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ASSOCIATION BETWEEN HIGH-FAT DIET, GUT HEALTH INDICATORS, AND HEARING THRESHOLDS IN ADULTS: A CROSS-SECTIONAL STUDY

Biological Science

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ABSTRACT

Hearing loss continues to rise globally, with significant functional and socioeconomic consequences. Recent work suggests that gut health and systemic inflammation may influence auditory function through interconnected biological pathways. To evaluate the relationship between dietary fat intake, gut health indicators, systemic inflammation, and hearing thresholds in healthy young adults. This cross-sectional analytical study included 120 adults aged 18–35 years, categorized into high-fat diet (HFD) and normal diet (ND) groups using a validated food frequency questionnaire. Gut health was assessed using the Gastrointestinal Symptom Rating Scale and stool cultures for Lactobacillus and Bifidobacterium species. Serum high-sensitivity C-reactive protein (hs-CRP) was used as a marker of systemic inflammation. Hearing thresholds were measured using pure-tone audiometry, with emphasis on high-frequency averages (4–8 kHz). Data were analyzed using SPSS version 28. Continuous variables were compared using independent t-test or Mann–Whitney U test as appropriate, while categorical variables were analyzed using chi-square test. The relationship between hs-CRP levels and hearing thresholds was analyzed by Pearson correlation. A p-value <0.05 was considered statistically significant. The HFD group demonstrated significantly higher gut symptom scores and lower levels of beneficial gut bacteria ($p<0.001$). Serum hs-CRP levels were elevated in the HFD group (3.8 ± 1.2 mg/L vs. 1.2 ± 0.6 mg/L; $p<0.001$). High-frequency hearing thresholds were significantly higher in the HFD group (mean difference ≈5.7 dB HL; $p<0.001$). A moderate positive correlation was observed between hs-CRP and hearing thresholds ($r = 0.41$, $p = 0.002$). High-fat diets are associated with gut dysbiosis, systemic inflammation, and early high-frequency hearing decline, supporting the gut–inner ear axis hypothesis.

KEYWORDS

High-fat Diet, Gut Microbiota, Hearing Thresholds, Gut–inner Ear Axis

INTRODUCTION

Hearing loss is an increasing global health issue that impacts communication, quality of life, and socioeconomic productivity (World Health Organization, 2021). Modifiable risk factors for early hearing deterioration are being investigated, in addition to age-related degeneration and noise exposure (Goman & Lin, 2016).

Evidence supports complex inter-organ communication networks, such as the gut–inner ear axis, in which intestinal homeostasis may affect cochlear function via immunological, inflammatory, and metabolic pathways (Liu et al., 2021). Dysbiosis, or disturbance of the gut microbiota, has been linked to a variety of chronic diseases, including increased intestinal permeability, endotoxemia, and inflammation (Moreno-Navarrete et al., 2020; Sonnenburg & Bäckhed, 2016).

Dietary patterns, especially high-fat diets (HFD), can alter gut microbiota composition, lowering beneficial bacterial communities like *Lactobacillus* and *Bifidobacterium* and boosting pro-inflammatory states (Haile et al., 2021; Hotamisligil, 2017). Low-grade systemic inflammation, characterized by high CRP levels, has been linked to vascular dysfunction, oxidative stress, and tissue injury, particularly in the cochlea (Kim et al., 2020; Mazurek et al., 2020).

Experiments show that a high-fat diet might cause metabolic and inflammatory changes that accelerate cochlear damage and hearing loss, especially at high frequencies (Hwang et al., 2019). However, human evidence relating fat intake, gut microbiome changes, systemic inflammation, and auditory function is weak, particularly among young, otherwise healthy populations.

Therefore, the present study was designed to evaluate the association between dietary fat intake, gut health indicators, systemic inflammation, and hearing thresholds in young adults, with the aim of exploring the potential role of the gut–inner ear axis in early auditory changes.

