothalamus.[68]

Gonadal hormones

Obesity has been shown to increase testosterone concentrations in pubertal females[69] and is linked to early onset of puberty.[70] Gonadal hormones including oestrogen, progesterone, and testosterone play a complex role in shaping the adolescent brain, particularly through the pruning of excess synapses, which contributes to reduced grey matter density. Obesity-induced early onset of puberty could therefore derail the trajectory of synaptic pruning and maturation of receptor density in cortical regions required for high-order cognition, reward signalling, and homoeostatic control of energy balance.

Hunger or satiety hormones

The adipose-secreted hormone leptin acts in the hypothalamus to supress hunger. However, obesity can lead to chronically high leptin resulting in central leptin resistance and leptin receptor desensitisation, which can reduce satiety signalling and promote food seeking. In children and adolescents with obesity, leptin desensitisation might underlie hyperactivation of reward regions seen in functional MRI studies (eg, Jastreboff and colleagues[71]), increasing high-calorie food intake and causing enduring changes in neural pathways. The gut hormone ghrelin acts as a hunger signal. Ghrelin controls motivation to eat via its interaction with the dopamine neurons in the ventral tegmental area.[72] Adolescents with obesity show a blunted ghrelin response following the ingestion of glucose, which was coupled with decreased activation of the prefrontal cortex following glucose and fructose ingestion.[73] As such, obesity-induced desensitisation to gut hormones could have an enduring effect on the reward system.

Microbiota–gut–brain axis signalling

In the past decade the gut microbiota has emerged as a major player in modulating brain health and shaping the developing brain. The development of the microbiota follows a similar trajectory to the brain, not reaching full maturity until early adulthood. Diet is a major influence on the microbial colonies in the gut, as such disruptions during adolescence, a crucial period of dynamic microbiota–host interaction, have the potential to profoundly alter brain–gut signalling.[74] A change in the gut microbiota that favours pathobiotic microbial overgrowth can result in dysbiosis, evoking a systemic inflammatory response. These mechanisms potentially alter brain function through changes to neurotransmitters and through local remodelling of neurocircuitry, causing long-term alterations in reward function and behavioural control (see Borre and colleagues[75] for review).

Immune system activation

Obesity promotes a low-grade inflammation throughout the body and excessive consumption of high-fat and high-sugar foods promote microglia activation in the brain.[76] Microglia play key roles in remodelling cortical white matter and dendritic spine density, so increased activation in adolescence by obesity could contribute to altered connectivity and more pronounced synaptic pruning during adolescence.[76] These neural changes might predispose individuals with obesity to dysregulated eating behaviour, including impulsive and compulsive food consumption following exposure to food cues.[18]

food consumption can produce profound effects on behaviour.

Adult rats exposed to a high-fat diet, for example, have altered GABAergic signalling in the hippocampus and prefrontal cortex.[83] Our own research has shown that consumption of a high-sucrose diet during adolescence is associated with a reduction in GABAergic neurons in cortical regions essential for cognition, and manifests as cognitive deficits and dysregulated behavioural control.[22,81] Furthermore, research in rodents points towards obesity-induced changes in extracellular matrix structures that surround neurons in the brain called perineuronal nets.[84] These structures regulate the firing of cortical neurons involved in cognition, frequently colocalising with parvalbumin interneurons, whereby they act to restrict plasticity and control synapse formation.[84] As cortical perineuronal nets have a protracted developmental trajectory, showing maturation in juvenile and adolescent periods in a regionally dependent manner, disruption of these structures could lead to alterations in plasticity and remodelling of connectivity between cortical regions. Nevertheless, enhanced understanding of neurochemical systems that are altered by consumption of hyperpalatable foods is crucial to the development of potential treatments and therapeutic interventions that could protect the brain and restore aspects of function when the young brain is in a highly malleable and receptive state.

Clinical significance

Childhood and adolescent obesity are among the most important global health issues. Statistics indicate that 18·5% of children and adolescents in the USA were living with obesity in 2015–16.[85] Similar trends are observed worldwide, with rates increasing rapidly in low-income and middle-income countries;[86] however, in developed nations (eg, Canada, USA), obesity rates have

started to reach a state of little or no change.[87] Childhood and adolescent obesity are of concern, as children and adolescents with obesity are more likely to maintain their weight status into and throughout adulthood, and are at increased risk of developing diabetes and cardiovascular disease at a younger than average age.[88]

As highlighted in this Review, the impact of diet quality on the developing brain has long-lasting implications that could further facilitate obesogenic dietary behaviours. Sustained and excessive consumption of foods high in saturated fats and refined sugars can influence the development of the brain circuits necessary to facilitate self-regulatory behaviours that are key to limiting overconsumption.[18] Thus, there is a crucial need to develop effective interventions to improve diet quality and reduce obesity in young people, to reduce the negative physiological impact of obesity across the lifespan. As adolescence offers partial protection against the development of excessive adiposity, there is an increasing need to focus on diet quality within this age range, rather than obesity as the main focus. Such therapeutic interventions could protect the brain and restore aspects of function, and might be most effective while the young brain is in a highly malleable state.

