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Food preference and inflammation: How taste shapes health

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Abstract

In the modern era, the abundance and accessibility of food have disrupted the ancestral mechanisms of food-seeking behavior and reward. Contemporary dietary choices are heavily influenced by sensory factors, particularly the texture and palatability of food, often favoring calorie-dense, lipid-rich options. However, this shift in dietary patterns, if unbalanced, increases the risk of weight gain and chronic inflammation. Our study investigated the relationship between lipid preference and inflammatory status using Wistar rats. The findings reveal a pronounced preference for sweet and fatty tastes, with the rats being 3.87 times more attracted to sweet flavors and 5.20 times more to fatty acids (linoleic acid) compared to water. Conversely, bitter and salty flavors elicited a rejection of 81% and 76%, respectively. Plasma analyses demonstrated elevated levels of triglycerides and markers of hepatic metabolism following a three-week diet rich in fatty acids. Interestingly, while CRP levels remained unchanged, a significant decrease in IL-10, a regulatory cytokine, was observed, suggesting impaired anti-inflammatory regulation. These results indicate that Wistar rats exhibit a strong inclination toward energy-dense flavors, which, when consumed excessively, modulate hematological, lipidic, hepatic, and immune parameters. Such dietary patterns may predispose individuals to inflammatory states and metabolic disorders, including diabetes and dyslipidemia. This study underscores the critical role of food preference in influencing metabolic and inflammatory markers, providing valuable insights into the interplay between diet and health.

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Introduction

The foods we consume serve as much more than mere sustenance; they are reservoirs of nutrients, medicinal compounds, and energy essential for sustaining life, supporting growth, and promoting well-being. Eating is thus a fundamental act, indispensable to survival. Yet, nutrition also emerges as a double-edged determinant, implicated in the development and prevention of non-communicable chronic diseases, as highlighted by the WHO (2002).

The complexities of dietary behavior have long captured scientific curiosity, rooted in mechanisms governing energy and nutrient regulation to maintain homeostasis (Bellisle, 1999). Among the many modulators of food intake, sensory factors stand out, intricately influencing eating habits (Nicklaus and Bournez, 2016). Today, dietary behaviors in humans and animals alike are shaped by a confluence of external stimuli, including food attributes such as appearance, texture, and increasingly, energy density and sensory appeal (Thibault, 2003).

In recent decades, a profound shift in eating habits has been observed. This includes increased consumption of calorie-dense foods, the widespread availability of processed products, the expansion of dining outside the home, and decreased energy expenditure due to sedentary lifestyles. These changes, collectively termed the "nutrition transition," are a driving force behind the escalating burden of non-communicable diseases (WHO, 2014).

The repercussions of these shifts are stark, evidenced by the rising prevalence of weight-related diseases such as cardiovascular disorders, type 2 diabetes, atherosclerosis, and dyslipidemias (WHO, 2002). These conditions are not mere medical challenges but public health crises, accounting for 41 million deaths globally in 2016—71% of all fatalities (Bennett et al., 2018). Alarmingly, 15 million individuals aged 30 to 69 years succumb annually to non-communicable diseases, with over 85% of these premature deaths occurring in low- and middle-income countries (WHO, 2018). Projections for Africa paint a dire picture: by 2030, the prevalence of such diseases is expected to surpass deaths from infectious diseases, perinatal conditions, and nutritional deficiencies combined (WHO, 2004).

Benin is no exception to this growing trend. A 2015 survey revealed that 23.2% of its population was overweight or obese, 12.4% had elevated fasting glucose levels, and 25.9% suffered from high blood pressure (WHO, 2015). These findings underscore the urgent need to delve deeper into the mechanisms governing dietary behaviors, not only to mitigate associated dysfunctions but also to alleviate the compounded economic and human costs.

Against this backdrop, our study seeks to unravel key questions: What are the gustatory preferences of Wistar rats? How does the consumption of calorie-dense foods influence lipid profiles and the inflammatory status of the organism? To address these, we propose the following hypotheses:

1. The palatability and taste of certain foods significantly influence their consumption, fostering an attraction that shapes dietary behavior.
2. Excessive lipid consumption, when imbalanced, predisposes organisms to weight gain and chronic inflammation, key risk factors for metabolic and cardiovascular diseases.

