

Ultra-processed Foods and Teen Neurobiology: Why Modern Diets Trigger Overeating and Metabolic Stress

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ABSTRACT

Background: Adolescence is a vital neurodevelopmental time, which is characterized by a high level of plasticity in reward-related circuitry and further maturation of executive control centers. Today, the world is becoming a place where the global food system is based on ultra-processed foods (UPFs), and is specifically designed to be hyper-palatable and rich in energy.

Objective: The current mini-review evaluates neurobiological and biochemical processes that cause UPFs to induce compulsive overeating and systemic metabolic stress in adolescents.

Methods: The survey of the current literatures was rigorous, focusing on the nexus between the NOVA food classification, adolescent neurobiology, the gut-brain axis and metabolic signalling pathways.

Findings: The evidence suggests that the UPFs take advantage of the developmental incompatibility between the early-maturing rewards systems, specifically the nucleus accumbens and the late-maturing system of prefrontal cortex (PFC). Some addictive neuroadaptations trigger a dopaminergic down-regulation, which is caused by chronic consumption. In addition, UPF-related systemic inflammation also leads to microglial activation in the hypothalamus, thus impairing leptin and insulin signalling. Components that are present in UPFs promote gut dysbiosis and increased intestinal permeability, which additionally promote neuroinflammation through the gut-brain axis. As a clinical presentation, these changes include poor impulse control, hippocampal-dependent memory loss and an early metabolic dysfunction including insulin resistance and metabolism dysfunction-related steatotic liver disease (MASSLD).

Conclusion: UPFs are not simply caloric surpluses, but in fact, biological disruptors that take over adolescent neurocircuitry. The mitigation of the UPF epidemic requires a paradigm shift from a purely caloric-focused view to that of considering the impact of industrial food matrix on neurodevelopment. The main focus of the public health strategies should be protecting adolescent brain against the addictive and inflammatory nature of modern eating habits.

KEYWORDS: Dopamine, Gut-brain axis, Metabolic stress, Neuroinflammation, Ultra-processed food.

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INTRODUCTION

The adolescence is a distinct developmental time frame characterized by an increase in neuroplasticity and fine-tuning of intricate neural networks. In this period, a process of brain synaptic pruning and enhanced myelination, particularly of the prefrontal cortex (PFC), which controls executive functioning, impulse control, and long-term planning takes place. At the same time, subcortical reward centres, the ventral striatum and the nucleus accumbens, are the most sensitive ones. Biological vulnerability is formed by the developmental gap, usually called the dual systems model. The consumption of ultra-processed foods (UPFs), as industrial compounds based on primary food constituents (in terms of the NOVA classification, oils, fats, sugars, starches, and proteins) and supplemented with flavourings, colourings, and emulsifiers is explicitly aimed at taking advantage of this vulnerability. In contrast to whole foods, UPFs are hyper-palatable, calorically rich and nutritionally insufficient, triggering neurobiological responses that are similar to the addictive behaviours.

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HIJACKING NEUROBIOLOGY OF REWARD

The Dopaminergic Overstimulation and the Bliss Point: The food business plays with the notion of sensory-specific satiety and the bliss point (the exact combination of salt, sugar and fat) to maximise the release of dopaminergic. The dopaminergic system reacts hyper- reactively in adolescents. Once a teenager eats a high-sugary UPF, the nucleus accumbens emits a strong increase of dopamine. With repeated exposure

to these, the neuro-adaptation occurs; to counter overstimulation, the brain overgrows the receptors of D2 dopamine. This is followed by a state of reward deficiency syndrome where the adolescent no longer derives pleasure by eating normal amounts of food, and has to consume increasing amounts of UPFs to obtain the same hit.

There is impairment of Top-Down Inhibition: In the brain, the prefrontal cortex is the last area that undergoes maturation. Consumption of UPF has been reported to impair the integrity of the PFC structure among adolescents. Fatty and sugary diets suppress the expression of interneurons in the PFC that contain parvalbumin, which is central to inhibition. As a result, the brakes of the brain become weak, whereas the accelerator the reward system has maximum impetus. This neuro-physiological change explains the reason why adolescents find it hard to stop using UPF despite having a conscious understanding of its harmful effects on human health.