MATERIALS AND METHODS

Study Design and Participants: This study was conducted as a Cross-Sectional Analytical Study between March 2024 and August 2025 at Sri Lakshmi Narayana Institute of Medical Sciences, Puducherry. A total of 120 healthy volunteers aged 18–35 years with no prior diagnosed hearing impairment and a willingness to participate in the study. They were recruited through advertisements in local communities and postings on Institutional Notice Boards. Patients with a history of Chronic Gastrointestinal Disease, like Inflammatory Bowel Disease, Diagnosed Hearing Loss, Chronic Otitis Media, Diabetic Mellitus, Autoimmune Disease, or those taking Antibiotics, Probiotics, Nonsteroidal Anti-Inflammatory Medications, and Hearing Aids in the last 3 months were excluded. Pregnant and lactating women were also not eligible. Written Consent was taken from all participants after the research protocol was approved by the Institutional Ethics Committee (Reference No: IEC/C-P/64/2024). The study was conducted and reported in accordance with the STROBE guidelines.

Study Objectives

Primary Objective: To compare high-frequency hearing thresholds (4–8 kHz PTA) between individuals consuming a high-fat diet and those consuming a normal diet.

Secondary Objectives: Evaluating differences in gut health indicators (gastrointestinal symptom scores and stool microbiota composition), comparing serum hs-CRP levels between groups, and assessing the association between systemic inflammation and hearing thresholds.

- To assess the association between systemic inflammation (hs-CRP) and hearing thresholds.

Sample Size Calculation

The sample size was calculated assuming a moderate effect size (Cohen's d ≈ 0.5) for differences in high-frequency hearing thresholds between dietary groups, with a power of 80% and alpha of 0.05, yielding a minimum required sample size of 102 participants. To enhance statistical robustness and account for potential exclusions, 120 participants (60 per group) were included. The recruitment and selection process of participants is illustrated in Figure 1.

Figure 1: Study Flow Diagram

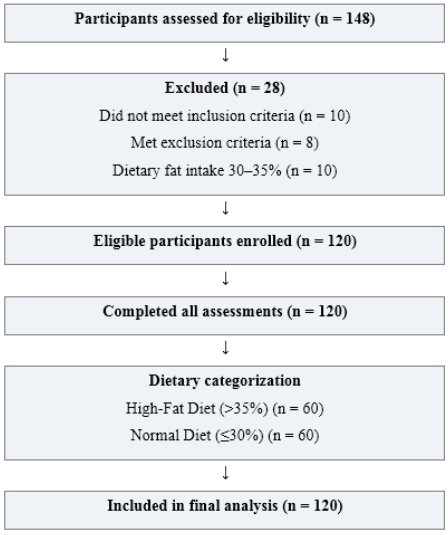

Figure 1: Study flow diagram illustrating participant recruitment, screening, eligibility assessment, and final inclusion of study participants.

Dietary Evaluation and Categorization: Food habits were analyzed using a validated, semiquantitative food frequency questionnaire consisting of 120 items, with a reference period of 12 months prior to data collection. Food composition, including total fat, was analyzed using INDI-FFQ software based on Indian food composition data. The participants were classified according to the percentage contribution of total energy from fat, adapted from established dietary recommendations (National Institute of Nutrition, 2020):

- **High-Fat Diet (HFD) Group:** Individuals consuming >35% of their total daily calories from fat (n=60).
- **Normal Diet (ND) Group:** Individuals consuming ≤30% of their total daily calories from fat (n=60).

Assessment of Gut Health Indicators

- **Gut Symptom Questionnaire:** A validated Gastrointestinal Symptom Rating Scale (GSRS; Revicki et al., 1998) was administered. It assessed the frequency and severity of five symptom clusters (reflux, abdominal pain, indigestion, diarrhea, and constipation) over the past week on a 7-point Likert scale (1=no discomfort to 7=very severe discomfort). A total score and sub-scores were calculated.
- **Stool Culture Analysis:** Participants provided a fresh stool sample in a sterile container. Stool samples were collected on the day of clinical evaluation prior to blood sampling. Samples were processed within 2 hours of collection. Standard microbiological techniques were employed for aerobic and anaerobic cultures on selective media (MRS agar for *Lactobacillus* and *Bifidobacterium* selective agar). Colony-forming units (CFU) per gram of stool were quantified for beneficial bacteria, including *Lactobacillus* and *Bifidobacterium* species.