Specific behavioural interventions that are effective for adolescent populations need to be considered. Interventions in this age group often fail when they do not consider adolescents' need to feel respected and the importance of social status (see Yeager and colleagues[89] for an overview). For instance, implicit attitudes and purchasing behaviours for calorie-dense foods are significantly reduced when adolescents are taught about the manipulative marketing practices used by the food industry.[90] The success of this intervention was linked to focusing on values adolescents felt were important (eg, social justice, reasserting one's autonomy, and rejecting adult control), highlighting the need to consider value-aligned interventions in this population. Moreover, interventions are often geared towards preventing obesity rather than improving diet quality per se. As highlighted above, this focus on obesity might not be the most appropriate approach, as physical indices of diet quality (eg, excess weight and adiposity) might not emerge until later in life.

Exercise interventions, to date, are among the most promising means of improving brain health and cognitive control. Across the lifespan, exercise interventions improve cognitive performance;[91] this improvement has been attributed to exercise-induced changes in the structural and functional integrity of the prefrontal cortex and hippocampus,[92,93] which are the cortical regions shown to be susceptible to obesity. Moreover, evidence has highlighted that aerobic exercise can decrease neural responsivity to appetitive food cues and modulate hunger circuits to improve dietary choices.[94,95] However, there is a shortage of research on the effect of exercise interventions on the adolescent brain, with current research focusing on older adults[91] or preadolescent children.[96] Considering the tangible benefits of exercise interventions, there is need to expand this line of work to adolescent populations, especially within young populations prone to dysregulated eating behaviours (eg, people with obesity or binge-eating disorder).

Translational challenges and outstanding questions

Whereas animal models allow for exquisite control over diet quality and food intake, food choice in humans is complex and can be framed as a socioenvironmental behaviour influenced by environmental cues, cultural preferences, social attitudes, and genetics. Because of the fluidity of human dietary choices, most diets consist of a combination of healthy and unhealthy calorie-dense items, contrasting the controlled laboratory models, which might only represent the extreme diets consumed by few people.

Thus, there is an essential need for large-scale prospective studies in humans, particularly adolescents. Such research is especially pertinent given the growing body of behavioural and imaging studies in humans highlighting the short-term[97] and long-term effect of obesity and obesogenic diets on cognitive processes and brain structures in humans.[98,99] It is only through large-scale collaborative approaches that the scientific community will be able to model the development of dietary self-regulation, the effect of poor diets on the developing brain, and the dose–response relationship between calorie-dense food consumption and maladaptive developmental neural trajectories. With the emergence of large-scale, open-source multisite projects, such as the Adolescent Brain and Cognitive Development study,[100] such projects are logistically feasible.

Finally, in this Review we focused on prefrontal cortex regulation of subcortical reward regions as the primary factor driving dietary decisions. This focus was specific to the developmental approach we applied, in which these subcortical regions are often fully developed before the prefrontal cortex. This difference in developmental trajectories is thought to contribute to the observed increase in reward sensitivity observed in adolescence, which makes adolescents more receptive to the rewarding aspects of appetitive calorie-dense foods. However, consumptive behaviours are modulated by an extended network of brain regions, including the hippocampus, hypothalamus, and amygdala.[101] Moreover, a growing research field is clarifying how the microbiota–gut–brain axis can effect neurodevelopment; a potential pathway might be the axis' role in maintaining metabolic homoeostasis (see Borre and colleagues[76] for an overview). As such, future work needs to examine how diet and the gut microbiota influence neurodevelopmental trajectories.

Conclusion

In this Review, we propose that adolescence is a period of dual susceptibility, during which the regulatory processes

governing dietary behaviour and decision making are still underdeveloped because of the continued development of the lateral prefrontal cortex. As such, emerging self-regulation and increased independence over food choices can make adolescents prone to choosing unhealthy food options. However, the inherent plasticity of the young brain could potentiate the adverse neurological effects of obesogenic diets. These diet-induced changes (outlined above) can manifest as poor cognitive control and heightened impulsivity into and throughout adulthood, further potentiating the cycle of dysregulated eating behaviours into adulthood.

In conclusion, the available evidence highlights that clinicians and researchers need to shift the focus from body size and obesity to diet quality itself. The ongoing physical growth observed during adolescence can partially accommodate for excessive caloric load without the considerable weight gain typically observed in adults. As such, the negative impact of a poor diet might not be overtly apparent. Nevertheless, as highlighted above, diet quality can have severe and detrimental impact on the developing brain, and focus should be drawn to understanding and intervening during this period of susceptibility.

Contributors

All authors completed the literature search, wrote the Review, and developed the Review's concept and structure. ACR prepared the figure.

Declaration of interests

We declare no competing interests.

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