Influence of Psychosocial Factors in the Risk of Developing Temporomandibular Disorders in Dental Students

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THESIS

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Abstract

Introduction: Temporomandibular disorders are a heterogenous group of disorders with a multifactorial disease process. It has been suggested that there is a relationship between sex, sense of coherence (SOC), stress manifestations and TMD however, this topic is controversial and poorly understood. This study aims to analyse this proposed relationship so dentists can better identify at risk populations and implement a biopsychosocial model of care to manage them more appropriately. Dental students are a group notoriously susceptible to intense stress manifestations and heightened prevalence of TMD. This research aims to study this claim with an improved design than past studies.

Materials and Methods: A total of 48 dental students attending Universidad Católica de Valencia (UCV) participated in this study. Students were given a series of questionnaires to evaluate intensity of stress manifestations, sense of coherence and the DC/TMD symptoms questionnaire. Following this, a clinical examination of each student's TMD was performed (using the DC/TMD clinical examination).

Results: More than 61.70% of students were diagnosed with at least one TMD. Students presenting with TMD-pain disorders showed heightened levels of some stress manifestations. There was no relationship found between TMD-joint disorders and any stress manifestations. Females were 1.8 times more likely to be diagnosed with a TMD-joint disorder than males. There was no sex predilection for TMD-pain disorders.

Conclusions: Dental students are an at risk group of developing a TMD. TMD-pain disorders have a negative effect on stress manifestations and SOC helps to buffer this negative effect.

KEYWORDS:

Male, Female, Stress manifestations, Sense of coherence, Dental students, Temporomandibular disorders

Resumen (en Español)

Introducción: Los trastornos temporomandibulares (TTM) son un grupo heterogéneo de trastornos con un proceso de enfermedad multifactorial. Se ha sugerido que existe una relación entre el sexo, el sentido de coherencia de Antonovsky (SOC), las manifestaciones de estrés y los TTM, pero este tema es controvertido y poco conocido. Este estudio tiene como objetivo analizar esta relación hipotética para que los dentistas puedan identificar mejor a las poblaciones de riesgo e implementar un modelo biopsicosocial de atención para manejarlas adecuadamente. Los estudiantes de odontología son un grupo notoriamente susceptible de presentar manifestaciones de estrés intenso y una mayor prevalencia de TTM. Esta investigación pretende estudiar esta hipótesis con un diseño mejorado respecto a estudios anteriores.

Materiales y métodos: Un total de 48 estudiantes de odontología de la Universidad Católica de Valencia (UCV) participaron en este estudio. Los estudiantes recibieron una serie de cuestionarios para evaluar la intensidad de las manifestaciones de estrés, SOC y el cuestionario de síntomas de DC/TMD. A continuación, se realizó un examen clínico de los TTM de cada estudiante (mediante el examen clínico DC/TMD).

Resultados: Más del 61,702% de los estudiantes fueron diagnosticados con al menos un TTM. Los estudiantes que presentaban trastornos de dolor por TTM mostraban niveles elevados de algunas manifestaciones de estrés. No se encontró ninguna relación entre los trastornos TTM-articulares y cualquier manifestación de estrés. Las mujeres tenían 1,8 veces más probabilidades de ser diagnosticadas de un TTM-articular que los hombres. No hubo predilección por el sexo para el dolor asociado a TTM.

Conclusiones: Los estudiantes de odontología son un grupo de riesgo de desarrollar un TTM. Los TTM asociados con dolor tienen un efecto negativo en las manifestaciones de estrés y el SOC ayuda a amortiguar este efecto negativo.

LAS PALABRAS CLAVE: Varón, Mujer, Manifestaciones de estrés, Sentido de coherencia (SOC), Estudiantes de odontología, Trastornos temporomandibulares

Introduction

A joint is a point where two bones make contact. Located just anteriorly to the external auditory meatus, the temporomandibular joint (TMJ) is the area connecting the lower jawbone to the skull (1).

The TMJ consists of:

1. The bones of the TMJ
    a. Mandibular condyle
    b. Glenoid fossa of the temporal bone
2. Intra-articular disc
3. Joint Capsule and ligaments
4. Muscles of mastication
5. Vascular supply and innervation of the TMJ

The bones of the TMJ

The foundation of the TMJ is formed by the mandibular condyle articulating with the concave glenoid fossa of the temporal bone. The articular eminence is a protruding convex bone situated just anterior to the glenoid fossa, the convexity guides the movement of the condyle (2).

Intra-articular disc

The TMJ is a bilateral synovial ginglymoarthrodial joint; the only joint of its kind in the human body, it allows the jaw to perform two basic types of movement: a hinging movement (ginglymus joint) and a sliding movement (arthrodial joint) (3). This type of joint movement is unique to the TMJ and is made possible by the intra-articular disc: a dense fibrous connective tissue separating the mandibular condyle and the glenoid fossa, creating two separate compartments in the joint space. The inferior

joint space is delimited inferiorly by the mandibular condylar head, this compartment predominantly produces a rotational movement. The superior joint space is delimited superiorly by the glenoid fossa, and predominantly produces a sliding movement (2,3).

Joint Capsule and Ligaments

The joint capsule encloses the articular disc, allowing for complex movement to occur. Synovial membrane lines the inner surface of the joint, secreting synovial fluid and has a triad of functions: lubrication of the articulation surfaces, removal of degradation products and nutrition of vascular parts of the joint. The capsule is thin anteriorly and posteriorly, but, laterally the thick temporomandibular ligament offers stability. Made of fibrous tissue, the ligament is lax, except in protrusive and intrusive movements where it limits movement in the anteroposterior direction. There are two more accessory ligaments: stylomandibular and sphenomandibular ligaments, their functions are uncertain but it is widely suggested they limit mandibular movement in the anteroposterior and lateral directions respectively (2).

Muscles of mastication

Musculature located in the head, face and cervical spine contributes to movement and stability of the TMJ (1,2). The primary muscles of mastication are the masseter, temporalis, lateral pterygoid and medial pterygoid (see table 1 and 2 and figure 1). Other muscles of mastication include, suprahyoid, infrahyoid, digastric and geniohyoid. Muscles should be clinically examined at regular intervals to assess the effect of parafunction on the muscles.

Table 1. Description, origin and insertion of the muscles of mastication

Muscle	Description	Origin	Insertion
Masseter	Quadrilateral, thick muscle with two portions (superficial and deep)	Anterior two thirds of zygomatic arch	Lateral Surface of angle of mandible
Temporalis Muscle	Large, broad, fan-shaped muscle	Temporal fossa	Coronoid process and anterior border of the ascending ramus of mandible
The Lateral (external) Pterygoid	Muscle with two separate parts (inferior belly and superior pterygoids)	Superior pterygoid: infratemporal surface of the greater wing of the sphenoid bone Inferior Pterygoid: lateral surface of the lateral pterygoid plate	Superior pterygoid: anterior part of the capsule and intra-articular disc and a small attachment inserts into the fossa in the anterior part of the head of the condyle Inferior pterygoid: fossa in the anterior part of the head of the condyle
The Medial (internal) Pterygoid	Quadrilateral, thick muscle	Medial surface of the lateral pterygoid plate and the lateral aspect of the medial pterygoid plate	Angle of the mandible on the medial surface, opposite the insertion of the masseter

Table adapted from Temporomandibular disorders: a problem based approach (2).

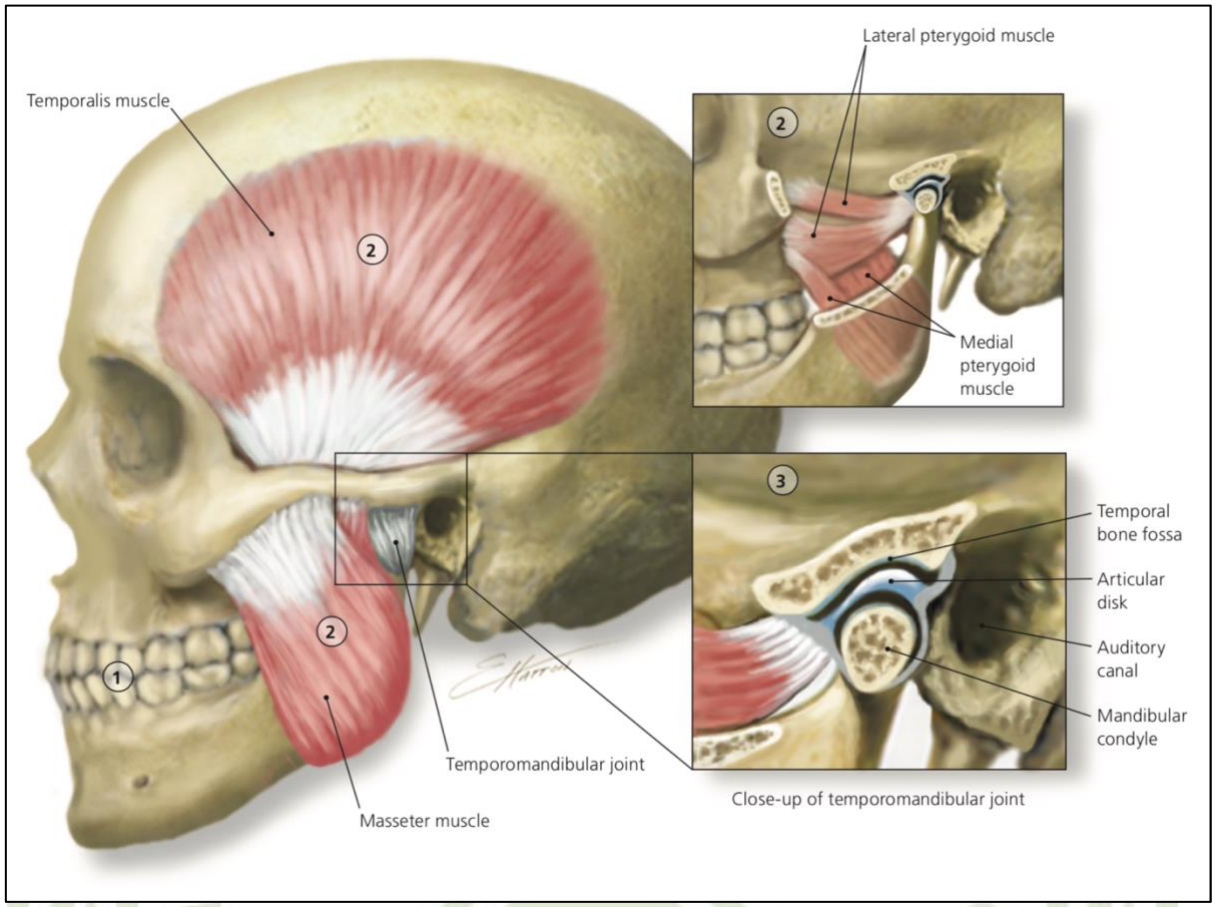

Figure 1. Anatomy of the temporomandibular joint and the structures responsible for the movement of the joint (Photo taken from Gauer et al.) (4).

Muscle	Function	Parafunction	Examination
Masseter	Superficial: primary elevator muscles of the mandible during jaw closure, assists in protrusion of the mandible Deep: main elevators of the mandible, assists in retrusion of mandible	Active during clenching of jaw Causes tenderness (particularly at the insertion)	The origin and insertion of the muscle is palpated both inside and outside the mouth (bimanually)

Temporalis Muscle	Anterior fibres: elevation of mandible Posterior fibres: retraction of mandible	In patients with bruxism, this muscle has the tendency of becoming symptomatic and painful (most often, the anterior fibres)	Can be palpated in the temple region (at its origin) when patient clenches and unclenches their teeth The insertion cannot be palpated
The Lateral (external) Pterygoid	Depression of the mandible Stabilisation of the condyle/ intra-articular disc/ fossa Unilateral contraction: lateral deviation of mandible Bilateral contraction: protrusion of mandible	Increased activity in both superior and inferior pterygoids causes referred pain in preauricular area (when examined with resistance)	Not accessible to digital palpation. Instead, response to resistance should be examined., The examiner's hand placed under the patient's chin and pressure is applied to close the mouth while the patient tries to resist. If there is tenderness in the lateral pterygoid this test will produce pain in the preauricular area.
The Medial (internal) Pterygoid	Elevation of the mandible Assists in protrusion and lateral deviations of the mandible	Muscle cannot be examined so the effect of parafunction is unknown	Not accessible for manual or digital palpation

Table 2. Function, parafunction and examination of the muscles of mastication

Table adapted from Temporomandibular Disorders: a problem based approach (2).

