Headache is one of the most common complaints voiced in a doctor’s examination room. Many of these headaches are "benign" in the sense that they are brought on by stress or anxiety, the amount of discomfort is mild to moderate, and relief is achieved within a few hours. A second type of headache occurs secondary to a medical illnesses, usually a minor condition such as the flu. Only in rare cases are these headaches a sign of serious conditions such as cerebral aneurysms or strokes. A third type of headache, affecting 10-20% of the population, is a migraine (1).

Migraines consist of periodic attacks of hemicranial pain, vomiting, photophobia, phonophobia, tiredness, irritability, and impaired concentration. In the case of a classical migraine, the headache itself is preceded by an "aura" consisting of visual problems, generally blind spots or hallucinations. A common migraine contains no premonitory aura (2). Several hypothesis have been proposed to account for the various features of a migraine, yet no clear mechanism exists to explain why or how a migraine headache occurs. Certain foods, such as chocolate, wine, and cheese, have been suspected of initiating migraines. It is the purpose of this paper to evaluate the evidence for dietary triggers of migraines, suggest a mechanism by which specific molecules in food could induce a migraine, and make recommendations regarding dietary treatment for migraine sufferers.

Research on the Role of Food in Migraine Headaches

Studies implicating food in the pathogenesis of migraines date back to the 1920’s when researchers began to examine and manipulate the diets of individuals suffering from migraines. In order to determine which foods might initiate a migraine, a method consisting of "elimination diets" was utilized. After initial evaluation, patients began an oligoantigenic diet consisting of one meat (lamb or chicken), one carbohydrate (rice or potato), one fruit (banana or apple), one vegetable (brassica), water, and vitamin supplements. This diet was continued for 3-4 weeks at which time, provided the headaches resolved, food challenges were added to the diet each week to detect which foods might be responsible for the migraines. Throughout this process, headache activity was recorded (3).

While the early studies indicated that many individuals appeared sensitive to certain foods, very few scientific studies in this area were performed until the 1980’s. Once again, the oligoantigenic diet with food challenges was used to determine specific foods which induced migraines in individuals. The individuals were then placed on diets which eliminated the determined triggers. In a study of eighty-eight children, 88.6% recovered completely, 4.5% improved greatly, and 6.8% showed no improvement. Of the eighty-two children who recovered or improved, 90% relapsed when the known triggers were added back to the diet (4). A similar study of 104 adults, 38.5% showed a 50% or greater reduction in headaches on the elimination diet (3). Berg and Braham (1994) confirmed these results in an equivalent study; 34.6% of the adults and 82.2% of the children reported a 50% or greater reduction in migraine attacks. In each of these experiments, the number of foods which were determined to trigger migraines in a given individual was between one and four. The most common foods implicated in migraine attacks were red wine, cheese, chocolate, and citrus fruits (5).

Red wine is the food item most often cited as a trigger for migraines (5,6,7). Histamine, the molecule in wine which is believed to induce headaches, is also present in egg whites, some types of fish, strawberries, tomatoes, and citrus fruits. Wantke et. al (1994) used red wine to precipitate headaches in patients suffering from histamine intolerance, a disease characterized by a loss of the enzyme responsible for breaking down excessive histamine. Plasma histamine levels were found to increase during the wine administration and during subsequent headaches. Further, when patients were given H1 (histamine receptor) blockers, the symptoms of wine intolerance and the headaches were eliminated (7).

Tyramine, a compound found in aged cheeses, fermented sausages, sour cream, and wine, has long been believed to elicit headaches. Double-blind, placebo-controlled experiments using challenges with foods containing tyramine were shown to induce headaches in 80% of patients prone to headaches (9). However, additional studies using the same protocol and same quantity of tyramine (125mg) failed to replicate these findings (10,11). While the evidence for tyramine as a factor in inducing a migraine is controversial, many tyramine-free diets are available in health food stores.
Food challenge experiments have also been performed with phenylalanine, another molecule found in certain cheeses, red wines, and chocolates. Headaches were produced in 50% of the individuals tested (12). Once again, there are contradictory results which show no increase in migraine activity upon ingestion of foods containing phenylalanine (13).

Mechanism of Food Induced Migraines: A Proposed Hypothesis

In the hundred years since migraines were recognized as a unique, severe type of migraine, several hypotheses have been put forth which attempt to explain the initiation of migraine headaches. A neural hypothesis suggests that overactivity of the raphe nucleus within brainstem results in the release of serotonin, a vasoactive compound which acts on cerebral blood vessels (1). A theory of spreading cortical depression asserts that electrical events or chemical compounds induce a spreading region of neural inactivity leading to reduced blood flow (2). A trigemino-vascular theory states that the irritation of the peripheral endings of the trigeminal nerve initiates the release of vasoactive compounds near cerebral blood vessels (14).

Regardless of the mechanism by which a migraine is initiated, most theories support the idea that the blood vessels in the brain undergo a temporary vasoconstriction followed by a period of prolonged vasodilation. Studies of cerebral blood flow support these vascular events, showing initial hypoperfusion followed by hyperperfusion (15). It is the period of vasodilation which correlates to the throbbing pain of a migraine.

The vascular fluctuations during migraine can be regulated using pharmacological agents. The observed clinical efficacy with these drugs yields insight into the physiology of vasoconstriction and vasodilation during migraine attacks. Dihydroergotamine and Sumatriptan, the drugs used most frequently to treat acute migraine episodes, are serotonin receptor agonists. Activation of the serotonin receptors on the smooth muscle surrounding the blood vessels leads to vasoconstriction. In addition, serotonin may act as a neuromodulator on the presynaptic terminal of neurons, turning on or off the release of vasoactive neuropeptides (16).

The release of vasoactive compounds controlling vasoconstriction and vasodilation of cerebral arteries are the events that may be modulated by compounds within certain foods. Histamine is a compound which stimulates the synthesis of nitric oxide (NO) in endothelial cells. The diffusion of NO into surrounding smooth muscle cells causes them to relax, thus producing vasodilation (17). Thus, consuming foods high in histamine, especially in individuals that lack the enzyme for breaking down excessive histamine, could plausibly lead to the vasodilation associated with the late stages of migraines. The role of tyramine and phenylalanine in the production of migraines is different than that of histamine. The chemical structure of tyramine and phenylalanine resemble that of the catecholamine neurotransmitters. Thus, these molecules could act as serotonin agonists, causing the vasoconstriction in the early stages of migraines by either directly causing constriction of the smooth muscle or by inhibiting the release of molecules that normally allow for smooth muscle relaxation.

Advising a Patient About Dietary Therapy for Migraines

The question inevitably arises as to whether or not a physician should recommend the restriction of certain foods in order to prevent migraine attacks. The results of many studies relating food and migraine activity are inconclusive. Whereas one experiment shows that a certain food or compound may cause migraines in individuals, an equally valid study contradicts it. In light of any clear cut answer, treatment decisions should be made on an individual basis with the involvement of the patient. Highly effective pharmacological treatments are available for both acute migraine attacks and prevention of future migraine headaches. Thus, dietary restrictions to avert migraine are usually not necessary and might be burdensome for the patient. On the other hand, some patients prefer to forego pharmacological treatments when more natural methods are available. With such a patient, one might recommend the elimination of a few of the foods most commonly associated with migraines from the patient’s diet. Until more definitive answers become available, a physician must use good judgment and keep an open mind in the treatment of migraines.
REFERENCES


