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Tension-Type Headache: A Life-Course Review

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Abstract

Background: Tension-type headache is the most prevalent primary headache type worldwide and is associated with a wide spectrum of disability. Although progress has been made in understanding the complex mechanisms that lead to the pathogenesis of tension-type headache, to date there are no clear-cut markers of what makes tension-type headache unique. Due to a relative lack of research (compared to migraine), the pathophysiology of tension-type headache is not well understood and there are gaps in the epidemiological data, particularly from Australasia.

Objective: To provide a structured narrative review of the prevalence and correlates of tension-type headache, with focus on a birth cohort of young adults from the Dunedin Multidisciplinary Health and Development Study (DMHDS) in New Zealand.

Method: A review of the literature was conducted to identify the epidemiological, diagnostic, methodological and pathophysiological factors that contribute to tension-type headache being a specific entity.

Results: Findings suggest that prevalence rates of TTH vary across global region, age, gender and method of assessment. A wide range of risk factors for TTH was identified, and recent advances in genetic and neurobiological research have increased understanding of the etiology of TTH. Few longitudinal studies have been conducted on TTH.

Conclusion: Further longitudinal epidemiological research is needed to help distinguish tension-type headache from migraine, particularly in young people. Identifying the specific markers of tension-type headache is a first step towards developing effective prevention and treatment strategies.

Key Words: Headache; Epidemiology; Longitudinal; Dunedin Study; Tension-type; Migraine

Conflict of Interest: There are no conflicts of interest associated with this study.

Abbreviations: CTTH: Chronic Tension Type Headache; DMHDS: Dunedin Multidisciplinary Health and Development Study; ETTH: Episodic Tension-Type Headache; ICHD: International Classification Of Headache Disorders; IHS: International Headache Society; NZ: New Zealand; TTH: Tension-Type Headache

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Introduction

Tension-type headache (TTH) is the most prevalent form of headache in all age groups across the globe [1-3]. TTH leads to considerable disability [4, 5] with up to 60% of individuals reporting decreased work effectiveness [6], increased absenteeism [7], and reduced social engagement [6]. The limited research on TTH, compared to that for other types of headaches [8], means that few recent advances have been made in the treatment of TTH [9]. Despite the availability of diagnostic criteria for TTH [10, 11], there is difficulty in distinguishing tension-type headache from

migraine due to overlapping features [12]. Evidence suggests that features common to both types of headaches include the presence of throbbing, unilateral pain [13, 14], muscle tension [15, 16] and aggravation by physical activity [17]. Further evidence suggests that stress, fatigue, alcohol and menstruation are common triggers for TTH and migraine [18]. TTH is the most costly type of headache for society, due to its greater prevalence [19]. As such, identifying the precursors and distinctive characteristics of TTH is a research priority.

The aim of this review is to identify factors that characterize TTH as a specific entity and to address a gap in epidemiological data from Australasia. Here we provide an overview of current research on TTH and highlight our findings with headache sufferers from the Dunedin Multidisciplinary Health and Development Study (DMHDS). The DMHDS is a prospective and representative cohort study that has collected information face-to-face about headache symptoms longitudinally from age 7 to age 38 years [20]. Given the lack of specific markers of TTH and the common transitions between TTH, migraine, and other headache subtypes, longitudinal prospective studies are required to address gaps in knowledge about the risk factors, specificity, incidence, and course of TTH in adults [5, 21, 22]. Our ongoing research is one of a limited number of longitudinal studies that can address these gaps in knowledge.

Headache classification

The classification of TTH is based on its phenotype as defined by the International Classification of Headache Disorders (ICHD). In extant research, TTH is classified according to the ICHD-II. The ICHD-II describes four categories for TTH: infrequent episodic; frequent episodic; chronic; and probable. The criteria further classify TTH based on the presence or absence of a coexisting disorder of the peri-cranial muscles.