Metabolic Stress and Systemic Inflammation

The Neuroinflammation and its Role: UPFs are powerful provoking factors of systemic inflammation, which is due to the composition of saturated fats, trans-fats, and refined carbohydrates, as well as the lack of anti-inflammatory polyphenols and fibres. These compounds stimulate the secretion of pro-inflammatory cytokines, including interleukin 6 (IL 6) and tumor necrosis factor alpha (TNF 2). These cytokines may bypass the blood-brain barrier (BBB) or communicate through the vagus nerve to trigger the microglia- the micro-residential immune cells of the brain. There is chronic microglial activation in the hypothalamus that constitutes the hypothalamic gliosis. Since the hypothalamus coordinates the maintenance of energy homeostasis, this inflammatory environment obliterates the ability of the brain to determine the energy stores and induces a sense of starvation in the face of caloric surplus.

Diabetes Insulin and Leptin Insensitivity: The increase in the glycaemic spikes accelerated by UPFs requires insulin release to continue. This eventually develops peripheral insulin resistance. Nevertheless, insulin enters the BBB and regulates the hippocampal activity concerning memory and satiety. The neuroplasticity that facilitates learning and memory is impaired when the brain gives in to insulin resistance. Similarly, the leptin hormone which transmits the adipocyte satiety/ Satiety to the brain becomes inefficient. UPFs stimulate hyperleptinemia, but circulating leptin is impeded by high levels of triglycerides. Such leptin resistance guarantees that the brain of the teenager does not get the satiating signal, promoting a metabolic vicious cycle.

The Gut-Brain Axis: The Second Brain under Siege

Dysbiosis and Additives in Microbiomes: The intestinal flora in adolescence is highly sensitive to the input of the diet. UPFs do not have pre-biotic fibres that provide a healthy

microbial ecosystem. Also, industrial food additives like carboxy-methyl-cellulose and polysorbate 80 (widely used emulsifiers) have been reported to damage the mucosal barrier of the gut. This erosion causes a leaky gut, which is an increase in intestinal permeability. The Lipopolysaccharides (LPS), pro-inflammatory elements of bacteria, penetrate into the blood, worsening neuroinflammation. A dysbiotic intestine also reduces short-chain fatty acids (SCFAs) such as butyrate, essential to ensure the integrity of the BBB and the production of brain-derived neurotrophic factor (BDNF).

Serotonin and Mood Regulation: About 90 to 95% of all serotonin in the body is produced in the gut. UPFs interfere with serotonergic signalling by destabilising the gut milieu. In teenagers, this is manifested in an increased level of irritability, anxiety and depression. To relieve these depressed moods, adolescents often turn to other UPFs, a trap of comfort food that solidifies the addictive loop.

Lasting Effects: Cognitive and Metabolic

Effects on Hippocampal activity: Particular susceptibility to the metabolic disruptions caused by UPFs is the hippocampus, critical in the formation and maintenance of memory. Empirical studies indicate that those who live on diets containing the highest level of UPFs show worse spatial memory tests and have lower levels of hippocampal volume. The implication of such findings is that the nutritional deficits incurred during the adolescence period can have long-term harmful effects on educational results and consequent cognitive reserve in adulthood.^{14,15}

Steatotic Liver Disease Caused by Early Onset Metabolic Dysfunction-Associated Steatotic liver disease (MASLD): The liver is almost the sole metabolic site of the high fructose in UPFs. This fast oscillation of fructose initiates the process of de novo lipogenesis in teenagers leading to the development of hepatic fats. MASLD has turned out to be the most frequent chronic liver disease that plagues children and teenagers in Western cultures and is directly proportional to the consumption of sugar-sweetened drinks and ultra-processed snack food.¹⁶

DISCUSSION

A complex interaction between hormonal and neurological reconfigurations mediating the developmental passage from childhood to adulthood is increasingly disturbed by the industrialized food environment of the modern world. The data outlined in this review suggest that UPFs are an example of evolutionary mismatch of the products that our biological systems are poorly adapted to, especially when some are of rapid-growth phases.