Vascular supply and innervation of TMJ

The TMJ is innervated by the mandibular branch of the trigeminal nerve, primarily by the auriculotemporal branch, that extends from the mandibular branch posteriorly to innervate the TMJ anterolaterally. Posteriorly, the TMJ is innervated by the masseteric branch and the deep temporal nerves. The blood supply to the TMJ is circumferential, via the internal maxillary and the superficial temporal branches of the external carotid artery. The articulating surfaces of the TMJ (the fibrocartilage lining the mandibular condyle and glenoid fossa, and the intra-articular disc) are avascular and not innervated. This trait facilitates physiological functioning of the jaw i.e. movement without bleeding and pain (2).

Articulatory System and Temporomandibular Disorders (TMDs)

The TMJs form an integral part of the articulatory system: a functional unit composed of the TMJs, intra-articular discs, mandibular/jaw muscles and occlusion. The structures of the articulatory system must work in synergy together to coordinate tandem dynamic function. Any stimuli effecting one component of, or one side of the system will have a knock-on-effect on the whole system. For this reason a thorough examination of the TMJ, masticatory muscles and occlusion should be performed as routine. Any deviation from physiological function of one or more of the components of the articulatory system could illicit a temporomandibular disorder (TMD) (2).

Symptomology

TMD constitute a group of clinical problems associated with pain and dysfunction, which involve the TMJ, masticatory musculature, surrounding bony and soft tissue components and combinations of these problems (4–6). Symptoms of TMD are varied, ranging from mild discomfort, to debilitating pain in the TMJ and its associated structures. Other symptoms include limitation or deviation of the

mandibular range of motion, TMJ sounds, earache, headache and facial pain (3,4). TMD is the most common cause of nondental pain in the orofacial region, as such, chronic pain is the overwhelming reason patients with TMD seek treatment (7). This pain can be intermittent or persistent and is often exasperated by provocation, either through physiological use (talking, chewing or yawning) or parafunctional habits (bruxism, tongue thrusting or fingernail/pen biting) (8,9).

Epidemiology

Due to the variability of diagnostic approaches, the epidemiology of TMD is highly discrepant. It is generally accepted that TMD affects more than 5% of the population and that management needs for people with TMD is estimated at 16% (8,10).

The introduction of the Diagnostic Criteria of Temporomandibular Disorders (DC/TMD) has provided researches with a standardised diagnostic tool for TMD. The DC/TMD was developed form the Research Diagnostic Criteria of Temporomandibular Disorders (RDC/TMD) that was published in 1992. After many years of refinement of the diagnostic algorithms, input from international members of TMD organisations and field trials, major steps have been taken from the old RDC/TMD to form the new DC/TMD, which, is now considered as the gold standard for the diagnosis of TMD. This tool has allowed researchers to understand better the epidemiology of TMD (8).

A recent meta-analysis using data collected from 21 observational studies in which TMD was diagnosed using the RDC/TMD or DC/TMD found that the overall prevalence of TMD was approximately 31% for adults and 11% for children/adolescents (11).

In 2006, the first and only prospective, longitudinal study of TMDs was launched. Termed the Orofacial Pain Prospective Evaluation and Risk Assessment (OPPERA study), it aimed to identify the risk factors of TMD. The study involved enrolling and monitoring over a 5-year period 4,000 men and women who were currently “TMD-

free" in 4 different sites around the United States. It found that TMD is a prevalent condition with 4% of the population developing TMD yearly. Its prevalence is highest in those aged 35-44 years and the least in those aged 18-24 years. Women are 4 times more likely than men to develop TMD (5).

Classification

In 2013, the International Research Diagnostic Criteria for Temporomandibular Dysfunction Consortium Network published an updated classification system for TMD (8). The consortium categorised TMDs into two main classifications: intra-articular disorders (within the joint) and extra-articular disorders (masticatory muscle disorders), these categories can be further subdivided, this is depicted in table 3 (4,7,8).

Table 3: Classification of Temporomandibular disorders (8).

Intra-articular disorders	**Extra-articular disorders (masticatory muscle disorders)**
Congenital and developmental disorders • Condylar hyperplasia • First and second branchial arch disorders • Idiopathic condylar resorption	Local myalgia
Degenerative joint disorders • Inflammatory: capsulitis, synovitis, polyarthritis (rheumatoid arthritis, psoriatic arthritis, ankylosing spondylitis, Reiter syndrome, gout) • Non-inflammatory: osteoarthritis	Myofascial pain disorder

Disc derangement disorders • Displacement with reduction • Displacement without reduction (closed lock) • Perforation	Myofibrotic contracture
Infection	Myositis
Neoplasia	Myoplasm
Temporomandibular hypermobility • Dislocation • Joint laxity • Subluxation	Neoplasia
Temporomandibular hypomobility • Ankylosis: true ankylosis (bony or fibrous) or pseudoankylosis • Post-radiation fibrosis • Trismus	
Trauma • Contusion • Fracture • Intracapsular haemorrhage	

Adapted from Schiffman E, Ohrbach R, Truelove E, et al. Diagnostic criteria for temporomandibular disorders (DC/TMD) for clinical and research applications: recommendations of the International RDC/TMD Consortium Network and Orofacial Pain Special Interest Group. J Oral Facial Pain Headache. 2014;28(1):6–27.

Most non-articular disorders present as myofascial pain focused to the muscles of mastication. In fact, myofascial pain accounts for more than 50% of TMDs (6). The most common intra-articular disorder of the TMD is articular disc displacement (7). Disc displacement results from changes in the disc-condyle relationship and can occur with reduction or without reduction. Disc displacement with reduction (DDwR) is characterised by progressive displacement of the articular disc against the mandibular condyle and articular eminence, this displacement can occur in any direction but, more often than not is an anterior displacement. When the mouth is closed, the articular disc is displaced in relation to the condyle and when the disc is open, the disc returns to the intermediate area between the condyle and the articular tubercle. This is usually accompanied by clicking, popping or crepitus sounds, pain and limitations of movement. If DDwR is not treated it could cause disc displacement without reduction this is when the articular disc displaces but does not reduce, causing TMJ pain and limited jaw opening (sometimes called a “close lock”) (6).

Aetiology

TMD is a heterogenous group of disorders (4). The etiopathogenesis of TMD is multifactorial and poorly understood. It is widely accepted that there are three contributing factors that can work in conjunction with each other to cause TMD: predisposing factors (increase the risk of developing TMD), initiating factors (cause the onset of the disorder) and, perpetuating factors (interfere with the healing process or enhance the progression of TMD). An aetiological factor can fall into one or all three groups (12,13).

Initiating factors cause the onset of the disorder and mainly include trauma or adverse loading of the masticatory system. Predisposing factors can be any pathophysiological, psychological or structural processes that alter the masticatory system, leading to increased susceptibility of developing a TMD. Perpetuating factors are those that interfere with the healing process or enhance the progression of TMD; behavioural factors (bruxism and abnormal head posture), social factors

(affecting perception and learned response to pain), emotional factors (anxiety and depression) and cognitive factors (negative thoughts and attitudes that can make resolution of TMD more difficult). The successful management of the disorder relies on the ability of the clinician and patient to cooperate in identifying and controlling these contributing factors (12,13). Current research aims to develop a mechanism and aetiology-based diagnostic protocol so that clinicians can provide personalised care of their patients. The new DC/TMD was a step in the right direction, but it is clear that more research needs to be done to address underlying TMD mechanisms and aetiologies (8).

There is a plethora of etiological factors that have been found to or suspected of contributing to TMD: occlusal factors, psychological factors, hormonal factors, microtrauma, parafunctions, joint hyperlaxity and joint hypermobility, hereditary factors, comorbid conditions, sleep and sleep breathing, to name a few (5,12,13). Some of these are out of the scope of this paper. For the purpose of this study we aim to focus on the relationship between TMD, psychosocial factors and sex.

Sex Predilection for TMD Prevalence and Severity

Sex and gender are important determinants of health and wellbeing (14). There are no universally accepted definitions of these evolving terms and they are often considered interrelated. However, it is widely agreed that the distinction lies in whether we are speaking from a social or biological stance. Sex is often equated with the biological (15). Sex is usually categorised as male or female and is defined by the differential organisation of chromosomes, reproductive organs and sex steroid levels (14,16). Within the realm of health sciences, sex can contribute to anatomical and physiological differences between the sexes. Gender can be used to refer to traits that society ascribes to male, female and gender-diverse people; within healthcare, it may reflect behaviours that influence exposure to health risks, access to healthcare or health-seeking behaviours (15).

There is a longstanding history of excluding women from clinical trials. This trend follows the historic idea of the male body being viewed as the default human body (17). Even in recent years this tradition has carried on with some researchers arguing that any biological sex differences are so negligible that they can be ignored in research (15). Even researchers who admit that there may be biological differences between the sexes advocate against the inclusion of women in research, often arguing that female bodies (both the human and animal variety) are too complex, too variable and too costly to be tested on. Many state that women's fluctuating hormones due to their menstrual cycle create results that are far too variable to be included in research (17). This trend was solidified in 1977, when the US Food and Drug Administration (FDA) recommended that women of child-bearing age be excluded from trials (16). This was their response to the thalidomide scandal (where doctors prescribed thalidomide to pregnant women for their morning sickness with the result of 10,000 children being born with thalidomide-related disabilities). These recommendations went unquestioned and resulted in inadequate representation of women in clinical trials for decades (17). This omission has restricted the generalizability of research findings and their relevance to clinical practice, in particular for women but also for men (14).

In recent years, the field of sex-based research has been undergoing a revolution, there is increased consideration, reporting and analyses of sex disaggregated data (14,16). Researchers are starting to appreciate that our anatomical dimorphism and varying quantities of sex hormones undeniably dictate our body's response to stressors, drugs and pathologies. Furthermore, they have uncovered that differences between the sexes goes deeper than this, to a cellular level. Cells of the human body can usually be classed as male or female (having either X and a Y chromosome or two X chromosomes) and researchers are starting to understand that despite the cells having the same structures, their sex still manipulates the way our bodies behave. Already, researchers have identified differences in the immune response, stress response, cancer proliferation and lung capacity (14–17). In light of this, more and more researchers are making an effort to include both sexes in

their research and to present sex-disaggregated data in their results. This work aims to avoid making erroneous generalisations and opens up the possibility of sex-specific potential treatment pathways (15).

Researchers of TMD started identifying a clear sexual disequilibrium in TMD prevalence between the sexes. In cross-sectional and case control studies, frequency of TMD is consistently greater in females than in males. There is a reported female-to-male ratio of 2:1 in the general population to more than 4:1 in clinical trials (18). The reason behind this sexual disequilibrium has been studied in depth, but is still unclear (6).

Women tend to develop TMD in their premenopausal years. For this reason, it has been proposed that sex hormones are a causative factor of TMD. Supporting this view, is the discovery that elevated levels of oestrogen has been found in patients with TMD (3). Furthermore, in two separate studies, it was found that pain in TMD increases approximately 20% in women taking oral contraceptives and 30% in postmenopausal patients receiving Hormone Replacement Therapy (HRT) (19). Despite the suggestion of a positive correlation, no definitive link between oestrogens and TMD causation has been established (6). One possible theory is that sex hormones enhances the expression of enzymes called matrix metalloproteinases (MMPs), which, causes cartilaginous breakdown of the TMJ's fibrocartilage and contributes to joint laxity, thus, causing TMD (3).

