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Histamine intolerance, DAO deficiency and their impact on health in the context of migraine development and treatment: a review

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ABSTRACT

Introduction and purpose

Migraine is a prevalent neurological disorder affecting up to 1 billion individuals worldwide. Histamine intolerance, caused by reduced activity of the diamine oxidase enzyme, has been increasingly linked to migraines, with headaches being one of its hallmark symptoms. Clinical studies reveal that migraine patients often exhibit significantly lower DAO levels compared to healthy controls. Genetic studies have further identified polymorphisms in the DAO gene as potential contributors to reduced DAO activity and heightened migraine susceptibility. Histamine may influence migraine pathophysiology through elevated plasma levels, which can activate nitric oxide and CGRP pathways, leading to neuronal sensitization and headache onset. Foods high in histamine are common migraine triggers in individuals with HIT or DAO deficiency.

Material and Methods

A comprehensive literature review was conducted using the PubMed and GoogleScholar databases, focusing on articles published since 2020. The search included the keywords: “DAO”, “histamine intolerance”,“migraine”, “DAO supplementation” in varius combination.

Results

DAO enzyme supplementation have demonstrated potential in reducing headache duration in episodic migraines, although their impact on migraine frequency and intensity remains inconclusive. Additionally, recent findings

suggest that serum DAO levels in migraine patients may vary, indicating a complex relationship between histamine metabolism, genetic predisposition, and migraine pathophysiology.

Conclusions
The role of histamine and DAO deficiency in migraine development and underscores the need for further research into targeted dietary and therapeutic approaches. Improved understanding of histamine metabolism and its genetic underpinnings could offer new strategies for managing migraines in histamine-sensitive individuals.

Keywords: DAO, histamine intolerance, migraine, DAO supplementation, migraine risk factors, migraine treatment, chronic migraine, migraine pharmacotherapy, anti-migraine therapy, migraine prophylaxis, headache, CGRP and migraine, non-pharmacological migraine treatment

Introduction

Histamine intolerance (HIT), as classified by the World Allergy Organization, is a non-allergic food hypersensitivity. It arises when the body's ability to break down histamine in the intestines is compromised, often due to reduced activity of the enzyme diamine oxidase (DAO). This reduced capacity can lead to symptoms triggered by dietary histamine, although the exact prevalence of HIT is unclear due to varying estimates influenced by study populations and classifications. Food intolerances in general affect about one in five individuals, with histamine-related reactions included in this category. [1]

Diagnosis of HIT involves assessing a detailed history of food sensitivities through elimination and reintroduction of certain foods. Clinical symptoms that align with histamine intolerance are considered alongside the exclusion of other potential disorders. Symptoms vary widely, necessitating differential diagnoses, but commonly include skin-related issues like eczema, flushing, or hives; gastrointestinal symptoms such as abdominal discomfort and altered bowel habits; and cardiovascular effects like low blood pressure and rapid heartbeat.

Histamine, a biogenic amine derived from the amino acid histidine, can be produced within the body or ingested through food. Internally, histamine is stored in mast cells and basophils, playing a critical role in allergic responses. It is metabolized through two key pathways: methylation via histamine-N-methyltransferase (HNMT) and oxidation via DAO, the latter being particularly crucial for degrading dietary histamine. [1]

Foods rich in histamine, such as aged cheeses, processed meats, fermented fish, and alcoholic beverages like beer and wine, contain biogenic amines formed through bacterial activity. Variations in the levels or activity of DAO may contribute to HIT symptoms, although the exact relationship between DAO activity and histamine intolerance remains under investigation. Studies suggest that individuals with DAO levels below 3 U/mL are at greater risk for HIT, while those with levels above 10 U/mL are less likely to experience intolerance. [1]

Migraine

Migraine is a multifaceted neurological condition, not limited to just headaches but involving a range of sensory and neurological symptoms. [2] These episodes, lasting between 4–72 hours, unfold across four distinct yet overlapping phases: [3]

1. Premonitory Phase: This early phase involves non-painful symptoms such as fatigue, mood shifts, and neck stiffness that can appear hours or even days prior to the headache.
2. Aura (experienced by a subset of patients): Roughly one-third of individuals, especially women, experience temporary neurological disturbances, with visual symptoms being the most common. These include flickering lights or blind spots, lasting 5–60 minutes, and may occur with or without the headache phase.
3. Headache Phase: The hallmark of migraine is intense, often unilateral, throbbing pain due to trigeminal nerve activation. This phase frequently involves nausea, vomiting, and heightened sensitivity to light, sound, and smells, severely impacting daily activities.
4. Postdrome Phase: Following the headache, patients often experience residual effects such as exhaustion, trouble concentrating, and heightened sensory sensitivity, commonly referred to as the "migraine hangover." [2,3,4]