Through a systematic exploration of these hypotheses, this research aims to shed light on the intricate interplay between dietary choices, metabolic health, and immune regulation, offering insights into strategies for combating the modern epidemic of non-communicable diseases.

Materials and methods

A rigorous examination of dietary and inflammatory profiles

Assessing the impact of sugar- and lipid-enriched diets on lipid, hematological, hepatic, and inflammatory profiles

To evaluate the effects of calorie-dense diets on hematological, lipid, hepatic, and inflammatory parameters, 20 female Wistar rats were divided into

three groups and subjected to dietary interventions enriched with linoleic acid or sucrose:

1. Control Group: 4 rats fed a standard diet (complete feed GVS-lapin engraissement).
2. Group1: 8 rats fed a standard diet supplemented with sucrose.
3. Group 2: 8 rats fed a standard diet supplemented with linoleic acid.

Each group was further divided into two subgroups of 4 rats based on the duration of dietary exposure: 3 days or 3 weeks. At the end of the treatment period, the rats underwent a fasting period of at least 12 hours before blood sampling.

Effects of dietary regimens on hematological parameters

Hematological profiles (complete blood count), as well as lipid and hepatic assessments, were conducted across all groups.

Blood collection and anesthesia

The animals were anesthetized via intraperitoneal injection of thiopental (0.015 mL/10 g body weight). Blood samples were collected retro-orbitally using three types of tubes, tubes without anticoagulant, tubes with EDTA anticoagulant and tubes with sodium fluoride anticoagulant.

Hematological and biochemical analyses

Complete Blood Count (CBC) was performed using EDTA blood samples analyzed with the MINDRAY BC 2800 Cell Counter. Glycemia was measured using the glucose oxidase enzymatic method (BIOLABO Scientific Instrument SA). For lipid Profile, total cholesterol, HDL cholesterol, and triglycerides were quantified using enzymatic assays, while LDL cholesterol was calculated using the Friedewald formula;

$$\text{LDL Cholesterol}\left(\frac{g}{L}\right) = \text{Total Cholesterol} - \{\text{HDL Cholesterol} + \frac{\text{Triglycerides}}{5}\}$$

Hepatic profile: Enzymatic activities of aspartate aminotransferase (AST) and alanine aminotransferase (ALT) were determined using kinetic methods.

Evaluating inflammatory status

C-Reactive protein (CRP)

CRP levels were determined using a CRP-LATEX agglutination test. Latex particles coated with anti-CRP antibodies were mixed with serum samples. The presence of CRP was indicated by visible agglutination within 2 minutes.

Cytokine quantification

Serum levels of IL-2, IL-10, and IFN-γ were quantified using sandwich ELISA kits (Abcam and BioLegend). The sandwich ELISA method employs high sensitivity and specificity for detecting low concentrations of analytes. The process involves (i) Incubating capture antibodies in wells to bind the target analyte; (ii) Washing unbound material, followed by incubation with detector antibodies conjugated to enzymes; (iii) Addition of a substrate (e.g., TMB), producing a colorimetric signal proportional to analyte concentration and (iv) Stopping the reaction and measuring absorbance at 450 nm using a spectrophotometer.

Statistical analysis

Data were recorded in Excel 2007 and analyzed using Student's t-test at a 5% significance level. Differences were considered significant for p-values < 0.05.

This methodology ensures robust and reproducible evaluation of the interplay between dietary intake and inflammatory/metabolic parameters, providing critical insights into the systemic impacts of calorie-dense diets.

Results

Food preferences

Fig. 1 illustrates the volumes of water consumed by Wistar rats in response to different taste solutions. The data indicate that control rats consumed an average of 40 mL of water over the study period. However, rats exposed to sweet and fatty solutions (sucrose and linoleic acid, respectively) exhibited a marked preference, consuming these solutions 3.87 times (sucrose) and 5.20 times (fatty acid) more

than plain water. Conversely, rats exposed to salty and bitter solutions displayed significant aversion, with rejection rates of 76% and 81%, respectively.