However, the role that oestrogen plays in TMD is controversial: a systematic literature review was conducted in 2015 to study the proposed correlation. It reviewed results of nine studies and concluded that the association between oestrogen level and TMD were weak, divergent and often contradictory (19). Researchers suggested that oestrogen may affect the regulatory effect of TMD pain

processing rather than the TMD structures: that it increases susceptibility to painful stimuli by modulating the limbic system (12,19).

Research into suspected differences in pain sensitivity between the sexes showed that females and males have comparable thresholds for cold and ischaemic pain, while pressure pain thresholds are lower in females than in males (20). The OPPERA study also found that chronic TMD cases have greater sensitivity to pressure pain. They concluded that pressure pain thresholds fluctuated in response to onset and remission of TMD but did not predict these changes and they hypothesised that this was because painful TMD enhanced central nervous system sensitization (increased responsiveness of nociceptors to normal or sub-threshold stimuli) (5). Researchers have suggested that this variance in pain sensitivity between the sexes could be contributing to the seemingly higher prevalence of TMD found in women (5,20).

The strong variance between female to male prevalence observed in studies of clinic versus population based samples (reported female-to-male ratio of 2:1 in the general population to more than 4:1 in clinical trials) could suggest that the disequilibrium has more to do with gender related behavioural factors rather than a heightened risk of TMD (5,18). Gender includes behaviours and activities that are determined by society and culture in humans and could contribute to the fact that women were found to seek specialised treatment for TMD three times more frequently than men (12,16). These discrepant findings could also be explained by the hypothesis that women are at a greater risk of developing a more severe form or a more persistent form of TMD than with a heightened risk of TMD onset. This view was backed up by the results of the OPPERA study, which found that women were only at a marginally higher risk of developing clinically verified TMD and that the rate of TMD symptoms was no greater in women than in men (5). These discrepancies could be explained by the concept of incidence-biased. Cross-sectional studies record status at only one point in time so they are more likely to

pick up on cases where TMD persists rather than in those that resolves quickly. The OPPERA study was a case-control study so whilst it didn't show a significantly greater risk of women developing TMD or having heightened symptoms, it did show that 54% of females versus 41% of males had persistent forms of TMD (5,18).

There are stark differences in the way in which the male and female sex respond to stressful situations and emotionally process challenges. Research shows that anxiety and depression are more readily diagnosed in women than in men: for example, women are twice as likely to be diagnosed with major depressive disorder (MDD) than in men. Whilst there is a clinical bias to diagnose depression in women more frequently than in men, researchers have still been able to pinpoint differences in sex-related influences on nervous system function. There is a suggestion that oestrogen fluctuations during women's childbearing ages may have a part to play as rates of MDD are the same for males and females in the prepubertal and postmenopausal ages. There is also a suggestion that gender-related psychosocial factors could be contributing to the differential prevalence as women were found to be more vulnerable to future depressive episodes in stressful situations after having experienced a negative life event. On top of this, researchers have reported that women now makeup 42% of the global workforce (an increase of 126% in the last 30years). Working women suffer from higher levels of stress to their male counterparts. This is often attributed to the social and cultural expectation that women must work whilst maintaining their traditional family roles and responsibilities. This has made women at a greater risk of developing stress-related diseases than ever before. Despite questions of the biopsychosocial reasoning behind it, research shows that women are more likely to suffer from anxiety, depression, somatisation and a lower sense of coherence than men (15,17). Interestingly, studies have found that higher levels of stress, depression, anxiety and somatisation are experienced in people experiencing TMD-related pain (9). In fact, the OPPERA study found that a high psychological distress is one of two principle phenotypes that contribute to onset and persistence of TMD (5). With this in mind, researchers are starting to hypothesise if sex-related biopsychosocial

factors causing heightened psychological distress that affects women more negatively than men are contributing to the disequilibrium in TMD prevalence between the sexes (20).

Psychosocial factors

Psychosocial factors are characteristics that influence an individual psychologically and/ or socially. Psychosocial factors encompasses the complex relationship between psychosocial resources and psychosocial risk factors. Social resources include social network and social support. Psychological resources involves coping ability, mastery, sense of coherence, and self-esteem. Psychological risk factors comprises vital exhaustion, depressiveness, hopelessness and hostility. The relationship between all the variables builds an important picture of an individual's psychosocial condition (21).

Over the years, researchers have used different techniques to try to quantify individual's psychosocial condition. In 1996, Terluin published his Four-Dimensional Symptom Questionnaire (4DSQ). It is a self-report questionnaire that was designed to assess common psychological symptoms: distress, depression, anxiety and somatisation. Characteristic distress symptoms include worry, irritability, tension, poor concentration, listlessness, sleeping problems and demoralisation. Distress is inherently linked with both anxiety and depression. When we remove distress symptoms from depression we are left with anhedonia and depressive thoughts. When we separate distress symptoms from anxiety we are left with symptoms of irrational fears, anticipation anxiety and avoidance behaviour. Somatisation is the tendency to experience medically unexplained somatic symptoms, to attribute them to physical illness, and to seek medical help for them. Terluin et al. discovered that the vast majority psychological symptoms could be described by just these four symptom dimensions (22). Similarly, in 1987, Antonovsky developed the 29 item Orientation to Life Questionnaire (SOC-29) to measure individual's sense of

coherence. Sense of coherence reflects a coping capacity of people to deal with everyday life stressors. The exam is on a 7-point likert scale and consists of three elements: comprehensibility (the ability to define life events as less stressful), manageability (to mobilise resources to deal with encountered stressors) and meaningfulness (experience motivation, desire and commitment to cope). Antonovsky also developed a shorter version consisting of 13 questions (SOC-13) where the score ranges from 13-91 (23).

For years, the relationship between psychosocial stressors on health has been a subject of interest and debate (24–26). Nowadays, it is widely accepted that the body and mind are inherently linked (26). What makes the relationship between psychosocial factors and ill health even more complex is the concept of clustering of multiple health risk behaviours. This is the idea that people with more negative psychosocial factors are more likely to engage in multiple health risk behaviours (e.g. smoking, reduced physical activity and high alcohol intake). Separately, these behaviours contribute to a decreased overall health, however, engaging in multiple risk behaviours constitute greater risk of disease development than the summated risk for each of the behaviours. The literature proves that these risk factors are more often than not found in the same individual, having a more profound impact on overall health (21).

Negative Psychosocial Factors and TMD

Over the years, researchers have been challenging the historic mechanical view of the development of TMD and suggesting that there is an important link to consider between TMD and psychosocial factors. This viewpoint had been extensively supported in the literature for some time, however, it was widely debated whether psychosocial factors were etiologic to the onset and progression of TMD, or, whether psychosocial distress was a consequence of TMD (27). It was also argued

that the negative correlation between psychosocial factors and TMD could be explained indirectly by the fact that people with anxiety and depression tend to participate in parafunctional habits (e.g. grinding and clenching) more readily than the TMD free population (6,27). Even scientists who agreed that psychosocial factors was an aetiology of TMD, argued over whether this factor fell under a predisposing, initiating or perpetuating factor (12,27,28). Due to the lack of longitudinal incidence studies on the association of the two, our understanding of this relationship remained limited, until the OPPERA study (5).

The OPPERA study found that psychosocial factors (e.g., somatic awareness, distress, catastrophizing, pain amplification, and psychosocial stress) had a significantly higher prevalence in subjects with TMD compared to healthy individuals (5). The OPPERA study aimed to uncover etiological influences on TMD pain. Prior to this study, researchers had identified many psychological variables that were linked to the development of TMD but few studies conducted a broad-based assessment of psychological functioning prior to the development of TMD. This was identified as a potential limitation and, as such researchers of the OPPERA study only included participants that had never had a prior TMD diagnosis (using RDC/TMD) and who reported no orofacial pain in the month before enrolment and no more than 4 months of orofacial pain in any month. The results showed that chronic TMD cases reported higher levels of psychological symptoms, affective distress, somatic symptoms and pain catastrophising compared to TMD-free controls. Since the OPPERA study, researchers have used the data collected by the study to conduct their own research. Notably, Fillingim et al. studied participants who initially were found not to have TMD when examined and followed them (for up to 5.2 years) in order to detect first onset TMD. In doing so, they identified psychological characteristics at enrolment that were linked to the development of first-onset TMD (5,29).

Studies into chronic pain and negative psychological factors have found that the two are intrinsically linked (29,30) . This link can be attributed to the ability of psychological stress to induce adaptive responses to physiological systems such as: increase hypothalamo-hypophyseal adrenal system activity, produce a dysfunctional pattern for pain inhibition and increase temporal somatization. These adaptive responses serve to cause sympathetic hyperactivity, decrease pain inhibition and reduce the threshold of negative, intense, long-term emotional experiences (30).

Chronic pain is the most common symptom of TMD mechanisms (7). Chronic TMD is a functional pain syndrome (along with conditions such as, fibromyalgia, irritable bowel syndrome and interstitial cystitis). These conditions have similar aetiology and often appear as comorbidities in individuals. These disorders all have the effect of producing chronic pain, the pain felt by sufferers is not considered a protective factor but, a result of increased central sensitisation and pain perception. Research has found that sufferers of TMD are more prone to experience allodynia (pain from innocuous stimuli), hyperalgesia (abnormally increased response to nociceptive stimuli) and secondary hyperalgesia (abnormally increased response to nociceptive stimuli delivered to the region surrounding, but not including, the zone of injury). If hyperalgesia takes place after tissue is injured, it results from increased sensitivity of primary afferent nociceptors found around the injured site (peripheral sensitization) and increased excitability of secondary afferent nociceptors found in the spinal cord (central sensitization) (30). The OPPERA study also concluded that painful TMD causes a trigger that elevates synaptic efficacy of neurons in nociceptive pathways, including pathways that participate in increased central nervous system sensitization (5). It is suggested that this correlation between painful TMD and increased central nervous system sensitisation can be explained by psychological stress altering physiological systems (as is the case seen in other chronic pain conditions), however, more research to establish a cause-effect relationship is needed as TMD symptoms, especially pain, are also discussed as

being a causative or intensifying factor in the development of depression and distress (29,30).

The realisation of the relationship between psychosocial factors and TMD has caused a shift away from a treatment plan solely focused on mechanistic models of TMD aetiology, (with some authors even claiming that, to do so would be to act unethically) and a move towards a biopsychosocial care medical model, involving a multidisciplinary response (29). To do this, clinician's need to clearly understand the epidemiology and aetiology of TMD to be able to identify patients that could be more susceptible, and work in cooperation with the patient to successfully manage and control their contributing factors (12,13).

Prevalence of Negative psychosocial Factors among Students

One population that has been flagged as more susceptible to TMD is the student population. Interestingly, they are also a group of society that notoriously suffer from mental health issues, they are continuously reported as having higher levels of psychological distress in comparison to the general population (31–33). Furthermore, healthcare students are particularly susceptible to psychosocial stressors (e.g. excessive workload, financial concerns and work-life balance) affecting their health. These stressors are further evident in the later phases of studies, when students are introduced to clinical practice and are subject to constant assessment, career planning and academic competition (34). As such, this group has been the subject of many investigations into the relationship of psychosocial factors and TMD (28,35,36).