Tension-type headache is typically characterized by bilateral pressing or tightening pain that occurs in the frontal or occipital areas, is of mild to moderate intensity, and is not exacerbated by physical activity [23]. There is no nausea or vomiting, but photophobia or phonophobia may be present. Probable TTH is defined as a headache fulfilling all but one of the diagnostic criteria. TTH is primarily diagnosed by the absence of features that characterize other primary headaches [21] and by the exclusion of other organic disorders [24]. On the basis that diagnosis of TTH is made by the absence of associated symptoms diagnostic of migraine, Monteith and Sprenger (2010) suggest that the sensitivity and specificity of classification for TTH could be contended [21]. An initiative to stimulate further research into the mechanisms and treatment of TTH prompted the release of a third edition *beta* version of the International Classification of Headache Disorders (ICHD-3 beta) in 2013 [25]. This version is purported to have made improvements over the ICHD-II, and the International Headache Society (IHS) contend that it would "be unhelpful to continue to use the previous ICHD-II for scientific work" (p.633; IHS 2013).

Current diagnostic ambiguity contributes to the significant number of people who are misdiagnosed or who are assigned a diagnosis of probable TTH, or who remain unclassifiable [26]. Findings show that, after examination by a physician, 35.6% of patients remained unclassified [27], one-third of TTH patients were misdiagnosed with sinusitis, followed by other misdiagnoses such as hypertensive or cervicogenic headache, [28] and as few as 1.3% of patients obtained a correct diagnosis of TTH [28]. The lack of specific markers of tension-type headache presents a substantial challenge in differential diagnosis of IHS-defined TTH and migraine due to the overlap of symptoms and precipitants of each headache type [12, 29, 30]. Many patients with chronic tension type headache (CTTH) over-use medication [24, 31], making it difficult to distinguish CTTH from medication overuse headache [32]. Further, TTH and migraine have been shown to frequently coexist [33, 34]. For instance, findings from our longitudinal study show that around four percent of adults experience coexisting TTH and migraine [30].

The coexistence of TTH and migraine, together with the difficulty in clinical settings to differentiate TTH from migraine, have led some researchers to argue that TTH and migraine are not distinct clinical entities but represent opposite ends of a pain severity continuum (i.e., continuum-severity theory) [35, 36]. However, the vast majority of people with TTH never seek medical attention [23, 31]. Hence the apparent lack of distinction between TTH and migraine in clinical settings may reflect referral bias [37]. Population-based evidence of significant differences in epidemiological characteristics suggests that migraine and tension-type headaches are distinct entities [36, 38]. We found, for example, that migraine was related to familial factors, anxiety, and stress reactivity, whereas TTH was associated with neck or back injury in early childhood [30]. Other studies have found that the prevalence of migraine is higher in women than men yet for tension-type headaches women are only slightly more affected than men [37, 38]. The risk factors for TTH appear to be different from those for migraine. Poor self-rated health, little sleep and an inability to relax after work are predictive of TTH whereas high work load, familial disposition, lack of secondary education and frequent TTH are risk factors for migraine [39]. Whether TTH and migraine represent distinct or similar entities continues to be debated. Nonetheless, the aforementioned research suggests that distinguishing TTH from other types of headaches purely on the basis of symptoms is difficult.

Epidemiology

According to the epidemiological literature on TTH, there are wide variations in the estimated prevalence of TTH. Although discrepancies are likely due to many factors, to a reasonable extent methodological differences can account for variability in prevalence estimates [24]. There is evidence that population estimates of TTH prevalence differ for questionnaire- versus interview-based assessments [22, 40]. There are inconsistencies between individuals' self-report of factors that trigger, aggravate, and ameliorate their headache and interview-based or objective measures. This work suggests that although questionnaires provide useful information about individuals' perceptions about their headache, they are subject to recall bias, and thus may not adequately identify actual relationships between TTH and other factors [41]. Further, the majority of epidemiological studies on TTH are cross-sectional and do not provide information on what

happens to prevalence rates over time, or the order of onset with respect to comorbid disorders, or how chronicity may develop [5, 22].

For a majority of individuals, first onset of TTH is before age 20 [7, 23], with peak prevalence between the ages of 30 and 39 years [31]. Data from our study show that 34.5% of New Zealand (NZ) children between 7-13 years of age experience frequent headaches. Only 9% of these children grow up to have TTH in adulthood. 11.1% of 26-year old study members were classified as having TTH and this rate increased to 17.5% at age 32 years [42, 43]. Of those with TTH at age 32, 10.3% had infrequent episodic tension-type headache (ETTH), 87.2% had frequent ETTH, and 2.6% had chronic TTH [43]. The lifetime prevalence of TTH in the general population ranges between 30% and 78%[24] and there is a slight decrease with advancing age [1].

