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## **Tension-Type Headache. Systematic review of the most widespread primary headache disorder**

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**ABSTRACT****Introduction**

Tension-type headache (TTH) is a primary headache disorder defined by the International Classification of Headache Disorders 3<sup>rd</sup> edition (ICHD-3) and is one of the most common types of headache worldwide. It is characterised by a bilateral, pressing or tightening pain of mild to moderate intensity and, unlike migraine, it is not accompanied by nausea or vomiting. It is commonly regarded as a non-harmful condition, however its significant prevalence in the general population establishes a substantial global health burden.

### **Aim of the study**

The purpose of this study was to review and summarize the available knowledge on the epidemiology, physiology, diagnosis, and clinical recognition of tension-type headache, and to discuss the difficult problems related to this disorder.

### **Materials and methods**

The data used for this review were collected from PubMed, Google Scholar, and the International Classification of Headache Disorders, 3rd edition. Literature search was carried out using the keywords “Tension-Type Headache”, “Primary Headache Disorders” and “Migraine Disorders”.

### **Conclusion**

Tension-type headache is the most common primary headache disorder in the general population, however it is underdiagnosed in clinical practice and research. Its complex, multifactorial pathophysiology underscores the need for independent therapeutic strategies for the patients, also for further research of the mechanistic cause of the disease. A correct distinction from migraine is indispensable to make a good diagnosis, especially because of the presence of overlapping symptomatology. The systematic application of headache diaries is important for clinical evaluation, although further validation and normalization of these are required to enhance uniformity in clinical and research settings. The integration of non-pharmacological treatments as adjunctive strategies should be further explored, especially in patients with chronic TTH. In paediatric population, where the clinical presentation may differ, diagnostic criteria require validation to ensure accurate recognition and management. Ultimately, further research is needed to refine our understanding of TTH and enhance patient care across diverse age groups and clinical settings.

### **Keywords**

Tension-Type Headache; Headache Disorders, Primary; Migraine Disorders.

### **Epidemiology**

Global burden assessments, such as those conducted by the Global Burden of Disease (GBD) study, have demonstrated that tension-type headache (TTH) affects nearly two billion individuals worldwide, with approximately 9.75 million cases reported in Poland (1). In this way, it contributes greatly to the global burden, measured by years lived with disability (YLDs), but when calculated per patient, it is lower compared to migraine (2). It is estimated that approximately 78% of the general population will experience TTH at some point during their lifetime; nevertheless, it remains the most underrecognized and underestimated primary headache disorder (3,4). The economic burden is high because of the fact, that many individuals with TTH do not seek medical help due to the typically mild and moderate nature of most episodes. But because of the fact, that TTH is so common, it leads to big overall losses in job productivity (1). Gender differences in the epidemiology of TTH appear to be modest when compared to other headache disorders such as migraine, which shows a strong female predominance. Nevertheless, the gender disparity is markedly less pronounced than in migraine, with reported female-to-male ratios frequently approaching 1.2:1 (5,6).

## **Pathophysiology**

Theories explaining the pathophysiological basis of TTH can be categorized into classical and more recent concepts. It is now understood, that TTH results from a many interactions of peripheral and central mechanisms, rather than just from stress or prolonged muscle contraction (7). The pathophysiological process of TTH can be divided into a series of successive stages, which begin at the peripheral level and ultimately lead to changes within the central nervous system and central sensitization (8). For many years, increased tension and contraction of the pericranial muscles were considered as a key mechanism in the development of TTH (9). However, electromyographic (EMG) studies have shown that muscle tension in TTH patients is not consistently elevated and does not demonstrate a strong association with headache occurrence (10). Similar to increased tension and contraction of the pericranial muscles, myofascial tenderness has been considered a significant mechanism in the development of TTH. This symptom itself is observed in the majority of patients with TTH (11). Myofascial tenderness is present not only during headache episodes but also, in some patients, between attacks (12,13). For many years, it remained unclear whether tenderness preceded the headache or results from it (14). Today, however, it is known that it is associated with mechanisms of peripheral and central sensitization - a process that plays a key role in the pathophysiology of TTH.