Past Studies exploring the link between psychosocial factors and TMD within dental students

In 2018, Ahuja et al. conducted a study on Dental Students of the D. J Dental College Modinagar, Ghaziabad, Uttar Pradesh, India to investigate the causes of stress in this group and to study this stress as an aetiology of TMD. The researches also aimed to understand the variances of the level of stress among students of different years of study, age and gender (the first known research to study the stress levels of dental students in different age groups). The results confirmed the findings of other studies that TMD prevalence is higher in females than in males and that dental students are an risk group for heightened levels of stress and TMD. Furthermore, they found that stress levels were highest in dental students over twenty-five years and that the twenty-one to twenty-five year old age group had the highest prevalence of TMDs (28).

In 2019, Sójka et al. created a similar investigative study that would analyse the relationship between psychosocial factors and TMD. They included the Orientation of Life Questionnaire to measure individual's SOC. Orientation of Life Questionnaire together with the Four-Dimensional Symptom Questionnaire (4DSQ) evaluated the students intensity of stress manifestations and their individual ability to cope with this stress. They agreed with the consensus that medical students experience more stress than the average population, they added that high SOC was acting as a buffer to limit the effects of the stressors on physical health. Despite this, there was a high incidence of TMD symptoms and parafunctional habits found in this group (36).

Most recently, in 2021 a study conducted by Namvar et al. (37) assessed the relationship between depression and anxiety with TMD in dental students of Zanjan University of Medical Sciences, Iran. Students completed a questionnaire consisting of 21 questions that measure psychological distress and RDC/TMD self-report

questionnaire to assess for TMD symptoms. Results showed that there is a correlation between stress, depression, anxiety and TMD symptoms. They found that stress was the most effective factor in developing TMD.

Justification of the Study

The aim of this study is to recreate the study conducted in 2019 in a group of medical students in Poland by Sójka et al. (36). The previous study had a few key limitations that were highlighted by the author, as such, this study aims to amend the design of the previous study and to conduct it in a new population. With the aim of, being closer to coming to a more generalised consensus on the relationship of psychosocial factors and TMD in this group and to aid in the implementation of a biophysiological model of care among certain at risk population groups.

The limitations of the previous study included the fact that this study was undertaken in one university in Poland and might not be applicable in other cultures with another population. They called for further studies to be done on other populations to confirm their findings. This study aims to combat this limitation by assessing if similar findings are found in a population of dental students attending Universidad Católica de Valencia (UCV), Spain.

The study was also limited due to it analysing the 3 factors of Sense of Coherence (manageability, comprehensibility, and meaningfulness) as separate values rather than using the collective value of SOC. Antonovsky clearly stated that only one single total score should be calculated as the 3 values are interlaced with each other, as such, it is the collective values of all 3 factors that gives person a particular SOC score, not the value of only 1 factor (23). This study has altered the design so

that SOC is only one variable, one overall score equating to an individual's coping capacity.

Sójka et al. (36) also noted that there was a considerable amount of missing data that may contribute to potential selection bias. The data in the original study was collected from individual's over a period of time (the questionnaires first and if the patient reported TMD symptoms they would be invited for a clinical examination). This design led to a lot of losses as 38 people who reported symptoms of TMD did not attend for a clinical TMD examination, this loss of data gives rise to selection bias. This study design has been altered to collect all data (questionnaires and clinical examination) from all participants at the same day.

Objectives

Primary Objective

- To evaluate the relationship between intensity of stress manifestations (using the 4DSQ), SOC and probability of TMD (using DC/TMD) in a group of dental students in the Universidad Católica de Valencia (UCV).

Secondary Objective

- To study the prevalence of TMD in students enrolled in the clinical years of dentistry at UCV,
- To compare the prevalence of TMD and intensity of stress manifestations amongst males and females,
- To describe the relationship between SOC and intensity of stress manifestations (using the 4DSQ)
- To compare the level of stress manifestations and SOC in category (males and females) and category TMD (present or absent).

Hypothesis

Null Hypothesis

- There is no relationship between intensity of stress manifestations, SOC and probability of TMD,
- There is not an increased prevalence of TMD in students in their clinical years attending UCV than the general population,
- There is no effect of sex on prevalence of TMD
- There is no variance in level of stress manifestations and SOC in category (males and females) and category TMD (present or absent),

Alternate Hypothesis

- There is a relationship between intensity of stress manifestations, SOC and probability of TMD,
- There is a higher prevalence of TMD in dental students in their clinical years in UCV than the general population,
- Females are diagnosed more frequently with a TMD,
- Females show greater levels of stress manifestations and SOC,
- Those with more intense levels of stress manifestations and a lower SOC are more likely to have a TMD present.

Materials and Methods

Design

This descriptive investigatory study used cross-sectional data. It is a type of observational study used to study the prevalence of all the variables in a population at a single point in time. As such, data provided by the subjects filling out the 4DSQ and SOC questionnaires, together with, the clinical diagnosis of TMD using the DC/TMD was collected on the same day. This analyses allowed us to collect all the different variables and to study their relation with TMD.

Population of the Study

The study required dental students from UCV, from the later clinical years (years three to five). The total population of this group is 92 students (36 from 3rd year, 27 from 4th year and 29 from 5th year). Both female and male students will be included in this study. The researcher aimed to have 50% female participants and 50% males.

Calculation of the Sample Size

The estimation of the sample size was undertaken using a formula for cross-sectional qualitative studies (38). Using the software available from Fisterra (Annex 1), the following data was inputted: total population number is 92 students (the number of dental students in years three, four and five), we assume the prevalence of TMD is 5% (8), consider 95% confidence interval for a single population proportion, and a 5% precision. Using these values, the minimum sample size was calculated at 41. The sample size adjusted for loss was 48 (when considering an expected loss of 15%). I considered this a very feasible sample size for our study.

Sampling Technique

This study aimed to assess the relationship between TMD and psychosocial factors in dental university students in their clinical years of study. For this reason, all students who are potential candidates to participate in this study were collected from class lists. They were approached individually by the principle investigator, Alice Martina Drayne and given information of the study to gauge their interest of being included. Given the need for a sample size of 41 students, a first contact was made to analyse how many students were available and willing to participate in the study. A total of 48 students was randomly selected from this group.

Criteria of Selection

Inclusion criteria:

To be eligible to partake in this study, a participant must meet all of the following criteria:

- Be a current Dentistry student of the Universidad Católica de Valencia,
- Currently enrolled in the clinical years (three to five) of the degree.

Exclusion Criteria:

A potential participant that meets any of the following criteria will be excluded from participation in this study:

- Not able and/or willing to provide the information to fill out the questionnaires,
- History of serious systemic disorders, neurological disorders, cervical spine injuries or postural deformities,
- Presence of a comorbid condition such as: malignant diseases, substance abuse, pregnancy or nursing.

Variables

Main Variables

- Variable “TMD Pain Disorders”- the patient either has a TMD pain disorder or does not. If they do have a TMD pain disorder there are 3 levels of diagnosis: myalgia (two subtypes; local myalgia and myofascial pain with referral), arthralgia (two subtypes; right arthralgia and left arthralgia) and headache attribute to TMD,
- Variable “TMD Joint Disorders”- the patient either has a TMD joint disorder or does not. If they do have a TMD joint disorder there are 3 levels of diagnosis: disc displacement (four subtypes; disc displacement with reduction, disc displacement with reduction with intermittent locking, disc displacement with reduction with limited opening, disc displacement with reduction without limited opening), degenerative joint disorder and subluxation.
- Variable “Stress”- the patient either has a low, moderately high or very high presence of stress manifestations (somatisation, distress, anxiety and depression) as assessed with the 4DSQ

- Variable “Coping” – the patient has a high, moderate or low level of sense of coherence as assessed with the SOC-13

Secondary Variables

- Variable “gender”
- Variable “age”
- Variable “year group”

Procedure to Follow

Firstly, a short presentation was given to students who were interested in being involved in the study. A full explanation explaining what will be required of them, what type of information will be collected and how it will be used was given. The investigator explained that if patients have a history of serious systemic disorders, neurological disorders, cervical spine injuries or postural deformities or a presence of a comorbid condition such as: malignant diseases, substance abuse, pregnancy or nursing or if they are simply unwilling to be involved in the study that they can decline without giving an explanation as to why.

After which, students who wish to still be involved in the study were given an informed consent form to read, time to understand/ ask questions and asked to sign. Informed consent had to be signed by all participants before starting. Secondly, all participants were given full instructions on how to proceed with the study.

Thirdly, participants were given an envelope with a number on the front (an excel document was kept separate detailing who the number corresponds to so in the case that a participant wanted their details extracted from the study, it could be done).

Inside the envelope (shown in Annex 2) there was 6 pages of questionnaires with the same number on the envelope printed on the top of each page. The questionnaires were (general data, 4DSQ, SOC-13 and DC/TMD symptom). To prevent respondents from giving an induced answer, participants were instructed to fill out the questionnaires, taking all the time they needed. At this time, no further questions were allowed and no prompts given by the investigator. The SOC, 4DSQ and DC/TMD questionnaires were all validated in English and have been used extensively for research purposes (8,22,23). The questionnaires can be found in Annexes 3, 4 and 5.

After filling out the above questionnaires (Annexes 3-5), participants were asked to put them back in the envelope and to hand it back to the investigator. The investigator started the DC/TMD clinical examination and filled out the DC/TMD examination form (a two page document that can be found in Annex 6).

To protect the participant's identity, under the line "patient" the investigator denoted the unique identifier number that was at the front of the envelope the participant handed over. The full instructions on how to conduct the DC/TMD clinical examination, had been read and understood by the main investigator and acted as a guide in any uncertainty (39). The clinical examination took approximately 15 minutes, after which, the investigator put the examination form in the envelope and sealed it shut.

After the collection of all the data, the investigator opened the envelopes, one-by-one and scored the 4DSQ and SOC and used the DC/TMD symptom questionnaire and examination form to diagnose the participants with a pain TMD and/or a joint TMD or no TMD as per the diagnostic criteria of the DC/TMD (8).

The 4DSQ assesses four dimensions of psychological distress; somatisation (16 questions), distress (16 questions), anxiety (12 questions) and depression (6 questions). Each 4DSQ item gives a score of 0 (if a symptom is absent), 1 point (if a symptom is "sometimes" present) or 2 points (if a symptom is "regularly", "often" or "very often/constantly" present). The scores are then interpreted by a scale of three "low", "moderate" or "very high" (22). This is all depicted in the score form that can be found in Annex 7.

The 13-item SOC assesses a person's Orientation to Life, that is, their ability to perceive life as comprehensible (questions 2, 6, 8 and 9), manageable (questions 3, 5, 10 and 13) and meaningful (questions 1, 4, 7, 11 and 12). A few of the items have inverse values (questions 1, 2, 3, 7 and 10) meaning that if a person inputs item 1, it corresponds to a score of 7 or if a person inputs item 3, it corresponds to a score of 5 etc… Total scores range from 13-91 points, these are divided into 3 quartiles. 13-63 points correspond to a low SOC, 64-79 points to a moderate SOC and 80-91 points to a high SOC. They are scored on a 7-point Likert scale (from 1 to 7) (23).

Following the scoring of the questionnaires, the data was put into an excel document. The data was then changed to be understood by the statistical analysis software (i.e. qualitative data was changed to numbers). The investigator inputted all of the data from excel into JASP and begun the statistical analysis (using version JASP 0.15).

Statistical Analysis

All the outcomes were expressed as a mean and standard deviation (SD). The assumptions of normality and homogeneity of variance were tested using Kolmogorov-Smirnov and Levene test, respectively. To analyse the relationship between variables a Pearson or Spearman correlation coefficient was used.

The prevalence of TMD among the studied population was assessed using the following equation. Prevalence proportion = $\frac{No.\ of\ students\ with\ TMD}{No.of\ students\ in\ the\ study}$

To evaluate the relationship between intensity of stress manifestations (using the 4DSQ) and probability of TMD (using DC/TMD), we used a univariate logistic regression to test the probability of the two events occurring. The outcome variable was TMD (i.e., “TMD-pain disorders” and “TMD- joint disorders”) and SOC and each of the stress manifestations (i.e., “distress”, “anxiety”, “depression” and “somatisation”) as covariates.