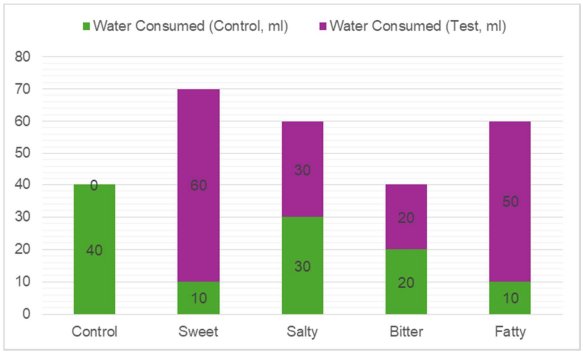

Fig. 1. Gustatory preferences in Wistar rats

Blood parameters

Hematological parameters

The hematological profiles of rats consuming sugar- and fat-supplemented diets are summarized in Table 1 and 2, respectively.

The hematological parameters of Wistar rats exposed to a sweet-flavored diet for 3 days and 3 weeks revealed notable immune system changes. While red blood cell parameters (RBC, hemoglobin, hematocrit, MCV, MCH, MCHC) showed no significant differences compared to controls, white blood cell (WBC) counts significantly increased ($p < 0.05$) after 3 weeks. Neutrophil counts decreased significantly, while lymphocyte and monocyte counts were elevated, indicating adaptive immune activation and sustained immune activity. Eosinophil and basophil counts remained unaffected. These findings suggest that prolonged exposure to a sugar-supplemented diet modulates immune cell profiles, potentially contributing to chronic low-grade inflammation. However, the lack of significant changes in red blood cell parameters indicates that erythropoiesis is not impacted. This highlights the specific effects of a sweet diet on immune regulation rather than general hematopoietic functions.

Table 1. Hematological parameters of rats fed a sugar-supplemented diet

Parameters	Control group- Day 0	Sweet flavor group–Day 3	Sweet flavor group–Day 21
Red Blood Cells (T/L)	6.31 ± 1.7	5.58 ± 1.55 NS	3.93 ± 1.47 NS
Hemoglobin (g/dL)	14.73 ± 1.69	14.07 ± 1.49 NS	10.56±3.2(NS)
Hematocrit (%)	44 ± 05	42 ± 04 NS	33 ± 12 NS
MCV (fL)	72 ± 14	78 ± 13 NS	83 ± 0.5 NS
MCH (pg)	24 ± 05	26 ± 04 NS	27 ± 04 NS
MCHC (%)	33	33 NS	33 NS
White Blood Cells (G/L)	5.28 ± 0.39	5.95 ± 0.58 NS	6.1 ± 0.1 *
Neutrophils (G/L)	1.98 ± 0.45	1.69 ± 0.26 NS	0.99 ± 0.1 *
Eosinophils (G/L)	0.133 ± 0.03	0.101 ± 0.04 NS	0.12 ± 0.06 NS
Basophils (G/L)	0	0 NS	0 NS
Lymphocytes (G/L)	2.91 ± 0.71	3.77 ± 0.85 NS	4.63 ± 0.09 *
Monocytes (G/L)	0.26 ± 0.03	0.39 ± 0.06 *	0.34 ± 0.03 *

Table 2. Hematological parameters of rats fed a fat-supplemented diet

Parameters	Day 0	Fatty diet - Day 3	Fatty diet- Day 21
Red Blood Cells (T/L)	6.31 ± 1.7	5.44 ± 1.18 NS	5.88 ± 1.23 NS
Hemoglobin (g/dL)	14.73 ± 1.69	12.66 ± 1.34 NS	13.68 ± 1.21 NS
Hematocrit (%)	44 ± 05	40 ± 01 NS	40 ± 03 NS
MCV (fL)	72 ± 14	75 ± 14 NS	68 ± 06 NS
MCH (pg)	24 ± 05	24 ± 02 NS	24 ± 02 NS
MCHC (%)	33	33 NS	33 NS
White Blood Cells (G/L)	5.28 ± 0.39	5.73 ± 0.47 NS	5.18 ± 0.16 NS
Neutrophils (G/L)	1.98 ± 0.45	0.97 ± 0.29 NS	0.80 ± 0.21 *
Eosinophils (G/L)	0.133 ± 0.03	0.057 ± 0.04 NS	0.07 ± 0.02 NS
Basophils (G/L)	0	0 NS	0 NS
Lymphocytes (G/L)	2.91 ± 0.71	4.50 ± 0.85 NS	4.15 ± 0.23 *
Monocytes (G/L)	0.26 ± 0.03	0.19 ± 0.04 NS	0.14 ± 0.03 *