At the most fundamental level, the peripheral mechanism of TTH is believed to involve the activation of hyperexcitable afferent nociceptive neurons located in the muscles of the head and neck (15). This process is triggered by physical stressors such as sustained or repetitive muscle activity, poor posture, or mechanical overload, which activate muscle nociceptors by mechanical, chemical, or ischemic stimuli (16). The activation of these nociceptors leads to the local release of inflammatory mediators - including substances like bradykinin, serotonin, prostaglandin E<sub>2</sub>, and calcitonin gene-related peptide (CGRP), which further lower the threshold for pain and promote peripheral sensitization (16).

Once established, the peripheral nociceptive input undergoes amplification through the process of peripheral sensitization, then the nociceptors in the afflicted muscles become more and more responsive to stimuli that would normally be subthreshold for pain induction (16). The persistent nociceptive signal is transmitted via thinly myelinated A $\delta$  fibers and unmyelinated C fibers to the dorsal horn of the spinal cord and the spinal nucleus of the trigeminal nerve (17). The convergence of these impulses leads to the summation of sensory input, forming the basis for the second key stage in the pathophysiology of TTH (18). Under normal conditions, such convergence is regulated by descending inhibitory pathways. However, repetitive and prolonged nociceptive input may overwhelm these mechanisms, resulting in central sensitization (8). Central sensitization is characterized by a lowered activation threshold of neurons in the dorsal horn and higher neural centres, which means that even painless stimuli can cause exaggerated pain response (18).

The specific genetic factors contributing to the development of TTH remain unidentified (19). However, some studies suggest a potential role of the serotonin transporter gene polymorphism (5-HTTLPR), particularly in individuals with chronic tension-type headache (CTTH) (20,21). Additionally, polymorphisms in the catechol-O-methyltransferase (COMT) gene have been associated with increased sensitivity to pressure-induced pain, which may have connection with symptom severity in TTH, especially in CTTH (22). Further research is required to explore these genetic associations and their potential impact on the clinical presentation and phenotype of TTH.

The pathophysiology of TTH is recognized as a complex process, initiated by peripheral nociceptive input. This input contributes to sensitization at the peripheral level, which, in turn, promotes central nervous system hyperexcitability and dysfunction of descending inhibitory pathways (17,18). Ongoing research in this area is important to create better treatment strategies that could help manage TTH more effectively and precisely.

### **Diagnostic Criteria According to ICHD-3**

The diagnostic framework for TTH has evolved over the past decades. Initial efforts to classify headache disorders culminated in the publication of the first edition of the International Classification of Headache Disorders (ICHD) by the International Headache Society (IHS) in 1988 (23). Subsequent revision - ICHD-2 in 2004 and the beta version of ICHD-3 in 2013 - reflected growing clinical evidence and feedback from practitioners worldwide (24,25). The current and definitive version, ICHD-3, was released in 2018 and provides the most comprehensive and clinically validated criteria to date (26). An short overview and comparison of the diagnostic frameworks for TTH will be presented in this section.

Three main phenotypes of tension-type headache can be distinguished: infrequent episodic TTH, frequent episodic TTH, and chronic TTH. Diagnosis requires that all criteria from A to E are fulfilled. Additionally, depending on the presence or absence of pericranial tenderness, each phenotype may be further subclassified into forms with or without pericranial tenderness.

Table 1. Comparison of Diagnostic Criteria for Tension-Type Headache

Infrequent episodic TTH	Frequent episodic TTH	Chronic TTH
A. At least 10 episodes of headache occurring on <1 day/month on average (<12 days/year) and fulfilling criteria B–D	A. At least 10 episodes of headache occurring on 114 days/month on average for >3 months (12 and <180 days/year) and fulfilling criteria B–D	A. Headache occurring on 15 days/month on average for >3 months (180 days/year), fulfilling criteria B–D
B. Lasting from 30 minutes to seven days	B. Lasting from 30 minutes to seven days	B. Lasting hours to days, or unremitting
C. At least two of the following four characteristics: 1. bilateral location 2. pressing or tightening (non-pulsating) quality 3. mild or moderate intensity 4. not aggravated by routine physical activity such as walking or climbing stairs	C. At least two of the following four characteristics: 1. bilateral location 2. pressing or tightening (non-pulsating) quality 3. mild or moderate intensity 4. not aggravated by routine physical activity such as walking or climbing stairs	C. At least two of the following four characteristics: 1. bilateral location 2. pressing or tightening (non-pulsating) quality 3. mild or moderate intensity 4. not aggravated by routine physical activity such as walking or climbing stairs
D. Both of the following: 1. no nausea or vomiting 2. no more than one of photophobia or phonophobia	D. Both of the following: 1. no nausea or vomiting 2. no more than one of photophobia or phonophobia	D. Both of the following: 1. no more than one of photophobia, phonophobia or mild nausea 2. neither moderate or severe nausea nor vomiting
E. Not better accounted for by another ICHD-3 diagnosis.	E. Not better accounted for by another ICHD-3 diagnosis	E. Not better accounted for by another ICHD-3 diagnosis.
Developed based on: Headache Classification Committee of the International Headache Society (IHS). The International Classification of Headache Disorders, 3rd edition. Cephalalgia. 2018 Jan 1;38(1):1–211.		