To analyse the effect of sex (i.e., males and females) and category TMD (i.e., present or absent) on the level of stress manifestations an analysis of variance (ANOVA) was done. Bonferroni post Hoc test was done to analyse multiple comparison.

A statistical software package was used to analyse the data (JASP, The Netherlands). The level of statistical significance was set at $p \leq .05$.

Ethical Statement

Ethical considerations are in agreement with the Helsinki Declaration. Approval was received from the Comité de Ética de la Investigación (CEI) de la Universidad Católica de Valencia San Vicente Mártir (UCV2021-2022/058, 08.02.2022). The anonymity of the participants was preserved and no financial nor any other burden was placed upon them. Copy of the certificate of passing the CEI can be found in Annex 8.

Results

Clinical Descriptive Analysis- Prevalence of TMDs

A total of 48 dental students from UCV participated in this study. The study sample consisted of 14 students from 3rd Year, 14 students from 4th Year and 20 students from 5th Year. 1 male student was excluded due to incorrect completion of the questionnaire (see flowchart depicted in Figure 2).

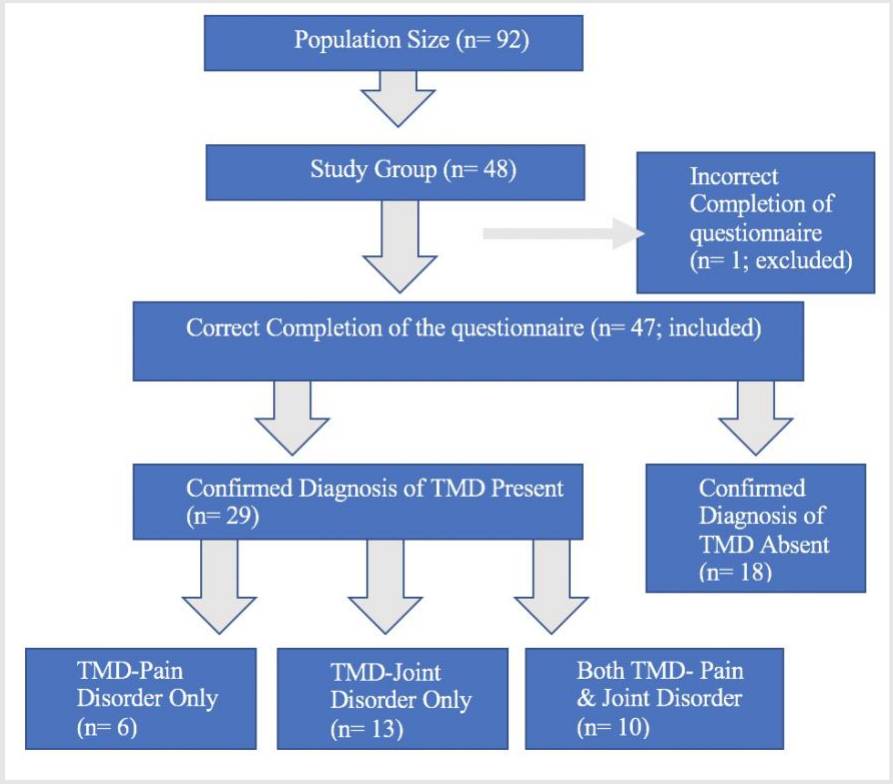

Figure 2. Flowchart of Study Participants, Inclusion and Exclusion and TMD Diagnosis

The final study group consisted of 47 students: 14 (29.79%) 3rd years, 14 (29.79%) 4th years and 19 (40.43%) 5th years. There were 24 females (51.06%) and 23 males (48.94%). The ratio of males and females was relatively proportionate among each year (as represented by the pie charts seen in Figure 3. Their age ranged between 20 and 29 (mean= 23.02) years old.

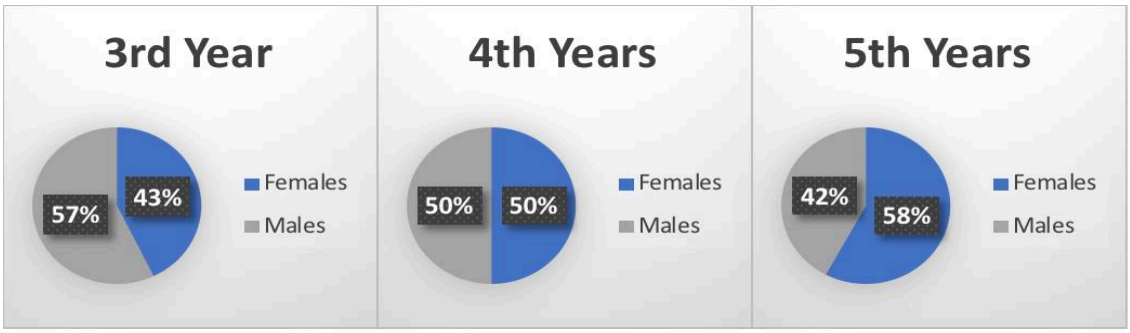

Figure 3. Distribution of Male and Female Student Participants Within Year Groups

29 (61.70%) students were diagnosed with a TMD and 18 (38.3%) students were verified to have no TMD present. The diagnosis of a TMD-pain disorder was clinically established in 16 participants (34.04%). The diagnosis of a TMD-joint disorder was clinically established in 23 participants (48.94%). 10 students were diagnosed with both a TMD pain and a TMD joint disorder, 6 with solely a TMD pain disorder and 13 with solely a TMD joint disorder (Figure 2).

3rd year had the highest prevalence of TMD absence whilst 5th years were more likely to be diagnosed with at least 1 TMD (73.68%). Figure 4 depicts the percentage of students within each year group diagnosed with; no TMD, only a TMD pain disorder, only a TMD joint disorder and those presenting with both a pain and joint disorder.

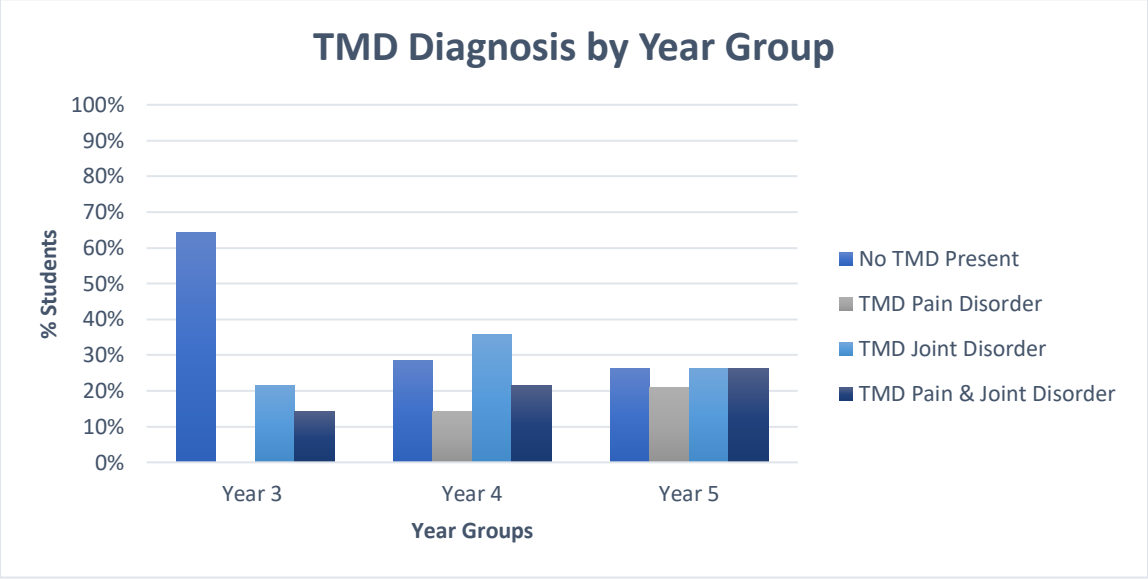

Figure 4. Bar chart of Diagnosis of TMD presence (with type) or absence by year group

The prevalence of females to males with at least one type of TMD was 15:14 female-to-male ratio. There was 9 females diagnosed with a TMD-pain disorder and 7 males. TMD-joint disorders represented the only diameter that showed a stark difference in prevalence. 15 females were diagnosed with a TMD-joint disorder compared to 8 males.

Of the 16 participants (34.04%) diagnosed with a TMD pain disorder 15 had clinically verified myalgia (8 local myalgia and 7 myofascial pain with referral), 8 were diagnosed with arthralgia (7 who presented arthralgia and myalgia together and 1 student who presented with arthralgia alone) and 3 were diagnosed with headache attributed to TMD (as a secondary diagnosis to myalgia).

Of the 23 participants (48.94%) diagnosed with a TMD joint disorder, 17 (73.91%) were diagnosed with a form of disc displacement (the prevalence of each form of disc displacement is displayed in Figure 5), 3 people with subluxation (2 presenting with a form of disc displacement and 1 student presenting with solely subluxation). 9 students were diagnosed with degenerative joint disease (4 presenting it with a form of disc displacement and 5 presenting with it alone).

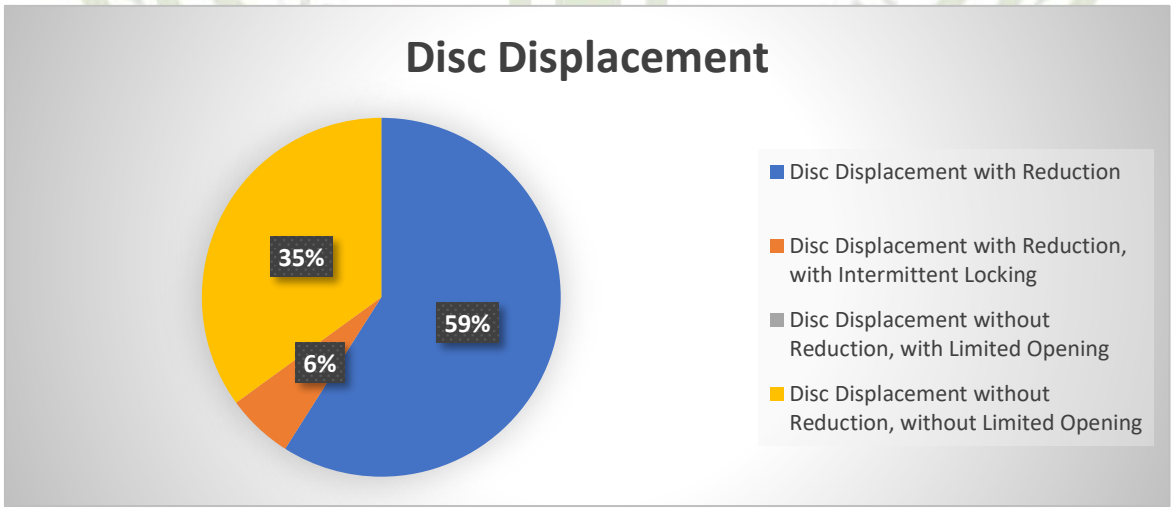

Figure 5. Prevalence of Different Forms of Disc Displacement

Spearman rho Correlation Coefficient

The variables “stress manifestations” and “SOC” were found to lack normal distribution, for this reason, Spearman rho correlation coefficient was used to evaluate their relationship

The results show that there are statistically significant relationship between all variables tested. There is a negative correlation between SOC and stress manifestations and a positive correlation between all variables of stress manifestation. The results from the Spearman correlation coefficients with a 95% confident interval between SOC and stress manifestations are summarised in Table 4 and visually depicted in the scatter plots in Figure 6 and Figure 7.