The analysis of hematological parameters in Wistar rats subjected to a fatty diet for 3 days and 3 weeks revealed significant immune modulations. Red blood Cell parameters (RBC, hemoglobin, hematocrit, MCV, MCH, MCHC) remained unchanged across all groups.

However, after 3 weeks of a fatty diet, neutrophil counts significantly decreased ($p < 0.05$), while lymphocyte and monocyte counts significantly increased ($p < 0.05$). These changes indicate enhanced adaptive immune activation and sustained immune modulation. No significant differences were observed in eosinophil and basophil counts, nor in white blood cell (WBC) counts at any time point.

Prolonged exposure to a fatty diet selectively alters immune cell profiles, particularly by decreasing neutrophils and increasing lymphocytes and monocytes, suggesting a potential shift in immune and inflammatory responses. These findings highlight the immune-modulatory effects of dietary fats without affecting erythropoiesis.

Biochemical parameters

Biochemical analyses included glucose, total cholesterol, HDL, LDL, triglycerides, and transaminases (ASAT and ALAT) were performed. Results are depicted in Fig. 2 and 3 for sugar- and fat-supplemented diets, respectively.

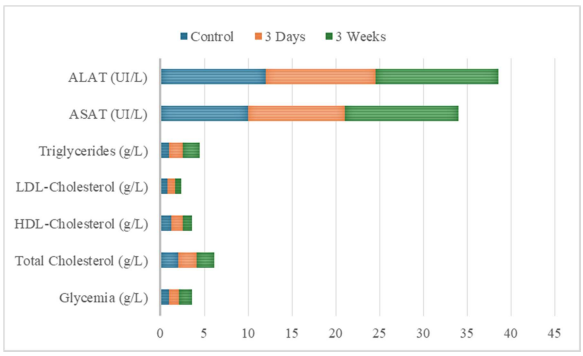

Fig. 2. Biochemical parameters of rats fed with a sugar-supplemented diet

The biochemical parameters of Wistar rats subjected to control, 3-day, and 3-week dietary conditions. Glycemia levels increased slightly over time, with the highest value observed after 3 weeks. Triglyceride levels significantly raised after both 3 days and 3 weeks compared to the control, indicating an impact of the dietary intervention on lipid metabolism. Total cholesterol, HDL-cholesterol, and LDL-cholesterol showed minimal variations across all groups, suggesting a limited effect on these lipid fractions. Enzyme activities (ASAT and ALAT) increased progressively, particularly after 3 weeks, pointing to potential hepatic stress or metabolic adjustments associated with prolonged dietary exposure (Fig. 2).

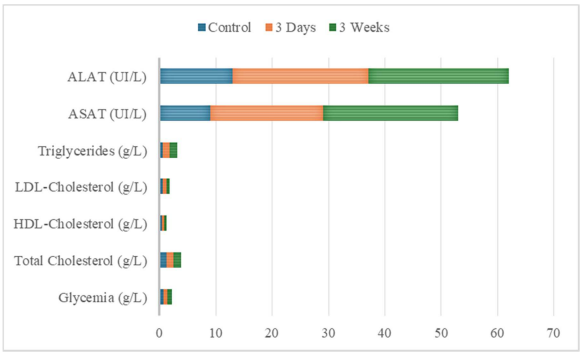

Fig. 3. Biochemical parameters of rats fed with a fat-supplemented diet

The biochemical analysis reveals significant changes in several parameters following 3 days and 3 weeks of dietary intervention compared to the control group. Glycemia levels increased from 0.63 g/L in the control to 0.8 g/L in both 3-day and 3-week groups, suggesting impaired glucose regulation. Triglycerides showed a marked rise, nearly doubling from 0.55 g/L in the control to 1.2 g/L after 3 days and 1.32 g/L after 3 weeks, indicating an impact on lipid metabolism. Total cholesterol and HDL-cholesterol remained relatively stable, while LDL-cholesterol exhibited minor variations. Liver enzyme activities (ASAT and ALAT) significantly increased, particularly ASAT, which rose from 9 UI/L in the control to 20 UI/L and 24 UI/L after 3 days and 3 weeks, respectively (Fig. 3).