The primary criteria distinguishing the phenotypes of TTH are the frequency and duration of headache episodes (criteria A and B). Criteria C refer to the pain characteristics, which are consistent across all three TTH phenotypes. Criteria D address features typical of migraine, the presence of which excludes a diagnosis of TTH. Criteria E pertain to other headache disorders classified in the ICHD-3, which likewise preclude a definitive diagnosis of TTH.

If one of the criteria from A to E is not fulfilled, a diagnosis of probable TTH can be made. The table below summarizes the diagnostic criteria for the various subtypes of probable TTH.

Table 2. Comparison of Diagnostic Criteria for Probable TTH		
Probable infrequent episodic TTH	Probable frequent episodic TTH	Probable chronic TTH
A. One or more episodes of headache fulfilling all but one of criteria A–D for infrequent episodic tension-type headache	A. Episodes of headache fulfilling all but one of criteria A–D for frequent episodic tension-type headache	A. Headache fulfilling all but one of criteria A–D for chronic episodic tension-type headache
B. Not fulfilling ICHD-3 criteria for any other headache disorder	B. Not fulfilling ICHD-3 criteria for any other headache disorder	B. Not fulfilling ICHD-3 criteria for any other headache disorder
C. Not better accounted for by another ICHD-3 diagnosis.	C. Not better accounted for by another ICHD-3 diagnosis	C. Not better accounted for by another ICHD-3 diagnosis
Developed based on: Headache Classification Committee of the International Headache Society (IHS). The International Classification of Headache Disorders, 3rd edition. Cephalalgia. 2018 Jan 1;38(1):1–211.		

### **Distinguishing Tension-Type Headache from Migraine**

The most common diagnostic challenge among primary headache disorders is distinguishing between TTH and mild forms of migraine without aura (26). This distinction is particularly difficult, as patients with frequent headaches may meet criteria for both diagnoses. Pain intensity in TTH is typically described as mild to moderate and lasts from 30 minutes to 24 hours, whereas migraine pain is moderate to severe in intensity and typically lasts from 4 to 72 hours (26,27). In individuals with coexisting TTH and migraine, headache intensity and frequency may be higher compared to those without migraine (28). Regarding localization, TTH pain is generally bilateral, pressing or tightening in quality, and not aggravated by physical activity (19,29).

In contrast, migraine pain is most often unilateral and pulsating in nature (27,26). However the common perception of migraine as a unilateral headache disorder, approximately 40% of patients report bilateral pain, whereas strictly unilateral headache is reported by around 26% (30,31). Phonophobia and photophobia are markedly more characteristic of migraine than of TTH, as are nausea and vomiting. However mild nausea may occasionally accompany CTTH (26,32). Autonomic symptoms such as eye redness and tearing are observed in migraine, but are absent in TTH (33,34).

### **The Role of Headache Diaries**

The use of headache diaries represents a key component in the management of patients with migraine and other headache disorders. These tools offer a good method for patients to record headache episodes and associated characteristics. In recent years, digital formats - particularly mobile applications - have gained prominence, enhancing accessibility and patient engagement in routine monitoring (35,36). Particularly important are responses to questions such as the frequency of headache days, the frequency of medication use, and the degree of impairment in daily functioning (35). Headache diaries enhance patient engagement by empowering individuals with information about their own symptom patterns. This increased self-awareness can lead to improved adherence to treatment regimens and active participation in self-management strategies (37,38). Moreover, headache diaries have demonstrated significant value in non-pharmacological approaches, such as psychotherapy (39). A major limitation however, lies in the heterogeneity of existing headache diaries. Standardized headache diaries should include clear definitions of what constitutes a headache episode, uniform pain intensity scales, and consistent protocols for documenting potential triggers or associated symptoms. Such standardization would facilitate more reliable comparisons across studies and ultimately contribute to more effective patient care (37,40).