Assessment of Inflammatory Marker: Venous blood samples from each participant were collected after an overnight fast of 10–12 hours. Serum was separated by centrifugation at 3000 rpm for 15 minutes and stored at -80°C until analysis. Serum high-sensitivity C-Reactive Protein (hs-CRP) levels were quantified using a standardized, particle-enhanced immunoturbidimetric assay on an automated biochemistry analyzer (Cobas c501, Roche Diagnostics). The intra- and inter-assay coefficients of variation were <5%.

Audiological Assessment: Hearing thresholds were evaluated using a calibrated diagnostic pure-tone audiometer (GSI AudioStar Pro) in a sound-attenuated booth meeting ISO 8253-1 standards. Audiological assessments were performed by a trained audiologist who was blinded to participants' dietary group allocation. Air-conduction thresholds were obtained at 0.25, 0.5, 1, 2, 4, 6, and 8 kHz for both ears using the modified Hughson-Westlake procedure (Carhart & Jerger, 1959). The pure-tone average (PTA) was calculated for low (0.5, 1, 2 kHz) and high (4, 6, 8 kHz) frequencies. The hearing threshold for each participant was defined as the better ear average at the specified frequency ranges.

Statistical Analysis: Data were processed with SPSS software version 28.0 (IBM Corp.). Normality testing of continuous variables was performed with the Shapiro-Wilk test. Descriptive data are reported as mean ± Standard Deviation (SD) for normally distributed variables and as median (IQR) for non-normally distributed variables. Categorical data are reported with frequency and percentage. Comparisons between HFD and ND groups on continuous variables were done with the Independent Samples T-test/Mann-Whitney U test according to their normality. Chi-Square test was employed on categorical data. Correlation between serum hs-CRP concentrations and high-frequency hearing thresholds was determined with Pearson Correlation Coefficient. A p-value of <0.05 was taken as significant in all tests.

RESULTS

A total of 120 participants (60 in the High-Fat Diet [HFD] group and 60 in the Normal Diet [ND] group) were considered in the final analysis. The groups were comparable in age, sex distribution, and body mass index (BMI) (Table 1).

TABLE – 1 BASELINE CHARACTERISTICS OF STUDY PARTICIPANTS

Characteristic	High-Fat Diet (HFD) Group (n=60)	Normal Diet (ND) Group (n=60)	p-value
Age (years), Mean ± SD	26.8 ± 4.3	27.1 ± 4.5	0.702
Sex, n (%)			
Male	32 (53.3%)	30 (50.0%)	0.843
Female	28 (46.7%)	30 (50.0%)	
BMI (kg/m²), Mean ± SD	24.1 ± 3.2	23.7 ± 2.9	0.458
Daily Fat Intake (% of total calories), Mean ± SD	38.5 ± 2.8	26.4 ± 2.1	<0.001

Note: Comparison of demographic and clinical characteristics between the High-Fat Diet (HFD) and Normal Diet (ND) groups. Data are presented as mean ± standard deviation (SD) for continuous variables and number (percentage) for categorical variables. Comparisons were performed using the independent-samples t-test or the chi-square test, as appropriate.

Assessment of gut health indicators revealed clear differences between the two groups. The HFD group reported higher gastrointestinal symptom scores on the Gastrointestinal Symptom Rating Scale than the ND group, and median levels of beneficial *Lactobacillus* and *Bifidobacterium* species were significantly lower in the HFD group (p<0.001) (Table 2).

TABLE – 2 GUT MICROBIOTA COMPOSITION (CFU/G STOOL, LOG10 TRANSFORMED)

Bacterial Group	HFD Group, Median (IQR)	ND Group, Median (IQR)	p-value
Lactobacillus spp.	5.2 (4.8 – 5.6)	6.8 (6.4 – 7.1)	<0.001
Bifidobacterium spp.	4.9 (4.5 – 5.3)	7.1 (6.7 – 7.5)	<0.001

Note: Comparison of gut microbiota composition between HFD and ND groups, expressed as median (interquartile range). Quantification of Lactobacillus and Bifidobacterium species was performed using standard culture techniques. Differences between groups were analyzed using the Mann–Whitney U test.