Overall, while Fig. 2 and 3 underline similar trends in glycemia and triglycerides, rats fed with fat supplemented diet, provides a clearer depiction of the progressive hepatic impact, emphasizing the cumulative effect of the diet over time.

Table 3. Cytokine levels in Wistar rats

Immune parameters (pg/ml)	Control	Sweet supplemented diet		Fat supplemented diet	
		Day 3	Day 21	Day 3	Day 21
IL-2	2.66±0.6	2.43±0.2 NS	2.27±0.12 NS	1.92±0.16 NS	2.2±0.57 NS
IL-10	2.73±0.3	2.41±0.14 NS	2.47±0.36 NS	2.23±0.3 NS	2.12±0.28 *
IFN-γ	0.51±0.03	0.52±0.04 NS	0.43±0.08 NS	0.45±0.01 NS	0.49±0.02 NS

Inflammatory status

CRP levels showed no significant variations across all experimental groups, indicating that neither sucrose nor linoleic acid influenced CRP levels in Wistar rats. We summarize the effects of sweet- and fatty-flavored diets on immune markers (IL-2, IL-10, and IFN-γ) in Wistar rats (Table 3).

No significant changes (NS) were observed in IL-2 levels across all groups and time points, suggesting that the diets did not strongly influence this cytokine. IL-10 levels decreased significantly ($p < 0.05$) after 3 weeks of the fatty-flavored diet, from 2.73±0.3 in the control group to 2.12±0.28. This reduction indicates potential downregulation of anti-inflammatory activity. IFN-γ levels remained stable with no significant differences (NS) in any group, suggesting that pro-inflammatory responses were not markedly triggered by either diet. While IL-2 and IFN-γ levels remained unaffected, the significant reduction in IL-10 after prolonged exposure to a fatty diet suggests a potential impairment of anti-inflammatory regulation.

Discussion

The act of eating, a complex behavior influenced by physiological and cultural factors, historically served our ancestors as a mechanism to seek and reward food consumption.

However, modern technological advancements and the resulting food abundance have profoundly altered these dynamics, as highlighted by Gaillard *et al.* (2006). Today, food choices are often based on texture and palatability, prioritizing taste over nutritional balance, which can lead to overconsumption and metabolic diseases, as noted by McCrory *et al.* (2000) and Rolls *et al.* (2005).

Our study aimed to investigate food preferences in Wistar rats and their relationship with inflammatory status. Using a two-choice test, a behavioral method for assessing spontaneous preference, we observed that rats displayed a strong attraction to sweet and fatty solutions, corroborating findings by Tsuruta *et al.* (1999), Takeda *et al.* (2000), and Hoebel *et al.* (2008), who demonstrated similar tendencies in rodents. Notably, Ahmed *et al.* (2007) revealed that rats preferred sweet solutions over intravenous cocaine, underscoring the compelling nature of sweet tastes. This preference likely stems from the rewarding and pleasurable properties of sugars and lipids, which activate specialized gustatory receptor cells in oral taste buds and stimulate reward-related neural circuits, including dopaminergic neurons in the ventral striatum (Ahmed, 2013). Such activation explains the inherent attraction to sweet and fatty tastes observed in both animals and humans.

Hematological parameters

Our findings showed that consumption of sweetened and fatty water significantly influenced leukocyte counts, particularly lymphocytes, neutrophils, and monocytes, while erythrocyte parameters remained unaffected. These results contrast with findings by Benallal (2016), who reported increased erythrocytes and leukocytes in rats fed a hypercaloric diet. Similarly, Kouakou *et al.* (2019) observed elevated erythrocytes and lymphocytes but reduced hemoglobin and granulocytes. Discrepancies may stem from differences in calorie concentrations and experimental duration, as outlined by Darimont *et al.* (2004). However, our study aligns with reports of increased lymphocytes and decreased neutrophils, suggesting that sucrose and linoleic acid may suppress neutrophil production while stimulating lymphopoiesis in the bone marrow.