Compulsive Character of Markets of Contemporary Food:

A long-standing debate in the existing literature regards the issue of whether UPFs are addictive drugs. The nutrients contained in traditional whole foods are in a complex structure of water and fibre, which slows down digestion

and signal transmission. Conversely, UPFs provide so-called naked calories, i. e., fast-absorbing sugars and fats that trigger an unusual pharmacokinetic pattern in the brain. The resultant dopaminergic releases could be compared to those that are caused by nicotine or alcohol. This creates a condition of passive overconsumption in the adolescent, where the impetus to eat is not directly connected to physiological needs of the body in the form of energy.^{17,19}

6.2. Interaction between Metabolic and Hedonic Signaling:

The loss in the quality of communication between the periphery and the central nervous system is among the most disturbing results. In a normal state, the energy homeostasis is maintained by the insulin, leptin, and hypothalamus crosstalk. Nevertheless, the richness of the UPFs in fructose and saturated fat causes the development of central resistance. In the event that the hypothalamus gets deaf to the satiety signals due to the loss of neuroinflammation and gliosis, the adolescent is already in the perceived energy deficit, even though the adiposity is prominent. This explains the paradox that is typified in clinical practice, whereby nutritionally starved people who are metabolically obese are observed at a cellular level.

Social, Economic and Environmental Determinants: The phenomenon of food swamp also needs to be mentioned in the analysis. In the lower socioeconomic groups, adolescents are also overrepresented within the sphere of UPFs, because such products are cheap and have a prolonged shelf life in comparison to fresh food. This environmental force, combined with violent marketing to the youth creates a biological loop of influence that cannot be fought by the will of the individual. The brain injuries on the hippocampus and the PFC also deteriorate cognitive flexibility needed to make healthier decisions during stresses.^{13,20}

Weaknesses of the ongoing research: As much as the relationship between UPFs and metabolic stress is strong; there are several challenges:

- Causality in Humans: A wealth of mechanistic evidence of microglial activation and synaptic pruning is based on rodent models. These findings are supported by neuroimaging in humans but longitudinal studies that start in early childhood are essential.
- The Cocktail Effect: The UPFs include hundreds of different additives. The identification of the particular emulsifiers, sweeteners or preservatives that qualify as the main forces behind gut-brain disruption is a biochemical challenge that is difficult to overcome.
- UPF definition: The NOVA classification is highly used and criticized at times by the breadth of its use. Further studies should begin to differentiate between the so-called minimally processed industrial foods and really ultra-processed products that are not based on any whole-food matrix.^{21,22}

Clinical and Pathological Implications: Pathologically, the acute to chronic metabolic stress of the youth is disastrous. We witness adulthoodification of pediatric illnesses, whereby such conditions as Type 2 Diabetes and MASLD (previously NAFLD) present themselves in the second decade of life. This change is reflected in the biochemical pointers; such as increased C-reactive protein (CRP), hypertriglyceridemia and fasting hyperinsulinemia, which are unequivocal signs that the adolescent body is experiencing some form of systemic stress.^{16,18,21}

CONCLUSION

Modern diet with its prevalence of ultra-processed foods is a great threat to the neurobiological and metabolic well-being of the adolescent population. The commandeering of the reward circuitry of the brain, incitement of hypothalamic inflammation and affecting the gut-brain axis, UPFs promote a biological environment that supports overeating and metabolic dysfunction. The solution to this crisis will require a complex approach to the problem, which cannot be reduced to calorie counting but rather a deep comprehension of the quality of food and its impact on the industrial food matrix. Protecting the growing teenage brain is more than metabolic health, but what is critical is the ability to preserve the cognitive and emotional processes of the next generation.

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