Migraines are classified based on the presence of aura and the frequency of episodes:

- With Aura: Involves transient neurological symptoms like visual scintillations, speech disturbances, or sensory changes. These may occur with or without the headache.
- Without Aura: Lacks the neurological symptoms but shares other migraine features. [3]

To diagnose migraine, a patient must have experienced at least five attacks fulfilling specific criteria:

- Two headache characteristics: one-sided pain, throbbing quality, moderate/severe intensity, or worsening with physical activity.
- One associated symptom: nausea, vomiting, or sensitivity to light and noise. [3]

Frequency-Based Classification

- Episodic Migraine: Occurs on fewer than 15 days per month.
- Chronic Migraine: Defined by headaches on 15 or more days per month, with migraine-specific features present on at least 8 of those days. [3]

The distinction between episodic and chronic migraines relies on counting headache-free "crystal clear" days each month, offering insight into the severity and frequency of attacks. This classification is essential for tailoring effective management and treatment strategies. [2]

Diamine oxidase

Diamine oxidase (DAO) is an enzyme encoded by the AOC1 gene located on chromosome 7 (7q34-36) which degrades histamine. The synthesis of this enzyme takes place mainly in the small intestine, ascending colon, placenta and kidneys. [5]

Decreased DAO activity may affect up to 15% of the population and may be caused by genetic changes single nucleotide polymorphism in the AOC1 gene, inflammatory changes in the small intestine, including ulcerative colitis and Crohn's disease, inflammation of the stomach and intestines, celiac disease, short bowel syndrome and also chronic renal failure, chronic urticaria, liver cirrhosis. DAO may be reduced by vitamin B6 and C deficiency and zinc and copper deficiency, which are DAO cofactors. Alcohol and some medications also reduce DAO levels. [5]

At low concentrations of DAO, allergic reactions, headaches and migraines may occur. [5]

Histamine and histamine intolerance

Histamine is a key mediator within the neuro-immuno-endocrine system, playing a significant role in various physiological and pathological processes. [14] Its synthesis occurs

through the decarboxylation of the amino acid L-histidine, a reaction catalyzed by the enzyme L-histidine decarboxylase. In the human body, histamine is predominantly stored in mast cells and basophils, which are immune cells involved in inflammatory and allergic responses. However, histamine is also found in other locations, such as enterochromaffin cells in the gastrointestinal tract and histaminergic neurons in the nervous system. [7]

The biological effects of histamine are mediated through its action as an agonist at four histamine receptor subtypes: H1, H2, H3, and H4. H1 and H2 receptors are widely distributed throughout the body, though H2 receptors are particularly abundant in the digestive system, including the stomach, duodenum, and small intestine, where they regulate gastric acid secretion. H3 receptors are primarily located in the central nervous system, where they play a critical role in neurotransmission. H4 receptors, on the other hand, are found in smaller quantities in specific tissues, such as the skin and tonsils. [6,8]

Histamine is involved in a wide array of physiological processes. It acts as a key mediator of inflammation, triggering vasodilation, increased vascular permeability, and immune cell recruitment. In the respiratory system, it contributes to congestion, bronchospasm, and increased mucus production. In the gastrointestinal tract, histamine produced by enterochromaffin cells is essential for stimulating the secretion of gastric acid. Beyond its well-recognized roles in the immune and digestive systems, histamine exerts pleiotropic effects within the nervous system, where it functions as both a neuromediator and a neurohormone. It influences critical processes such as thermoregulation, wakefulness, appetite control, as well as cognitive and behavioral functions. [6]

Importantly, food serves as the primary exogenous source of histamine for humans, particularly in fermented or aged products. Given its multifaceted roles in the body, histamine's regulation is essential to maintaining homeostasis and preventing adverse effects associated with its dysregulation, such as allergies, histamine intolerance, and certain neurological disorders. [6]

Histamine intolerance is a type of non-immunological food hypersensitivity with a diverse cause and a wide range of symptoms. It is caused by an imbalance between the amount of histamine in the body and the ability to degrade it. It often coexists with reduced DAO activity. [5,20,21]

Histamine in the diet

Histamine is present in many foods and their presence increases with maturation.