Biochemical parameters

Our biochemical analysis showed progressive increases in glycemia, triglycerides, HDL cholesterol, and ASAT levels in rats fed linoleic acid, while LDL cholesterol decreased. These results align with findings by Benyoub (2011) and Bensalah (2012), who reported increased glucose and triglyceride levels in hyperlipidemic rats. However, differences in HDL and LDL cholesterol may result from lower calorie concentrations and shorter experimental durations in our study.

Our findings on the biochemical parameters in Wistar rats mirror patterns observed in humans consuming high-sugar and high-fat diets. The progressive increases in glycemia and triglyceride levels following linoleic acid consumption are consistent with studies linking excessive dietary fat and sugar intake to insulin resistance and dyslipidemia in humans (Bahia *et al.*, 2006; Choi *et al.*, 2013). Elevated glycemia reflects impaired glucose homeostasis, a hallmark of prediabetes and type 2 diabetes, commonly associated with diets rich in simple carbohydrates and fats. The significant rise in triglycerides, accompanied by stable total cholesterol levels and a decrease in LDL cholesterol, diverges slightly from typical human patterns, where hyperlipidemia usually involves increases in both LDL and total cholesterol. This discrepancy could be attributed to species-specific metabolic responses or differences in dietary fat concentrations and study duration. In humans, prolonged high-fat diets often result in elevated LDL cholesterol and reduced HDL cholesterol, contributing to atherogenic risk (Benyoub, 2011; Bensalah, 2012). The increase in HDL cholesterol in our study may reflect the shorter experimental period and lower calorie content compared to human diets leading to dyslipidemia. The observed increase in ASAT activity highlights hepatic stress, a phenomenon also documented in humans with high-fat diets. Elevated ASAT and ALAT levels in humans are markers of non-alcoholic fatty liver disease (NAFLD), a condition strongly associated with excessive caloric intake and obesity (Fronczyk *et al.*, 2014; Louala, 2017). Similar to our findings in rats, the progression toward liver dysfunction begins with subtle enzymatic changes, preceding overt pathology. The biochemical effects of sucrose and linoleic acid consumption in rats reflect early metabolic changes seen in humans on high-fat and high-sugar diets, emphasizing the importance of diet in modulating metabolic health and the potential for long-term complications such as diabetes, dyslipidemia, and liver disease (Gaillard *et al.*, 2006).

Inflammatory markers and cytokine analysis

The inflammatory markers assessed in this study—CRP, IL-2, IFN-γ, and IL-10—provide insight into the immunological impact of high-sugar and high-fat diets in Wistar rats and offer parallels to human responses. CRP, a widely recognized marker of systemic inflammation, remained unchanged in rats across all dietary groups. CRP levels remained unchanged across all groups, indicating the absence of detectable inflammation. This finding contrast with human and animal studies where hypercaloric diets and excess adiposity were associated with elevated CRP levels (Bahia *et al.*, 2006; Choi *et al.*, 2013; Fronczyk *et al.*, 2014; Tortolano, 2017; Louala, 2017). Elevated CRP is a marker of low-grade inflammation linked to metabolic disorders such as type 2 diabetes (Schmidt *et al.*, 1999). The absence of CRP elevation in our study may reflect the lower sugar and fat concentrations in our experimental diets and the relatively short study duration.

In humans, however, high-sugar and high-fat diets are strongly associated with elevated CRP levels, particularly in individuals with excess adiposity or metabolic syndrome (Bahia *et al.*, 2006; Choi *et al.*, 2013). Elevated CRP in humans is indicative of low-grade, chronic inflammation driven by adipose tissue expansion and pro-inflammatory cytokine release (Ouchi *et al.*, 2003). The discrepancy in our findings could stem from the lower concentrations of dietary fat and sugar in the experimental protocols, as well as the shorter duration of exposure, which may not have been sufficient to induce measurable systemic inflammation.