Systemic Inflammation

Serum hs-CRP levels were significantly elevated in the HFD group compared to the ND group (3.8 ± 1.2 mg/L vs. 1.2 ± 0.6 mg/L, p<0.001), indicating a state of low-grade systemic inflammation associated with high dietary fat intake.

Pure-tone audiometry demonstrated a clear difference in hearing acuity between the groups, specifically at higher frequencies. While low-frequency hearing thresholds (0.5-2 kHz PTA) were similar (HFD: 10.2 ± 3.1 dB HL vs. ND: 9.8 ± 2.9 dB HL, p=0.445), high-frequency thresholds (4-8 kHz PTA) were significantly elevated in the HFD group (Table 3, Figure 2).

TABLE – 3 HIGH-FREQUENCY PURE-TONE AVERAGES (DB HL)

Frequency (kHz)	HFD Group, Mean ± SD	ND Group, Mean ± SD	Mean Difference (95% CI)	p-value
4 kHz	15.8 ± 4.6	10.1 ± 3.8	5.7 (4.1 to 7.3)	<0.001
6 kHz	18.5 ± 5.2	12.3 ± 4.1	6.2 (4.4 to 8.0)	<0.001
8 kHz	19.1 ± 5.8	13.9 ± 4.9	5.2 (3.2 to 7.2)	<0.001
4–8 kHz PTA	17.8 ± 4.9	12.1 ± 4.0	5.7 (4.1 to 7.3)	<0.001

Note: Comparison of hearing thresholds at high frequencies (4, 6, and 8 kHz) and overall high-frequency pure-tone average (PTA) between HFD and ND groups. Data are presented as mean ± SD with mean differences and 95% confidence intervals. Statistical comparisons were performed using an independent-samples t-test.

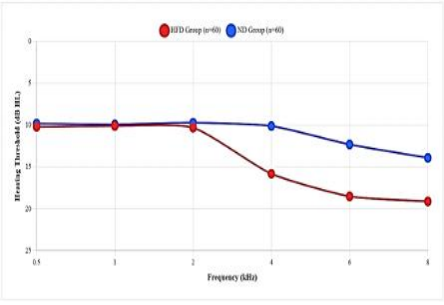

Figure 2: Comparison of pure-tone hearing thresholds across frequencies between High-Fat Diet (HFD) and Normal Diet (ND) groups. The y-axis is inverted (higher values indicate worse hearing). Both groups showed similar thresholds at low frequencies (0.5–2 kHz), but the HFD group demonstrated significantly elevated thresholds at high frequencies (4–8 kHz). The divergence at 4 kHz and beyond indicates early high-frequency hearing impairment linked to higher dietary fat intake. Error bars represent standard deviation. ***p<0.001 for differences at 4, 6, and 8 kHz.

A significant moderate positive correlation was observed between serum hs-CRP levels and the high-frequency (4-8 kHz) pure-tone average across all participants (r = 0.41, p = 0.002) (Figure 3). This suggests that higher systemic inflammation is associated with poorer high-frequency hearing.

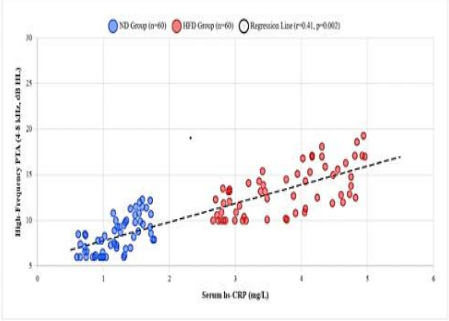

Figure 3: *S*catter plot showing the relationship between serum high-sensitivity C-reactive protein (hs-CRP) levels and high-frequency (4-8 kHz) pure-tone average (PTA) across all 120 participants. Each point represents one participant, color-coded by dietary group. A positive correlation was seen (Pearson's r=0.41, p=0.002), indicating that higher systemic inflammation is associated with poorer high-frequency hearing. The HFD group (red) clustered in the upper-right quadrant (higher CRP, worse hearing), while the ND group (blue) predominantly occupied the lower-left region. The regression line (black dashed) demonstrates the linear relationship between inflammation and auditory decline.