Prevalence of TTH varies by age, gender, geographical region [1, 40] as well as according to education level and employment status [43]. The prevalence of TTH in our NZ females is twice that of males, consistent with the female-to-male ratio reported in various studies (ranging from 1.16:1 to 3:l [1]). There is also evidence that the prevalence of TTH increases with higher levels of education [3]. Individuals with TTH in our study were more likely to have obtained a bachelors or graduate-level degree compared to those without headache [44]. For chronic tension-type headache (CTTH), however, there may be an inverse relation between headache and education level [45]. Data from studies examining the relation between TTH and employment status are also equivocal. TTH has been reported to be associated with unemployment [46], whereas 32-year-old members of the DMHDS with TTH were significantly more likely to be employed than headache-free individuals. The roles of education and employment in the development of episodic and chronic tension-type headache clearly need further investigation.

Prevalence rates of TTH vary widely across global regions [40]. The global one-year prevalence of TTH has recently been estimated at 32 % (30% for episodic TTH, 2.4 % for chronic TTH) [40]. Episodic TTH rates range from 10.8% [47] to 37.3% [40] and chronic TTH rates range from 0.6 % to 3.3% [40]. The cross-study weighted aggregate rate of TTH is 13% [22], substantially higher than that for migraine with aura (4.4%) and chronic migraine (0.5%) [22]. The average one-year prevalence rate is greater in European countries (53%) followed by South America (31.5%), North America (30%), Asia (18.5%), the Middle East (10.3%), and Africa (7%) [40]. There is a gap in epidemiologic research on TTH from Australasia [40]. In our study, the one-year prevalence of TTH was 11.6% at age 38 years. Most studies [22], including ours, report a greater prevalence of TTH than migraine. Presented in **Table 1** is selected epidemiology of tension-type headaches in adults **(Table 1).**

TTH as a dynamic condition

Research suggests that the one-year prevalence rate of TTH changes during adulthood and that TTH is a dynamic condition [48, 49]. For instance, rates of TTH increased from 79% to 87% over a 12-year period among young adults in Denmark [7]. Our prevalence rates have similarly fluctuated in adult participants as they have aged: from 11.1% at age 26; 16% at age 32; and 11.6% at age 38. In young adulthood, individuals are in the process of establishing their careers, social networks, and health practices. It is possible that for some young adults, coping with these life transitions impacts physical and psychological processes that increase their susceptibility to TTH. This idea is supported by findings showing that compared to healthy controls, individuals with TTH display higher levels of perceived stress and deficient problem-solving skills [46, 50]. Although the observed variation between our prevalence rates and those of the Danish epidemiologic study by Lyngberg and colleagues (2005) [7] may to some extent reflect methodological differences, recent work suggests that prevalence rates of TTH are influenced by culture and degree of urbanization [40]. Future research could explore this notion.

Lyngberg and colleagues further reported that, of individuals diagnosed with episodic or chronic TTH, 45% went into remission, 39% had an unchanged frequency of episodic TTH, and 16% had either unchanged or newly diagnosed chronic TTH [7]. Similarly, a clinical 10-year follow-up study showed that 75% of individuals initially diagnosed with episodic tension-type headache continued to experience episodic TTH, and 25% of the study population progressed to chronic TTH. In those diagnosed with initial chronic tension-type headache, 31% remained chronic, 21% had developed medication overuse, and the remaining 48% had reverted to the episodic form [51]. In our study, 9.2% of study members experienced remission from age 26 to 38 years, 6% reported TTH at two of the three age group periods, and 2.7% had a late onset of TTH at age 38.

At the same time our findings suggest that, for many individuals, TTH is a persistent problem throughout young adulthood. Up to 40% of cohort participants diagnosed with TTH at age 26 were also diagnosed with TTH at age 32 and 43.5% of study members diagnosed with TTH at age 32 were also diagnosed with TTH at age 38 [43].

Precipitants and correlates of TTH

Researchers have identified multiple precipitants and correlates of TTH [48]. The most frequently reported precipitants of TTH are stress or mental tension [5], followed by sleep problems or fatigue [52]. There is also evidence that factors such as weather changes [18], menstruation [53], and inability to relax after work [5] are all associated with TTH.