Table 4: Summary of relationship between SOC and intensity of Stress Manifestations

	Spearman rho	**R^2 (%)**	**p-value**	**95% CI**
SOC vs. Somatization	-0.54	29	< 0.001	-0.72 to -0.30
SOC vs. Distress	-0.63	40	< 0.001	-0.78 to -0.42
SOC vs. Anxiety	-0.50	25	< 0.001	-0.69 to -0.25
SOC vs. Depression	-0.41	17	0.004	-0.62 to -0.14
Somatisation vs. Distress	0.72	52	< 0.001	0.54 to 0.83
Somatisation vs. Anxiety	0.49	24	< 0.001	0.23 to 0.68
Somatisation vs. Depression	0.48	23	< 0.001	0.22 to 0.67
Distress vs. Anxiety	0.79	62	< 0.001	0.65 to 0.88
Distress vs. Depression	0.66	44	< 0.001	0.46 to 0.79
Anxiety vs. Depression	0.66	44	< 0.001	0.51 to 0.82

Note: CI= 95% of confident interval

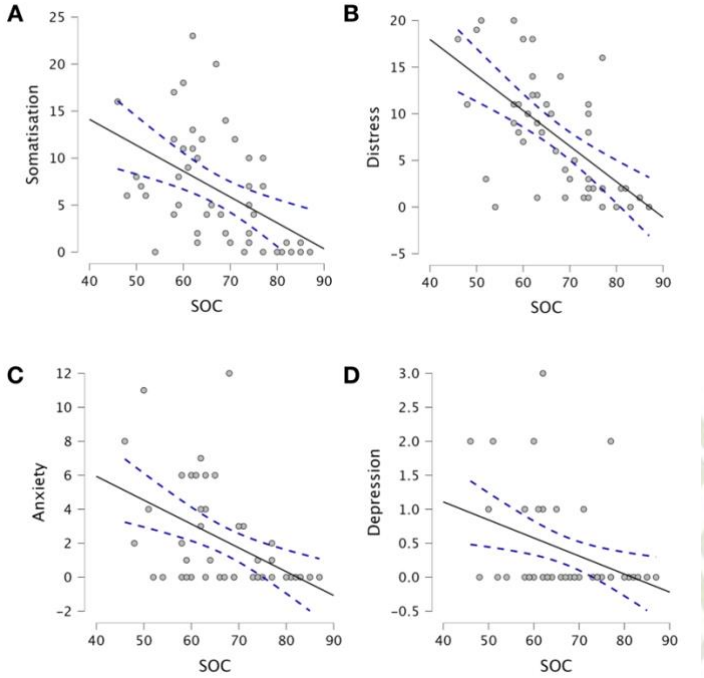

Figure 6. Scatter Plots showing the Negative Correlation between SOC & each Variable of Stress Manifestations;

A: SOC vs. Somatisation, B: SOC vs. Distress, C: SOC vs. Anxiety, D: SOC vs. Depression

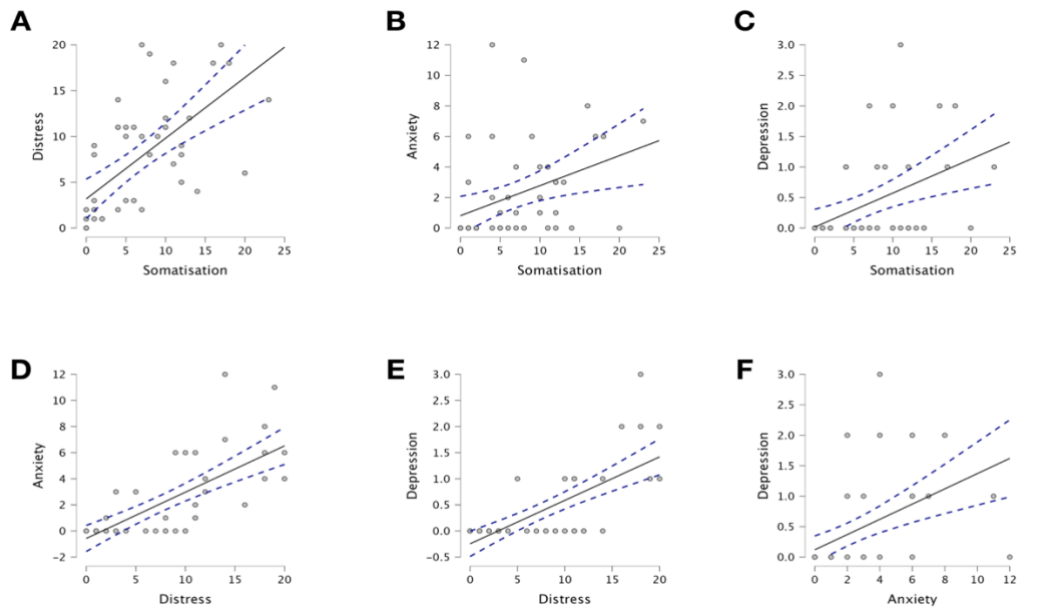

Figure 7. Scatter Plots showing the Positive Correlation between all Variables of Intensity of Stress Manifestations Correlations between Stress Manifestations; A: Somatisation vs. Distress, B: Somatisation vs. Anxiety, C: Somatisation vs. Depression, D: Distress vs. Anxiety, E: Distress vs. Depression and F: Anxiety vs. Depression.

Univariate Logistic Regression

The results showed statistically significant correlations between "TMD-pain disorders" and most variables of stress manifestations (i.e., "distress", "depression" and "somatisation"). There was no statistically significant relationship found between TMD-pain and anxiety. This is depicted in Table 5.

Table 5: Univariate Logistic Regression Analysis- Model Summary TMD- pain disorders

Covariates	Model	Nagelkerke R^2	p-value	Sensitivity	Specificity
Depression	H_1	0.11	0.044*	0.19	0.94
Anxiety	H_1	0.06	0.158	0.13	0.94
Distress	H_1	0.16	0.017*	0.31	0.87
Somatisation	H_1	0.30	< .001*	0.50	0.87
SOC	H_1	0.20	0.007*	0.31	0.87

(indicating statistically significant (p < 0.05) result)*

Table 5 shows that out of all the stress manifestations, somatisation and SOC had the strongest ability to predict the probability of a TMD- pain disorder. The somatisation model accounts for (30%) of the variability in TMD-pain disorders, whilst the SOC model accounts for 20% of the variability in TMD-pain disorders. These results are visually depicted in Figure 8, (showing that those with intense somatisation had a greater probability of being diagnosed with a TMD- pain disorder) and Figure 9 (showing participants with a higher SOC had a greater probability of not being diagnosed with a TMD- pain disorder).

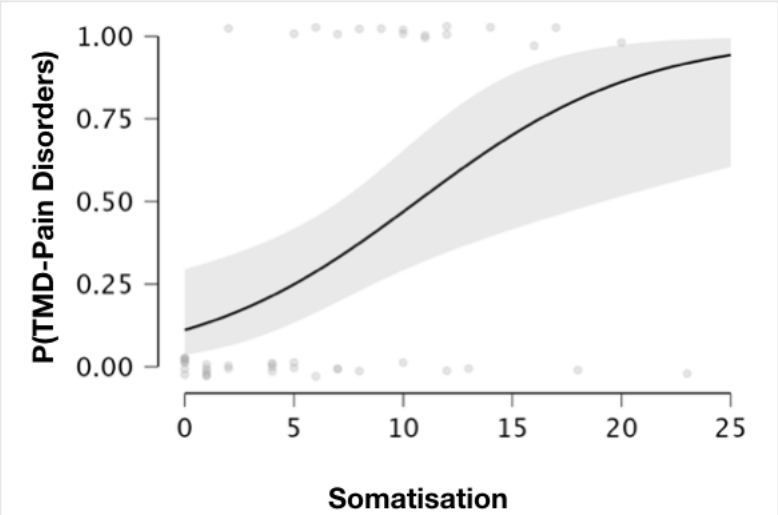

Figure 8. Conditional Estimate Plot showing the variability of TMD-pain disorders as a result of Somatisation

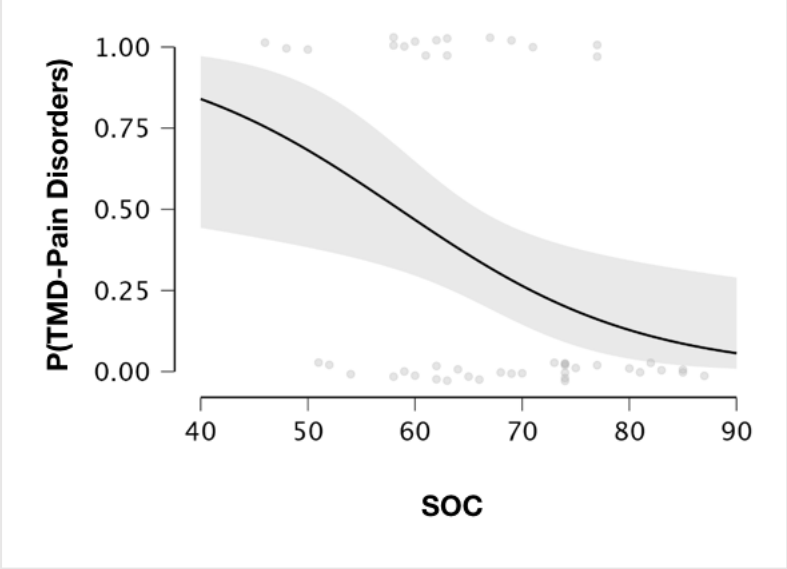

Figure 9. Conditional Estimate Plot showing the variability of TMD-Pain Disorders as a result of SOC

A Logistic Regression Model Comparison was employed to depict which model, (SOC or somatisation) had the best fit for the data, this is shown in Table 6.

Table 6: Logistic Regression Model Comparison

Model	Deviance	AIC	p-value	Nagelkerke R^2	AUC
H_0 Somatisation	60.28	62.28			
H_1 Somatisation	48.78	52.78	< .001*	0.30	0.83
H_0 soc	60.28	62.28			
H_1 soc	52.93	56.93	0.007*	0.20	0.73

(indicating statistically significant (p < 0.05) result). AIC: Akaike's information criterion, AUC: area under curve.*

The Somatisation H_1 model exhibited the best fit for the data with the lowest AIC (52.78) and the lowest deviance (58.78). In addition, it showed the highest Nagelkerke's R^2 (0.3) and AUC (0.83), indicating a good predictive ability.

We then tested the dependent variable "TMD-joint disorders". As shown in Table 7, there was no relationship found between any of the covariates tested and TMD- joint disorders. The Nagelkerke R^2 values were very low and none of the results shown were statistically significant ($p < 0.05$). Further, there was a low sensitivity and specificity for each of these tests.

Table 7: Univariate Logistic Regression Analysis- Model Summary TMD- joint disorders

Covariates	Model	Nagelkerke R^2	p	Sensitivity	Specificity
Depression	H_1	0.05	0.196	0.30	0.79
Anxiety	H_1	0.02	0.413	0.35	0.71

Distress	H_1	0.05	0.168	0.48	0.58
Somatisation	H_1	0.02	0.370	0.52	0.75
SOC	H_1	0.003	0.727	0.44	0.58

ANOVA

The ANOVA showed that there was not an effect of sex on depression (F = 2.38, p = 0.130). There was a tendency of significance of the effect of TMD- pain disorders on depression (F[1.43] = 3.76, p= 0.059). There was no interaction effect between sex and TMD-pain disorders on depression (F[1.43] = 3.15, p = 0.083).

Confirming the null hypothesis, the ANOVA showed that there was not an effect of sex on anxiety (F[1.43] = 0.23, p = 0.631), that there was not an effect of TMD-pain disorders on anxiety (F = 1.76, p = 0.191) and that there was no interaction effect between sex and TMD- pain disorders on anxiety (F[1.43] = 1.94, p = 0.171).