DISCUSSION

This cross-sectional study shows an association between a high fat dietary intake, alterations in gut health indicators, increased systemic inflammation and elevated high-frequency hearing thresholds in young adults. The study showed that adults on a high fat diet (HFD) experienced a combination of effects comprising objectively measured levels of gut dysbiosis with lower expression of key *Lactobacillus* and *Bifidobacterium* species, increased systemic levels of inflammation expressed by high serum hs-CRP, and marked increases in high frequency hearing thresholds inclusive between 4 and 8 kHz relative to those on normal diet (ND) groups. The moderate positive relationship seen between systemic CRP and auditory hearing thresholds suggests a possible link inflammatory processes and early auditory changes. In fact, this observation of commensurate depletion of *Lactobacillus* and *Bifidobacterium* in the HFD group aligns with the existing literature on diet-induced microbial imbalance. Indeed, the preclinical literature confirms that HFD induces rapid decreases in microbial richness and reductions in critical saccharolytic species, helps maintain the integrity of the gut barrier and derives anti-inflammatory compounds such as ScFAs (Cani et al., 2008; David et al., 2014). The severity in human trials confirms these observations, in fact reflecting comparable reductions in *Bifidobacterium* levels in response to HFD in humans with MetS (Metabolic syndrome), whereby such microbial imbalances have contributed to increased gut permeability and endotoxemia (Delzenne et al., 2011; Le Chatelier et al., 2013). These observations in a young, healthy group continue to reflect the hypothesis along these lines, suggesting corresponding microbial pathologies well in advance of MetS manifestation. Supporting these microbial alterations in the HFD group is the corresponding increase in observed gut symptom scores, suggesting a clinic-based manifestation in subclinical gut discomfort, possibly reflecting increased gut permeability, a known harbinger of chronic immune system activation (Martel et al., 2022).

It is established here that elevated serum hs-CRP levels in the HFD group relate to a condition of low-grade, systemic inflammation. This is in agreement with the literature that suggests excessive saturated fat consumption promotes activation of pro-inflammatory signalling, frequently mediated by Toll-like receptor (TLR) signalling in response to the presence of greater circulating microbial products, such as LPS, due to the compromised integrity of the gut lining, in a process described as “metabolic endotoxemia” (Cani et al., 2007; Shi et al., 2006). In the study by Moreno-Navarrete et al. (2020), there is clear elucidation of the mechanism by which the reduction of beneficial bacteria by the HFD leads to disordered gut homeostasis and attendant inflammation, also propagating to other body systems outside the gastrointestinal tract, in agreement with the finding reported in this study regarding the role of hs-CRP in assessing the impact of HFD on the health of the ear and hearing. It must be noted, furthermore, that the observed inflammatory condition in the study described here, with a mean CRP of 3.8 mg/L, most closely resembles sub-clinical, persistent inflammation not due to infection, but of sufficient intensity to potentially impact other body processes (Pearson et al., 2003).

The present study demonstrates reduced high-frequency hearing thresholds in individuals consuming a high-fat diet, with a mean shift of 5.7 dB HL at 4–8 kHz. Although modest, such differences are clinically relevant, as early changes at higher frequencies often precede more generalized hearing loss and are considered important in public and occupational health contexts (Dobie, 2021). This observation is consistent with cochlear physiology, as the basal turn—responsible for high-frequency sound processing—is particularly vulnerable to metabolic stress, oxidative injury, and reduced perfusion (Tavanai & Mohammadkhani, 2017). These findings are supported by experimental studies; for example, Kim et al. (2020) demonstrated that high-fat diets in animal models accelerate cochlear damage, including hair cell loss, increased oxidative stress,

and altered lipid metabolism affecting the stria vascularis and cochlear blood flow. Additional studies have also shown increased expression of inflammatory cytokines within the cochlea in response to diet-induced peripheral inflammation, suggesting a link between systemic inflammation and cochlear injury (Fujioka et al., 2006). In this context, the moderate positive correlation (r = 0.41) between serum CRP levels and high-frequency hearing thresholds further supports the role of inflammation as a potential contributing factor. A similar trend was also seen in epidemiological studies, such as those by Hwang et al. (2019), where elevated CRP and other inflammatory markers were linked to hearing impairment in large population cohorts.