The content of histamine in foods differs in dependence on their source, freshness, types, pH, salt or proteins content, processing and storage. [6]

Biogenic amines are resistant to heat, so cooking generally does not significantly break them down if they are already present in food. However, boiling certain vegetables in water can lower their biogenic amine levels, as the compounds transfer from the food to the boiling water. For instance, boiling spinach reduced its histamine content by 83%, with the lost histamine detected in the water. [6]

On the other hand, heating does not always decrease biogenic amine levels. Cooking methods like boiling and grilling have been found to increase the concentration of biogenic amines in some vegetables, such as aubergines, green beans, and yellow beans. Research showed that grilling seafood and meat raised their histamine levels, while boiling these foods reduced histamine in meat specifically. For vegetables, boiling had little to no effect, with only slight reductions in histamine content in some cases. [6]

Table 1. foods high in histamine [7]

Food category	**Histamine (mg/kg, mg/l)**
Canned tuna	1-402
Smoked or salted/canned Sardine	14–150/3-2000
Cheddar	0-2100
Emmentaler	5-2500
Salami	1-654
Champagne	670
Sauerkraut	0-229
Parmesan	10-581

Eggplant	26
Spinach	30-60

Histamine, DAO and headache

Histamine can induce headaches in both healthy individuals and migraine patients in a dose-dependent manner. This type of headache, classified as a vascular headache, is primarily mediated by nitric oxide. Upon activation of H1 receptors, which are expressed in large intracranial arteries, histamine stimulates the release of nitric oxide from endothelial cells. Studies have shown that migraine patients have elevated plasma histamine levels, both during headache episodes and in symptom-free periods. [7,17]

An increased presence of brain mast cells has been linked to pathological conditions such as migraines, cluster headaches, and multiple sclerosis. Many individuals with migraines exhibit histamine intolerance, which is often associated with reduced activity of the enzyme diamine oxidase (DAO). This intolerance can trigger headaches after consuming histamine-rich foods, such as aged cheese or wine. Symptoms can often improve or resolve entirely with a histamine-free diet and treatment with antihistamines. [3,16]

The precise pathophysiological mechanisms behind headaches remain unclear, and migraines are often considered difficult to treat. Biogenic amines found in wine, such as histamine, tyramine, and putrescine, are known to contribute to headaches and histamine-related symptoms. Notably, red wine contains significantly higher concentrations of histamine—over 2200 µg/L—compared to white wine, which contains approximately 900 µg/L. [19]

Studies have shown that certain DAO (diamine oxidase) genotypes and allelic variants are linked to an increased risk of migraines. A high prevalence of DAO deficiency, affecting nearly 90% of migraine patients, has been reported. Recent findings indicate that oral supplementation with DAO capsules can significantly reduce the frequency and severity of headaches in migraine sufferers. Similarly, it was demonstrated that headaches, as one of the

many symptoms of histamine intolerance (HIT), were markedly alleviated with oral DAO therapy. [19]

Additionally, there is a hypothesized connection between migraines, celiac disease, non-celiac gluten sensitivity, and reduced DAO activity. [12] Patients with low serum DAO levels appear to experience more severe migraine symptoms compared to individuals with normal DAO activity. Notably, 63% of patients with histamine intolerance reported bloating combined with headaches as one of the most common symptom pairings. [19]

These findings suggest that the histamine content in food may play a significant role in triggering headaches and migraines. However, further research is needed to fully understand these associations and develop targeted interventions. [19]

As previously noted, certain genetic variants of the DAO gene have been linked to an increased susceptibility to migraines. [13] Research has demonstrated that DAO activity in blood samples is significantly reduced in migraine patients compared to healthy individuals. In one study, a cohort of 137 migraine sufferers exhibited notably lower DAO activity than 61 healthy controls. A clear deficiency in DAO, defined as less than 80 histamine-degrading units (HDU)/ml, was more frequently observed in the migraine group than in the control group. [10]

From a clinical perspective, these findings suggest that implementing a low-histamine diet or, in cases of DAO deficiency, DAO supplementation therapy could serve as a promising strategy for migraine prevention or as a prophylactic approach to reduce the frequency and severity of chronic migraines. [10]

While uncertainties remain, the existing evidence allows for the formulation of three potential hypotheses linking dietary (exogenous) histamine to CGRP (calcitonin gene-related peptide) signaling in migraine generation. [10]

Hypothetical Mechanism 1

A diet rich in histamine could stimulate the local release of CGRP, which then diffuses into the peripheral bloodstream, leading to elevated plasma CGRP levels. This increase may sensitize afferent neurons within the spinal and trigeminal sensory systems, creating a persistent state of hyperexcitability that facilitates the onset of migraines. To test this hypothesis, the first step would involve measuring CGRP plasma levels and assessing CGRP release from trigeminal tissues in vivo after consuming a high-histamine diet. [10]