There was a tendency of an effect of sex on distress but the results were found not to be statistically significant (F[1.43] = 3.45, p = 0.070). There was statistically significant differences on main effect of TMD-pain disorders on distress (see Table 8). There was no interaction effect between sex and TMD-pain disorders on distress (F[1.43] = 1.48, p = 0.230).

There was a tendency of an effect of sex on somatisation but, the results were found not to be statistically significant (F[1.43] = 3.91, p = 0.055). The main effect of TMD-pain disorders on somatisation were found to be statistically significant (as depicted in Table 8). There was no interaction effect between sex and TMD-pain disorders on somatisation (F[1.43] = 1.22, p = 0.276).

From the data collected, there was found to be no effect of sex on SOC (F[1.43] = 0.78, p = 0.383). However, there was a statistically significant effect found between TMD-pain disorders on SOC (shown in Table 8). There was no interaction effect between sex and TMD-pain disorders on SOC (F[1.43] = 0.004, p = 0.953).

Table 8. Two-Way ANOVA: Main Effect of TMD- Pain Disorders on Distress, Somatisation and SOC

Cases	$F_{[1.43]}$	p
ANOVA- Distress	5.36	0.025*
ANOVA- Somatisation	14.47	< 0.001*
ANOVA- SOC	7.09	0.011*

(indicating statistically significant (p < 0.05) result).*

As depicted in Table 8, the ANOVA showed statistically significant differences on main effect of TMD-pain disorders on distress, somatisation and SOC. Post Hoc Bonferroni Correction was conducted and showed statistically significant mean differences among some groups.

For somatisation, the group “Males TMD-pain absent” and “Males TMD-pain present” differed significantly with a p-value of 0.01 and a mean difference (MD) and 95% CI of -7.67 (-13.79 to -1.55). This result is shown in the descriptive plot presented in Figure 9 where we can see the somatisation scores between men who had TMD-pain disorders differed significantly, whereas, there was not as great of difference on main effect seen in females. Between the groups “Females, TMD-pain present” and “Males, TMD-pain absent”, Post Hoc Bonferroni correction showed a MD and 95% CI of 9.04 (3.41 to 14.66), p-value of < 0.001 (Figure 10). There was

also a MD and 95% CI of -5.95 (-9.10 to -2.79), p-value of < 0.001 between the groups “TMD-pain absent” and “TMD-pain present”.

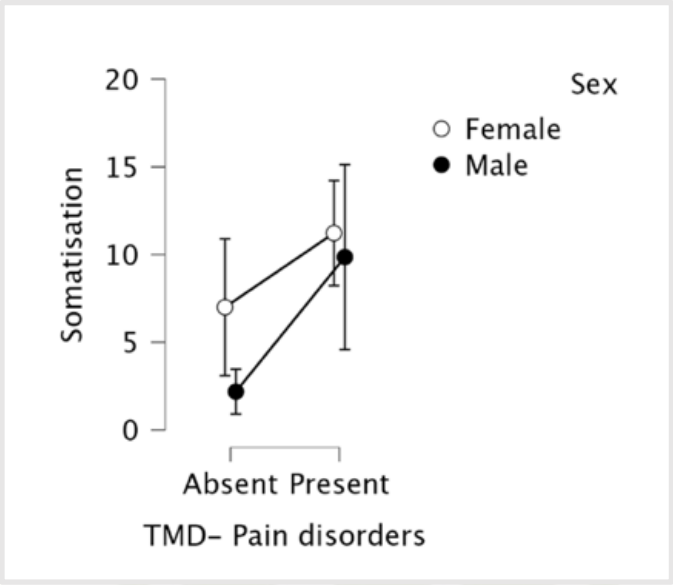

Figure 10. Descriptive plot depicting the Interaction Effect of Sex and TMD-Pain Disorders on Somatisation

For distress, Post Hoc Bonferroni correction showed a MD and 95% CI of -4.196 (-7.85 to 0.54), p-value of 0.025 between the groups “TMD-pain absent” and “TMD-pain present”. There was also a MD and 95% CI of 7.56 (1.05 to 14.08), p-value of 0.02 between the groups “Females, TMD-pain present” and “Males, TMD-pain absent”.

For SOC, Post Hoc Bonferroni correction showed a MD and 95% CI of 8.13 (1.97 to 14.29), p-value of 0.011 between the groups “TMD-pain absent” and “TMD-pain present”.

Looking at the main effect of “sex” and “TMD-joint” on stress manifestations and SOC, there was found to be no statistically significant effect of sex or TMD-joint

disorders on any of the units of stress manifestations or on SOC. However, there was a statistically significant interaction effect found between sex and TMD-joint disorders on somatisation and SOC. This is depicted in the Table 9.

Table 9. Sex ∗ TMD- Joint Disorders on Stress Manifestations and SOC

Cases	$F_{[1.43]}$	p
ANOVA- Depression	3.64	0.063
ANOVA- Anxiety	1.04	0.313
ANOVA- Distress	3.61	0.064
ANOVA- Somatisation	4.56	0.038*
ANOVA- SOC	4.74	0.035*

(indicating statistically significant (p < 0.05) result)*

Post Hoc Bonferroni correction was conducted on the dependent variables that were found to be statistically significant (SOC and somatisation). There was no difference found between any groups (p > 0.05) on SOC. However, we could see some statistically significant differences between the groups on somatisation. Comparisons of sex on somatisation showed a MD and 95% CI of 4.07 (4.07 to 0.65), p-value of 0.021. Also, there was a MD and 95% CI of 7.69 (1.16 to 14.22), p-value of 0.018 between groups “Females, TMD-Joint present” and “Males TMD-Joint present”. The descriptive plot in Figure 11 clearly shows how females with a TMD-joint were more likely to have greater intensity of somatisation than males with a TMD-joint

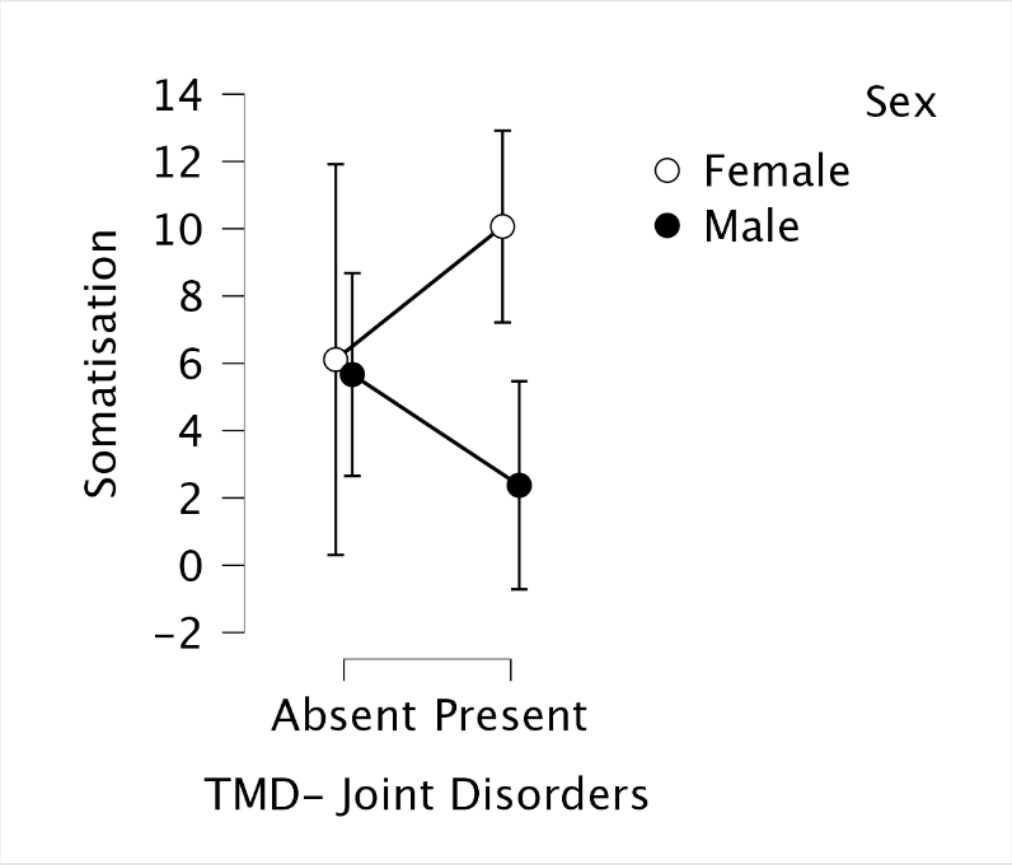

Figure 11. Descriptive Plot showing the Interaction Effect of Sex and TMD-Joint on Somatisation

Discussion

The primary objective of this study was to evaluate the relationship between intensity of stress manifestations (using the 4DSQ), SOC and probability of TMD (using DC/TMD) in a group of dental students in the Universidad Católica de Valencia (UCV).

Our results gathered from the univariate logistic regression indicated that there was no correlation between stress manifestations, SOC and TMD-joint disorders. However, a positive correlation between some stress manifestations, SOC and the probability of having a TMD- pain disorder existed. The results showed that we can accept the null hypothesis that there is no relationship between anxiety and the probability of having a TMD. However, in every other factor tested (SOC, distress, depression and somatisation) a correlation to TMD-pain disorders was found.

The result that there was no correlation between anxiety and TMD is inconsistent with recent research showing a correlation between both anxiety and TMD-joint and anxiety and TMD-pain disorders (40). Most surprising is that there was no connection between anxiety and TMD-pain disorder as a strong link has been seen in other research and anxiety has even been highlighted as a major contributor to the transition from acute to chronic pain (40,41).

An explanation for this could be that the 4DSQ does a good job at separating distress symptoms (which is a more general view on how taxing it is for people to maintain psychosocial homeostasis and is often a stand-alone problem) from more specific anxiety symptoms. Once you separate distress from anxiety you get the very core of the anxiety disorder. More often, the research that shows a correlation between anxiety and TMD-pain disorder uses a scale that doesn't include distress as its own diagnosis so what is being measured would actually be classified as distress and not anxiety if they used the 4DSQ scale (22,40,41). However, it should be noted that Sójka et. al (36) reported a statistically significant relation between anxiety and TMD-pain disorders and they used the 4DSQ. It is an interesting inconsistency that shows differing results amongst different population studies and calls for the need of more research into this field for further clarification.

The strongest correlation found was the relationship between the intensity of somatisation on TMD-pain disorders. Somatisation was found to account for 30% of the variability in TMD-pain disorders. This is consistent with research conducted by Fillingim et al. (29) who found that the highest hazard ratio for predicting TMD onset was a measure of somatic symptoms experienced. They also concluded that rate of TMD was highest in those in the upper tercile of intensity of somatic symptoms. These finding are accordant with the OPPERA study that found that somatisation was the psychological manifestation that differed most between TMD- patients and TMD- free controls (5). The fact that prospective, case-control and this cross-sectional study all come to the conclusion that increased somatisation increases probability of TMD implies that somatisation is associated with both onset and chronicity of TMD (5,29).

Our results showed that there is a correlation (although weak) between depression and TMD-pain disorders and distress and TMD-pain disorders. For this reason, we can accept the alternate hypothesis that there is a relationship between depression and TMD-pain disorders and distress and TMD-pain disorders. Research has shown a strong correlation between depression and pain disorders for a long time. It is widely reported that depression is associated with reported severity of pain, sensitivity to pain and physical disability in rheumatological conditions. They have also been shown to predict pain-related sequalae, more specifically, they are the strongest predictors for the transition from acute to chronic pain (41).