Inflammation can cause hearing impairment in multifactorial ways: it can cause microvascular endothelial dysfunction, and hence reduction in cochlear blood flow; it can cause generation of oxidative stress, resulting in the damage of the vulnerable hair cells and the cochlear ganglion cells; and it can cause the initiation of inflammatory cascades within the cochleae, resulting in the disturbances of the ions in the inner ear environment of the cochleae, respectively (Haase et al., 2011; Wong & Ryan, 2015). This study, with the onset of all four factors of HFD and Dysbiosis, and the association of hearing impairment, provides a clear link (array of their association) or a plausible story of an affected individual's diet, resulting in a disturbed gut environment and hearing loss.

These findings must be contextualized within the nascent but growing field that explores gut-organ axes beyond the gut-brain paradigm. Based largely on animal studies, the concept of a "gut-inner ear axis" has been forwarded (Liu et al., 2021). Liu et al. (2021) provided mechanistic insight by showing that gut microbiota dysbiosis in ageing mice accelerated cochlear senescence, which was ameliorated by fecal microbiota transplantation, strongly implicating microbial factors in auditory ageing. This study provides what seems to be among the first direct human observational evidence for this axis. A related study by Glickman et al. (2021) reported an association between metabolic syndrome (often driven by diet) and hearing loss, but it did not measure gut health parameters. Previous research on nutrition and hearing has largely focused on deficiencies in specific micronutrients, such as vitamins B12 and D, antioxidants, or overall dietary quality (Choi et al., 2014; Mazurek et al., 2020).

In contrast, the present findings suggest a possible link between high-fat dietary intake and gut dysbiosis, systemic inflammation, and subsequent effects on cochlear function in a young adult population.

However, this study has certain limitations. As this was a cross-sectional study, cause-effect relationship could not be assessed. Some factors, including physical activity, stress, and noise exposure, were not fully assessed and could have influenced the results. Dietary and symptom data were collected via recall, which may have introduced bias. Gut microbiota analysis was based on culture methods, which provide limited detail compared with sequencing-based techniques.

Regarding inflammatory markers, CRP is only a partial representation of the inflammatory profile. The inclusion of additional biomarkers, such as IL-6, TNF-α, and circulating LPS levels, would provide a more comprehensive understanding of the underlying mechanisms. Also, as the study population mainly consisted of young adults, it remains unclear whether similar or more pronounced effects would be observed in older individuals.

Despite these limitations, the findings highlight a potential role of dietary patterns in auditory health. Strategies aimed at improving gut health, including increased intake of dietary fibre, prebiotics, and probiotics, may help restore microbial balance, enhance short-chain fatty acid production, and reduce systemic inflammation, thereby offering potential protection to cochlear function (Holmes et al., 2020; Markowiak-Kopeć & Śliżewska, 2020).

Future studies could take this further by following individuals over time to understand better how diet, gut health, inflammation, and hearing change together. Interventional studies looking at dietary modification or probiotic supplementation may also help determine whether these associations are reversible. In addition, exploring the underlying mechanisms—such as inflammatory pathways, metabolites like short-chain fatty acids, and possible neural links including the vagus nerve—would provide deeper insight into how the gut and cochlea may be connected (Bonaz et al., 2018).

CONCLUSION

A high-fat diet in young adults is linked to early changes in gut health, increased systemic inflammation, and measurable shifts in high-frequency hearing. The association between inflammatory markers and hearing thresholds suggests that inflammation is a likely contributor. Together, these findings suggest a meaningful gut–inner ear connection and highlight diet as a potentially modifiable factor in early auditory decline.

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