Although many studies have found that TTH is related to stress, few studies have investigated subcomponents of stress or the timing with which the stress occurred. In our study, we found that overall levels of stress did not differ between adults with TTH and those who were headache-free. TTH sufferers did, however, report a high level of stress about bodily changes [54]. It is possible that the stress of a changing body during adolescence increases an individual's vulnerability to developing TTH. Individuals with TTH may also have different stress adaptive mechanisms involving both cardiovascular activation and the pain control system [55]. In response to ongoing cognitive stress, individuals with TTH maintained increased heart rate and blood pressure, whereas these decreased for migraineurs and controls.

Table 1 Selected epidemiology of tension-type headaches in adults.

Reference	Characteristic	Tension-type headache		
		ETTH	**CTTH**	**TTH**
	(Unspecified)			
	Prevalence:			
Sahler K (2012) [40]	Global	30.00%	2.40%	
	Europe	53.00%	2.30%	
	South America	31.50%	3.60%	
	North America	30.00%	2.20%	
	Asia	18.50%	2.40%	
Waldie et al., (2007) [43]	New Zealand	11.60%		
Sahler K (2012) [40]	Middle East	10.30%	3.50%	
	Africa	7.00%	1.10%	
	Female-to-male ratio			
Lynberg AC. et al., (2005) [7]				3:01
Rasmussen BK.et al., (1993) [18]				1:03
Schwartz BS. et al., (1997) [6]		1.16:1	2:01	
Waldie KE et al., (2002) [30]				2:01
	Education Level (high-university level/low- school level)			
Queiroz LP et al., (2009) [45]			Low	
Robbins MS et al., (2010) [8], Schwartz BS et al., (1997) [6]	High	Low		
Waldie KE et al., (2007) [43]		High		
Yu S et al., (2012) [47]				Low
	Employment (employed/ unemployed)			
Yu S et al., (2012) [47]	Unemployed			
Waldie KE et al., (2002) [30]		Employed		
	Age of onset			
Lyngberg AC et al., (2005) [7]	<20 yrs			
Mueller L. et al., (2002) [23]				
	Persistent TTH			
Lyngberg AC et al., (2005) [7]	39.00%			
Moerk H, et al., (2000) [5]	75.00%			
Waldie KE et al., (2007) [43]	40-43.5%			
	Change in TTH			
Lyngberg AC et al., (2005) [7]	25years	79.00%		
	37 years	87.00%		
Waldie KE et al., (2008) [42]	7-13 years	34.50%		
	26 years	11.10%		
	32 years	17.50%		
	Remission of TTH			
Lyngberg AC et al., (2005) [7]	12 year period	45.00%		
Waldie KE et al., (2007) [43]	6 year period	9.00%		
	Coexist migraine			
Waldie KE et al., (2002) [30]	4%			
Rasmussen BK et al., (1992) [33]	83%			
Lyngberg AC et al., (2005) [38]	94%			

Note: ETTH episodic tension-type headache, CTTH chronic tension type-headache.

Moreover, stress-induced pain responses were abnormally large while pain recovery was delayed for individuals with TTH compared to controls. These findings complement other findings that pain sensitivity mediates the relationship between stress and headache intensity in individuals with CTTH [56].

Our findings also add to evidence that TTH is frequently co-morbid with anxiety and depression [57] and with reports of repressed anger and resentment [57-59]. We also found that adults with TTH were less antisocial in childhood and less aggressive during adolescence and early adulthood than individuals

Table 2 Selected factors associated with tension-type headaches in adults.

Reference	Tension-type headache
	TTH (Unspecified)
Lyngberg AC et al., (2005) [7]	Poor health, lack of sleep, inability to relax
Rasmussen BK.et al., (1993) [18], Jensen R et al., (2010) [32], Scher AI. et al., (1998) [46]	Stress, mental tension, fatigue, psychological problems
Couch JR. et al., (2007) [68], Côtéa P et al., (2000) [70], Waldie KE et al., (2002) [30]	Neck, back or head injury
Russell MB et al., (2006) [64], Ostergaard S et al., (1997) [65]	Genetic predisposition
Eskin, M. et al., (2013) [94]	Problem solving, stress, depression
Baskin SM et al., (2006) [57]	Anxiety, depression,
Adler CS et al., (1987) [58]	Repressed anger, resentment
Waldie KE et al., (2002) [60]	Lower aggression, less antisocial
Waldie KE. (2001) [54]	Stress about bodily changes
Aaseth K et al., (2010) [73], Bell BD et al., (1999) [81]	Changes in brain structure

without headache [60]. This complements recent evidence that individuals with TTH have difficulty with outward expression of anger; inward anger is thought to increase the intensity level of headache through somatization [61]. Our findings using the SF-36 Health Survey [62] also showed that adults with TTH report significantly less vitality, poorer social functioning, and poorer physical and emotional health in everyday roles than individuals without headache [30]. Presented in **Table 2** are selected factors associated with tension-type headaches in adults.