Hypothetical Mechanism 2

Excess dietary histamine may activate peptidergic afferent neurons in the gastrointestinal tract, causing enhanced neurotransmitter and CGRP release in the spinal dorsal horn. This could result in central sensitization that extends to the trigeminocervical complex, increasing the likelihood of migraine attacks. To evaluate this mechanism, researchers could measure CGRP concentrations in the spinal dorsal horn and trigeminocervical complex following high dietary histamine intake. [10]

Hypothetical Mechanism 3

Accumulated histamine in the gut may enter the bloodstream after excessive dietary intake, leading to a sustained increase in plasma histamine levels. Elevated plasma histamine could progressively sensitize afferent neurons throughout the body, raising the likelihood of nociceptive events. In the **t**rigeminovascular system, this process may amplify trigeminal afferent activity, thereby promoting migraine development. To explore this hypothesis, the first step would involve measuring plasma histamine levels after consuming a histamine-rich diet. [10]

These hypotheses provide a framework for further investigation into the role of dietary histamine and CGRP signaling in migraine pathophysiology, highlighting potential avenues for targeted research and therapy. [10]

In summary, there appears to be a bidirectional functional relationship between histamine and CGRP, although the exact mechanisms underlying this connection remain unclear. One possible link is that exogenous histamine could drive CGRP activity within the trigeminovascular system. Consequently, elevated intestinal histamine levels—often associated with DAO deficiency—may contribute to an increased risk of migraine attacks by promoting fluctuations in CGRP levels. [10]

Migraine is a widespread neurological disorder and the third most common condition globally, affecting up to 1 billion people. Histamine intolerance (HIT), which results from a deficiency of the diamine oxidase (DAO) enzyme, is frequently associated with headaches, one of its most well-documented symptoms. In a clinical study involving 198 participants, subjects were divided into migraine and control groups, and DAO levels were measured using ELISA. The findings revealed that mean DAO levels were significantly lower in migraine

patients compared to healthy individuals. Furthermore, DAO deficiency was observed in 87% of migraine patients, a much higher prevalence than in the control group. [11,18]

In another randomized controlled trial (RCT) 1 month of oral DAO enzyme supplementation led to a reduction in headache duration by 1.4 hours in patients with episodic migraines. However, the treatment did not significantly impact migraine frequency or pain intensity. [11]

The connection between DAO deficiency and migraines appears to have a genetic basis. A recent analysis of 22 genome-wide association studies identified 38 genes as susceptibility loci for migraines. Another study reported that the C2029G polymorphism in the DAO gene is significantly more common in migraine patients than in healthy controls. Additionally, the C314T variant in the HNMT gene and the C2029G DAO polymorphism were found to interact, further increasing the risk and severity of migraines. Research examining allelic variants also identified the DAO SNP rs10156191 as being associated with reduced DAO activity and a higher risk of migraine onset, particularly in women, suggesting both genetic and sex-based influences on migraine susceptibility. [11]

Interestingly, a recent study investigating the relationship between serum DAO levels, histamine concentrations, and three DAO gene polymorphisms (rs10156191, rs1049742, and rs1049793) found no significant differences in the frequency of DAO gene variants between migraine patients and controls. Surprisingly, serum DAO levels were actually higher in migraine patients compared to the control group, highlighting the complexity of the relationship between DAO activity, histamine metabolism, and migraine pathophysiology. [11,15]

Summary

Histamine, a key mediator in the neuro-immuno-endocrine system, plays a critical role in various physiological and pathological processes.

Histamine intolerance (HIT), often associated with reduced diamine oxidase (DAO) activity, arises from an imbalance between histamine intake (primarily from food) and the body's ability to degrade it. Foods rich in histamine, such as aged cheese, wine, sauerkraut, and canned fish, can trigger symptoms, including headaches and migraines, in susceptible individuals.

Migraines, a widespread neurological disorder, have been closely linked to elevated histamine levels, DAO deficiency, and specific genetic variants of the DAO gene (e.g., C2029G polymorphism and SNP rs10156191). This deficiency may cause increased plasma histamine, which can trigger migraines via mechanisms involving nitric oxide and CGRP (calcitonin gene-related peptide) signaling.

Three proposed mechanisms suggest that dietary histamine could increase CGRP levels, leading to neuronal sensitization and migraine onset through peripheral and central pathways. While oral DAO supplementation has shown promise in reducing headache duration, its effects on migraine frequency and intensity remain limited.

In conclusion, histamine plays a significant role in migraine pathophysiology, particularly in individuals with DAO deficiency or histamine intolerance. Further research is needed to better understand the relationship between histamine metabolism, DAO activity, and genetic predisposition, which may pave the way for targeted dietary and therapeutic interventions.

Disclosures

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