Cytokines are key immune mediators synthesized primarily by lymphocytes. Our study focused on IL-2, IFN-γ (pro-inflammatory), and IL-10 (anti-inflammatory). IL-2 and IFN-γ levels remained unchanged, likely due to the absence of induced inflammation, as evidenced by stable leukocyte counts. This result is consistent with findings that these cytokines elevate only during active immune responses (Khan, 2006). However, linoleic acid significantly reduced IL-10 levels, aligning with findings by Exel *et al.* (2002), who observed IL-10 reductions in animals with metabolic disorders. Reduced IL-10 may indicate impaired regulatory immune functions, even in the absence of overt inflammation. Our finding highlights the specific impact of dietary fat on the immune balance.

IL-2 and IFN-γ, both pro-inflammatory cytokines, showed no significant changes in our study. In humans, these cytokines typically rise in response to infections or metabolic stress, such as obesity-related inflammation. IL-2 promotes lymphocyte proliferation and immune activation, while IFN-γ mediates inflammation and macrophage activation. Elevated levels of these cytokines are often seen in obesity and type 2 diabetes, where chronic metabolic stress induces immune activation (Khan, 2006; Schmidt *et al.*, 1999). The absence of significant changes in IL-2 and IFN-γ in our study suggests that the experimental diets did not provoke sufficient metabolic or inflammatory stress to trigger an adaptive immune response, possibly due to the short exposure period or mild caloric content.

IL-10, a regulatory cytokine critical for controlling inflammation, decreased significantly in rats consuming a fatty diet for 3 weeks. This aligns with findings in humans, where reduced IL-10 levels are linked to impaired anti-inflammatory regulation in conditions such as obesity and metabolic syndrome (Exel *et al.*, 2002). In humans, decreased IL-10 contributes to an imbalance between pro- and anti-inflammatory responses, exacerbating chronic inflammation and metabolic disorders. The reduction of IL-10 in our study suggests early signs of disrupted immune regulation, even in the absence of overt inflammation (e.g., elevated CRP or leukocytosis).

In humans, high-fat and high-sugar diets are well-documented to induce chronic low-grade inflammation, mediated by adipose tissue dysfunction and an overproduction of pro-inflammatory cytokines such as TNF-α, IL-6, and IFN-γ (Fronczyk *et al.*, 2014; Tortolano, 2017). This inflammation is often accompanied by reduced levels of anti-inflammatory mediators like IL-10, leading to a persistent inflammatory state that underpins metabolic diseases such as type 2 diabetes, cardiovascular disease, and non-alcoholic fatty liver disease. While our study did not observe significant changes in CRP or pro-inflammatory cytokines, the reduction in IL-10 in fatty diet-fed rats mirrors the early immunological disruptions seen in humans.

Conclusion

Does the intestinal microbiota vary depending on the proportion of fats in the body or in consumed foods? How do bacteria modulate eating behavior and the inflammatory status of the organism? These questions lie at the heart of understanding the intricate relationships between dietary habits, lipid/inflammatory status, and the intestinal microbiota.

Our results suggest that dietary sucrose and linoleic acid modulate hematological and biochemical parameters without inducing systemic inflammation, as evidenced by unchanged CRP and cytokine levels. However, the reduction in IL-10 highlights potential subclinical immunomodulatory effects of dietary lipids, warranting further investigation into their long-term impacts on immune regulation and metabolic health. These findings emphasize the critical role of diet composition and duration in shaping physiological and immunological responses.

These findings have important implications for public health. To mitigate the growing prevalence

of early-onset metabolic diseases, we recommend reducing the consumption of sugars and fats by promoting healthier dietary habits, particularly in children. Public awareness campaigns could highlight the benefits of a balanced diet and regular physical activity. Incorporating daily physical exercise would help expend excess energy, reduce fat accumulation, and improve overall well-being.

Ultimately, these combined efforts—dietary changes, increased physical activity, and a deeper understanding of the microbiota's role—could lead to more effective prevention and management of metabolic diseases, fostering healthier lifestyles across populations.

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