While the biological mechanisms that predict progression from episodic TTH to chronic TTH have yet to be clarified, the epidemiologic literature has identified both depression and anxiety, and medication overuse, as primary factors that increase the risk of headache progression [1]. Taken together, the research discussed above suggests that more precise classification of TTH may require a more refined analysis of known precipitants and determinants.

Genetics and neurobiology

Genetic factors are most certainly involved in TTH pathogenesis [63], with a higher genetic influence for frequent ETTH than infrequent ETTH [64]. The population relative risk in relatives of individuals with chronic TTH was increased threefold compared with normal controls [65].

The exact neurological mechanisms of TTH are unknown. Although TTH was previously considered to be primarily psychogenic, recent evidence strongly indicates a neurobiological basis [66]. A distillate of recent research suggests that peripheral pain mechanisms are of importance in episodic TTH, whereas chronic TTH is related to sensitization of pain pathways in the central nervous system resulting from prolonged nociceptive stimuli from pericranial myofascial tissues [25, 67].

Several studies demonstrate a relationship between neck or head injury and headache [68-70]. Findings from this work suggest that the relationship between injury and headache is not strongly related to the temporal proximity of the injury. Instead, individuals with lifetime injuries to the head or neck had increased risk of headaches, even if the injuries were remote to the onset of headache. In our research, nearly one quarter of young adults with TTH suffered from a childhood neck or back injury [30]. It is plausible that a neck or back injury can lead to an increased vulnerability in the skeletal and pericranial muscles that increases susceptibility to TTH. This idea is supported by evidence that disturbances of the muscles of the upper cervical spine and neck–shoulder region play a role in the pathogenesis of tension-type headache [71, 72]. Research suggests that myogenic referred pain caused by active myofascial trigger points in the head, neck, and shoulder muscles might contribute to patterns of head pain in individuals with TTH [24].

Recent findings from epidemiological research examining the association of chronic headache and chronic rhinosinusitis in middle aged adults revealed that CTTH is significantly associated with chronic rhinosinusitis [73]. Indeed, individuals with chronic rhinosinusitis had a ninefold increased risk of having chronic headache compared with the general population.

Some researchers have proposed that sympathetic hyperactivity is the mechanism by which psychological factors such as stress triggers the development of TTH. There is evidence that muscle nociceptors can be stimulated by endogenous neurotransmitters and/or hormones such as serotonin, norepinephrine, and bradykinin [74]. This raises the possibility that activation of the sympathetic nervous system and hypothalamic–pituitary–adrenal axis in response to stress may be important in TTH pathogenesis. Sympathetic hyperactivity has been implicated in other pain disorders such as fibromyalgia [61], and a number of studies have demonstrated differences in short-term nervous system response between TTH patients and controls in response to stress [75-77].

If prolonged hyperactivity of the sympathetic nervous system in response to psychological stressors contributes to the abnormal pain processing observed in TTH, this may be accompanied by long-term changes in cardiovascular function and haemodynamics. Findings from other work suggest that nitric oxide-related central sensitization may be an important mediator in pain mechanisms of TTH [78, 79].

There is evidence that chronic TTH is associated with changes in brain structure. Research using magnetic resonance imaging (MRI) voxel-based morphometry showed that patients with CTTH have significant gray matter decreases in areas of the brain involved in nociceptive transmission [73]. The gray matter reduction correlated with lifetime duration of headache. There is

also evidence of white matter abnormalities in elderly patients with TTH (34% of the sample compared with 7% of headache-free controls) [80, 81]. Further, recent research shows that CTTH is associated with impaired motor learning [82]. CTTH patients had a deficit in use-dependent neuroplasticity within networks responsible for task performance [82].