### **Neuroimaging**

Routine neuroimaging in TTH has been widely debated, as numerous studies have demonstrated a very low efficiency for detecting clinically significant intracranial pathology in patients without focal neurologic signs or other “red flags” (41,42). Given the high prevalence of TTH in the general population and the low incidence of abnormal radiologic findings, imaging is typically reserved for selected cases. Numerous guidelines based on systematic reviews and meta-analyses indicate that CT imaging in patients with TTH and a normal neurologic examination rarely reveals clinically significant abnormalities (42,43). Nevertheless, advanced MRI techniques have provided evidence of subtle structural brain alterations in TTH patients, including increased grey matter volume in specific cortical areas and a higher prevalence of white matter hyperintensities (44,45). The implications of these findings suggest that chronic TTH might be linked to repetitive nociceptive input affecting brain regions involved in pain processing, as mentioned in the pathophysiology section of this article (46).



### **Tension-Type Headache in the Paediatric Population**

TTH in children is defined similarly to adult population - as a bilateral, pressing or tightening pain of mild to moderate intensity and is not aggravated by physical activity (26,47). However, the clinical presentation in this population often differs from textbook definitions. Headache episodes may be shorter in duration compared to adults, and symptom reporting tends to be more heterogeneous - mostly due to age-related and developmental factors (47,48). The phenotype may resemble migraine, as the pain is non-pulsating and less frequently accompanied by nausea, vomiting, or marked photophobia; nonetheless, a certain degree of sensitivity to light or sound may be present in some cases (47,49). For the majority of paediatric patients, tension-type headaches are commonly precipitated by situations associated with psychological stress and heightened anxiety (50). Studies have also indicated a slight female predominance among adolescents with TTH (51). This gender difference is less pronounced than in migraine and may be associated with hormonal influences that become more prominent during puberty (51). Unfortunately, current diagnostic criteria such as those outlined in the ICHD-3 are extrapolated from adult populations and may not fully capture the unique characteristics of TTH in children. Consequently, ongoing efforts are focused on refining these criteria and developing age-specific diagnostic tools that incorporate inputs from headache diaries, objective physical assessments, and input from caregivers to ensure accurate diagnosis and effective management (47).

### **Future directions in research and therapy**

Future research directions are rapidly evolving, driven understanding of multifactorial pathogenesis of TTH, which encompasses peripheral neuromuscular dysfunction and central sensitization processes and neuroplastic changes (46). To advance this understanding, there is a growing emphasis on developing refined experimental models, such as chronic animal models, that accurately reproduce the prolonged peripheral nociceptive input and central alterations observed in patients, thereby facilitating the discovery and evaluation of new therapeutic targets (52). Also advances in genetic research are beginning to shed light on predispositions linked to serotonergic dysregulation and other molecular alterations, thereby opening the door to personalized medicine approaches, that may improve therapy according to an individual's neurochemical and genetic profile (20,21,53). There is a growing interest in non-pharmacological methods as a complementary approach to the treatment of tension-type headache (53). Manual therapy, muscle endurance exercises, postural correction training, and interventions targeting myofascial trigger points are all being investigated for their potential to reduce abnormal peripheral nociceptive input, and consequently, to decrease the frequency of TTH episodes (19,53,54). Psychological interventions and biofeedback have also been reported to contribute to the modulation of pain pathways and to enhance overall quality of life in individuals with TTH (54). Moreover, there are promising reports regarding the use of botulinum toxin type A and vitamin D; however, their efficacy has not yet been conclusively established (46). Ultimately, the integration of interdisciplinary approaches, including basic science research, high-quality clinical trials and innovative treatment techniques, has the potential to transform the management of TTH into a precise model of care, that comprehensively addresses the biological, social, and psychological dimension of this disorder.

## **Summary**

Despite its high prevalence, TTH is not widely recognised as a serious medical concern. Nevertheless, a deeper understanding of this disorder is essential, as it may lead to improved treatment outcomes and more comprehensive, multidisciplinary approach - especially in patients with CTTH. While future prospects are promising, further research is still necessary to fully understand this complex and often overlooked disorder.

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Not applicable.

### **2. Data were obtained from**

PubMed, Google Scholar

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### **7. The authors declare no conflicts of interest.**

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