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Perspective

Processed Food–Rich Diets and Epigenetic Reprogramming: A Hypothesis of Long-Term Biological Imprinting

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ABSTRACT:
Diet represents one of the most influential environmental factors shaping human biology, not only through immediate metabolic effects but also via long-lasting regulatory mechanisms. While the health consequences of processed and ultra-processed food consumption have been widely discussed in relation to obesity and metabolic disease, their potential role in epigenetic reprogramming remains insufficiently explored. This letter proposes the hypothesis that chronic exposure to processed food–rich diets may induce persistent epigenetic modifications that influence immune regulation, metabolic homeostasis, and disease susceptibility across the lifespan and potentially across generations.
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Epigenetic regulation, including DNA methylation, histone modifications, and non-coding RNA activity, serves as a dynamic interface between environmental exposures and gene expression. Nutritional inputs are among the most potent modulators of these processes. Processed foods are often deficient in essential micronutrients involved in one-carbon metabolism, such as folate, choline, and vitamins B6 and B12, while simultaneously containing excessive refined sugars, industrial fats, and chemical additives. This imbalance may disrupt normal epigenetic signaling pathways, leading to aberrant gene expression patterns that persist long after the initial exposure [1-2].

Accumulating evidence suggests that Western-style dietary patterns are associated with distinct epigenetic signatures linked to inflammation and metabolic dysregulation. High intake of refined carbohydrates and saturated fats has been shown to alter DNA methylation profiles of genes involved in immune signaling, insulin sensitivity, and oxidative stress responses. These changes may not directly cause disease but instead create a state of biological priming, lowering the threshold for pathological responses when additional environmental or physiological stressors are encountered later in life [3].

The immune system appears particularly vulnerable to diet-induced epigenetic modulation. Immune cell differentiation, activation, and tolerance are tightly regulated by epigenetic mechanisms. Chronic exposure to pro-inflammatory dietary components may promote stable epigenetic shifts favoring heightened innate immune activation and impaired regulatory pathways. Such reprogramming could contribute to sustained low-grade inflammation, altered immune tolerance, and increased susceptibility to immune-mediated disorders, even in the absence of overt immune pathology [4].

Beyond individual health, the possibility of transgenerational epigenetic effects raises broader implications. Experimental and epidemiological studies suggest that nutritional exposures during critical developmental windows such as prenatal

life, early childhood, and adolescence can induce epigenetic modifications that persist into adulthood and, in some cases, influence offspring phenotypes. Diet-induced epigenetic alterations in germ cells or during early embryogenesis may therefore transmit altered disease risk profiles across generations, independent of changes in DNA sequence [5]. Although direct evidence linking processed food consumption to transgenerational effects in humans remains limited, the biological plausibility of this mechanism warrants careful investigation.

Importantly, epigenetic reprogramming is not inherently deterministic. Unlike genetic mutations, epigenetic marks are potentially reversible, offering a window for intervention. Diets rich in whole, minimally processed foods provide bioactive compounds, including polyphenols and methyl-donor nutrients, that support epigenetic stability and adaptive gene regulation. Such dietary patterns have been associated with epigenetic profiles linked to reduced inflammation and improved metabolic resilience, suggesting that nutritional quality may mitigate or reverse maladaptive epigenetic programming [6].

Despite growing interest in nutritional epigenomics, few studies have directly examined the long-term epigenetic consequences of sustained processed food consumption. Longitudinal cohort studies integrating dietary assessment with epigenome-wide analyses could clarify whether specific epigenetic signatures are consistently associated with ultra-processed diets. Furthermore, interventional studies evaluating epigenetic plasticity in response to dietary modification would provide critical insight into the reversibility and clinical relevance of these changes.

In conclusion, this hypothesis proposes that processed food–rich diets may act as chronic environmental signals capable of inducing epigenetic reprogramming with long-term biological consequences. Rather than causing immediate disease, such diets may subtly reshape gene regulatory landscapes, influencing immune function, metabolic balance, and disease vulnerability over time and potentially across generations. Recognizing processed food consumption as a driver of epigenetic imprinting may broaden our understanding of diet-related disease risk and highlight the importance of nutritional quality in shaping long-term health trajectories.

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