In sum, advances in genetic and neurobiological research have increased our understanding of the complex mechanisms that may lead to TTH. As the preceding discussion illustrates, there is strong evidence for a genetic predisposition for frequent ETTH and chronic TTH. Moreover, sensitization of pain pathways in the central nervous system, and disturbances to the cervical spine or musculature from prior injury, are positively associated with TTH. Research has progressed our current understanding regarding the means through which psychological factors lead to TTH, suggesting sympathetic hyperactivity as a possible mechanism. Further, some studies indicate that CTTH is associated with structural and functional changes in the brain. By utilizing these findings, future interventions may provide better treatment outcomes for people with TTH.

Treatment

There have been few recent advances in treatment options for TTH. The treatment of tension-type headaches can be divided into pharmacologic and non-pharmacologic therapies [32, 83]. For a majority of individuals with TTH, the mainstays in pharmacotherapy are simple analgesics and nonsteroidal inflammatory drugs (NSAIDs) [32]. In our cohort, over half of adults with TTH were taking at least one form of prescription medication during the past 12 months compared to 35% of controls. In addition, 45% were taking analgesic medications during the past 12 months (most were simple analgesics) compared to 25.6% of controls. Although simple analgesics and NSAIDs have proved effective in the treatment of acute TTH, their efficacy decreases with increasing frequency and chronicity of TTH [83].

In individuals with very frequent episodic TTH and those with chronic TTH prophylactic, pharmacotherapy in the form of antidepressants is widely used [32]. The tricyclic antidepressant amitriptyline is the recommended drug of first choice as it has been shown to have a significant and clinically relevant effect in the prevention of CTTH [84, 85]. Other evidence suggests that the noradrenergic and specific serotonergic antidepressant mirtazapine [86], and the serotonin and noradrenaline reuptake inhibitor venlafaxine [87] may have comparable efficacy in the treatment of CTTH to that of amitriptyline and may be better tolerated. The effectiveness of other pharmacotherapies tested for TTH has been variable. For instance, findings on the effectiveness of muscle relaxants appear equivocal [88, 89]. Research suggests that botulinum toxin is ineffective or potentially harmful, thus is not recommended as treatment for CTTH [90]. Overall, research suggests that current pharmacotherapy options are far from optimal. Future pharmacotherapies that appear promising in the treatment of CTTH include third-generation antidepressants, nitric oxide inhibitors, Na^{+} and/or $Ca2^{+}$, channel modulators, and anticonvulsants [32].

Non-pharmacologic management of TTH encompasses a wide variety of physical and psychological therapies. There is evidence for the utility of therapies such as electromyography biofeedback alone or in combination with relaxation [91] and physiotherapy [92], and increasing evidence that cognitive-behavioral therapy may be effective in TTH [93]. However, for many therapies (e.g., acupuncture, emotional freedom therapy) there is currently no robust scientific evidence for efficacy [9]. There is some evidence that the combination of non-pharmacologic and pharmacologic therapies may be effective in the treatment of TTH [17]. Given that the underlying pathophysiology of TTH is complex, future studies focusing on the interaction of multi-modal treatments are warranted [94].

Summary and Conclusions

TTH is the most prevalent primary headache type worldwide and is associated with significant disability. The prevalence of TTH differs by geographical region, with the prevalence of TTH in NZ comparable to that of Asia and the Middle East. Further, the prevalence of TTH in young NZ adults is dynamic, with peak prevalence at age 32 years. For a large proportion of sufferers, however, TTH is a persistent problem.

Although current diagnosis is primarily clinical and based on negative associations and by exclusion, a majority of individuals with TTH do not seek medical attention. Thus, identifying the factors that characterize TTH as a specific entity has proven difficult. Findings from our study suggest that TTH and migraine are associated with different developmental characteristics. NZ adults that have suffered a neck or back injury, and those who are educated and employed are more likely to have TTH. Stress about bodily changes in adolescence is also a significant precipitant of TTH. Neurobiological studies suggest that TTH is associated with sympathetic hyperactivity and structural changes in the brain.

Further research is needed to reliably determine what is so special about tension-type headache. Research using the updated beta version of the ICHD may shed new light on clinical features that better characterize TTH. Moreover, a classification system developed from studies that further examine the epidemiology, neurobiology, and genetics of TTH is required.

This review also highlights the need for further longitudinal research. Prospective studies that elucidate the multiple correlates of TTH across the lifespan can provide clues regarding etiologic mechanisms and factors that characterize TTH as a specific entity.

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