It is a little unclear whether the correlation shown between the aforementioned psychological factors and TMD-pain disorders is purely aetiological or reactionary (if psychological symptomology is an aetiology of pain or if pain is an aetiology of psychological symptomology). There is an abundant amount of research showing the link between pain leading to psychological symptoms but, pre-morbid psychological symptoms causing the onset of pain or representing a risk factor for future development of pain is more of a controversial topic (12,27,28). Prospective

studies (including the OPPERA study) has suggested that psychological factors pose a risk factor to the eventual onset of chronic pain, but more prospective studies need to be carried out to affirm this link (5,42). The physiological mechanism of the stress response could shed some light on the proposed link between psychosocial factors and TMD and is discussed, in depth later in this section.

Our results also showed a strong negative correlation between SOC and TMD- pain disorders, allowing us to reject our null hypothesis that there was no relationship. This means that the higher value of SOC an individual had, the less probability they would be diagnosed with a TMD-pain disorder. This result mirrors that of Sójka et al. (36) who reported that students without symptoms of TMD reported higher scores in Antonovsky's Orientation to Life Questionnaire (assessing meaningfulness, comprehensibility and manageability). This link could be attributed to the salutogenic idea that poses the hypothesis sense of coherence acts as a buffer to pain as it pools resistance/ deficits influences on pain (43).

Pain is a subjective experience, it is made subjective by our pain behaviour, that is, the set of learned behaviours that is set in motion when one experiences pain. Pain behaviours are influenced by emotions, cognitions and environmental factors that are initiated to help the sufferer cope better with the pain. With this in mind, it is a logical conclusion that our meaningfulness, comprehensibility and manageability (SOC) of the pain influences our pain behaviour and thus, our pain perception, making us more (or less) resistant to the influence of pain (43,44). Our pain behaviour is influenced by past experiences of similar pain that we have so that the body will remember the reaction it took the last time to help tackle the proposed threat (44). This physiological system is maintained with the release of cortisol during the stress response which, has the ability to consolidate fear- based emotional memories and condition a sensitized physiologic stress response (44,45). This system and how it affects pain intensity, pain perception and pain sequelae is described by the physiological stress response's biological cues outlined below.

The biological cues explaining the association between psychosocial factors and TMD- pain disorders are not fully understood. A popular explanation for the link is that whilst psychosocial factors do not directly influence the onset of TMD-pain disorders, they trigger a set of underlying processes that modulate pain. To understand this, we first need to understand that pain elicits the body's physiological stress response- signals from the amygdala activates the hypothalamic pituitary (HPA) axis causing a secretion of sympathetic catecholamines (epinephrine and norepinephrine) from the brainstem. This then causes an increase in cortisol (a neuroendocrine hormone). Cortisol has a prolonged effect working as an anti-inflammatory (45,46).

However, our perception of what is stressful and thus, what induces this response is individual and if we interpret something harmless as threatening or if we are constantly eliciting the stress response, it could cause a maladaptive response to the pain, exasperate the physiological stress response leading to cortisol dysfunction, inflammation and pain. The moment the exasperation of the stress response leads to cortisol dysfunction is the moment the acute condition becomes chronic (as depicted in figure 12). This chronic inflammation can lead to a cycle of inflammation, pain and depression or other psychological factors (45). This could explain why this studies' ANOVA found a statistically significant relationship of TMD-pain disorders on the SOC, distress and somatisation. Also why the research showed a tendency to a correlation between TMD-pain disorders and depression.

Another important consideration is that pain itself is a stressor that can initiate a maladaptive response to the pain and increase sensitivity to an afferent nerve stimulus (peripheral sensitisation). This, in itself can explain why SOC and stress manifestations are related with TMD-pain disorders. Even further than that, this system is modulated by factors, such as genetics and sex that can further explain

why some people are more privy to TMD-pain disorders than others (45). This could help explain why females with TMD-joint suffered much greater levels of somatisation than their male counterparts.

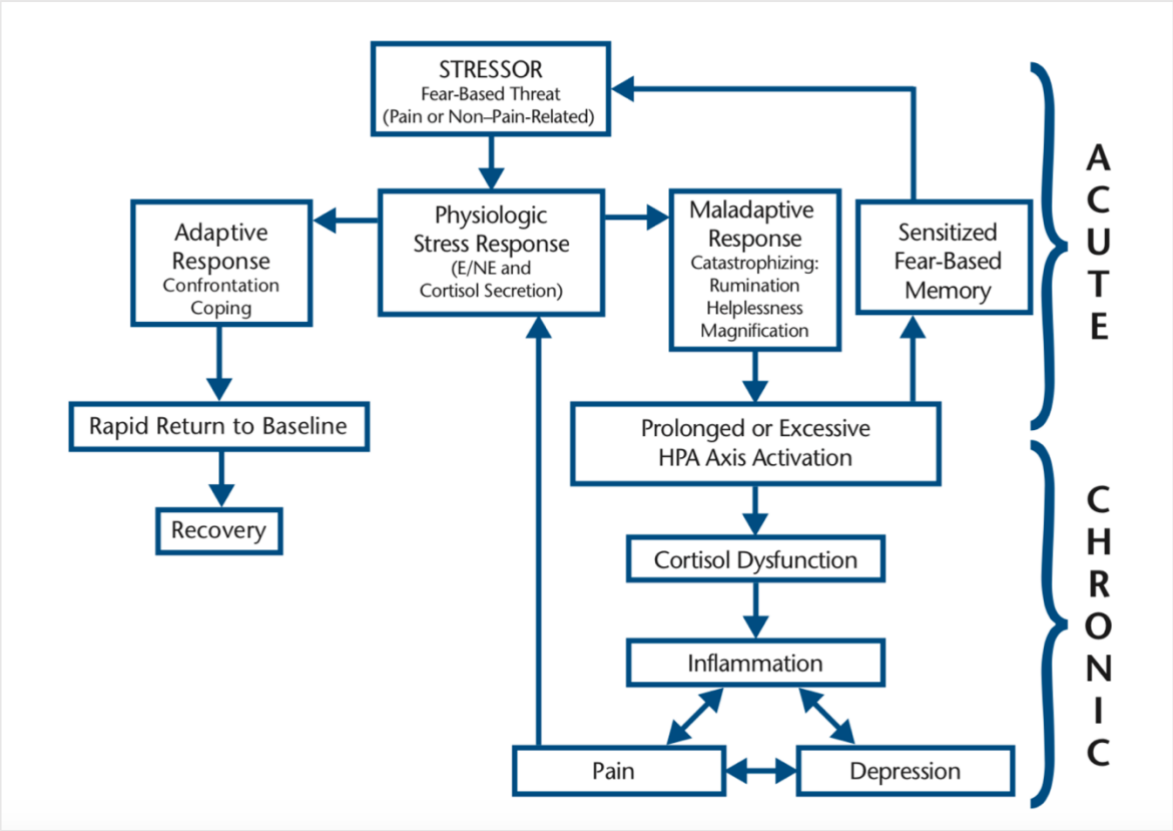

Figure 12. Proposed role of stress related hypothalamic-pituitary-adrenal (HPA) axis activation in the transition from acute to chronic pain. (Photo taken from Hannibal et al. (43)).

Researchers have been focusing on genes that not only are critical to the stress response but also, have an association with clinical TMD and pain. A gene that fits these dimensions is the gene catechol *O*-methyltransferase (*COMT*). COMT regulates catechol neurotransmitter catabolism (critical for the stress response (as described above)) and has been shown to have a strong association with pain sensitivity (5,47). Low COMT activity and stress increases epinephrine load and thus, increases pain sensitivity. As both factors cause an increase in epinephrine

and pain it is difficult to pinpoint which variable has more of an effect. Low COMT activity is associated with HPS (high pain sensitivity) haplotype (5,48). The relationship is made more complex by the fact that sex is also an important factor to consider when studying COMT (due to the estrogenic down-regulation making females produce less COMT). Additionally, males and females have shown to react differently to this effect (i.e. males with high stress and more number of copies of the HPS haplotype, have a decreased pain sensitivity, however, for women it has the opposite effect) (48). In any case, it is certain that COMT can help explain the relationship between genetics, sex, stress manifestations and increase cases in TMD-pain disorders and can help explain why some people (those with HPS haplotype or high stress) are more at risk of developing TMD-pain disorders than others (5,48).

Another group who are said to be at risk of developing TMD disorders than others are females. Many research has highlighted the heightened prevalence of TMD disorders in women than in men. This is only partly corroborated in our research. We saw that the prevalence of TMD-joint disorders was much higher in females than in males (15 females compared to 8 males). However, there was not much difference in prevalence in TMD-pain disorders (9 females compared to 7 males) and not much difference in the prevalence of having at least one TMD (15 females to 14 males).

There is a reported female-to-male ratio of 2:1 in the general population to more than 4:1 in clinical trials (18). This result was almost mirrored in our research where we saw a 1.8:1 female-to-male ratio of people diagnosed with TMD-joint disorders. However, this trend was not present in categories “TMD-pain disorders” and the category “at least 1 TMD”. This was surprising, as in other studies females reported increased diagnosis of TMD-pain disorders than men. It was proposed that the fluctuating oestrogen during pre, post and peri-menopausal times predisposes

women to increased pain sensitivity and this could explain why some women experience increased diagnosis of TMD-pain disorders (49).

Oestrogen is also proposed to have an effect on TMD joint disorders (49). The possible theory that could help explain the sexual disequilibrium is the suspected effect of oestrogens on TMD-joint laxity by enhancing MMP, causing cartilaginous breakdown of the TMJ'S fibrocartilage (3). Studies have shown that disc disorders peaked in women of child-bearing age, our study population consisted of students aged 20-29, this could explain the sexual difference between TMD-joint disorders diagnosed in males and females in this study (49).

However, the individuality of oestrogen levels, fluctuations during a females menstrual cycle coupled with varying levels throughout a females life makes it very difficult to design a study that can draw correlations between the effect of oestrogen on a females predisposition to getting a TMD disorder (17). This limitation of TMD study designs is made clear by the fact that TMJ Degenerative Joint Disorders increase in women over 50 years old whilst in the same age group disc displacement disorders decrease (49). On top of this, TMD is made up of 12 different forms, so this makes it complicated to try to pinpoint the mechanistic role in contributing to each one of the forms (8).

Another possible explanation for increased prevalence of TMD-joint disorders in females in this study could be explained by differences in TMJ anatomy. The male condyle is larger and has a longer condylar lingual length with long elliptical condyles. This could explain why males are more protected from mechanical fatigue of the TMJ and thus, decreased TMD-joint prevalence. The female joint is smaller and rounder resulting in increased joint loading and possible contribution to the development of TMD-joint disorders (50).

Corroborating with other research, was this study's finding that students have an increased prevalence of TMD. This could be due to the findings that heightened stress manifestations are often found in this population at increased intervals (34). As explained before, the chronic activation of the stress response can predispose to TMD (30,45). This study's findings show a relationship of TMD-pain on distress and a tendency to a relationship between TMD-pain on depression. There was a high prevalence of distress symptoms. Although, mild distress states do not interfere much with normal functioning, 34% of students who participated in this study were diagnosed with moderate distress, bordering on severe symptoms. Severe distress states are characteristic of nervous breakdown and can cause withdrawal from occupational duties (22). With this in mind, it is clear that this population is at risk of severe stress manifestations and better support mechanisms should be inputted to soften the effects of a demanding and competitive educational environment (31–33).

This group was also found to have a high level of somatisation (25.53% of students had a moderate to high level of somatisation). This study showed that there was a strong correlation between TMD-pain on somatisation (particularly among the female cohort). This could be explained with the fact that the participants are students studying in a medical field, thus they have a heightened awareness of bodily sensations and much more likely to self-diagnose their symptoms that would often go unnoticed in another population with similar sensations. This idea is supported by a study conducted in two Nigerian Universities on 385 medical students, they found that 14.3% had psychosomatic disorder (51). This is compounded by the fact that medical students are less likely to seek healthcare for their proposed symptoms as they harbour anxiety over feeling patronised for revealing them (52). These mechanisms together could be what contributes to increased prevalence of TMD in this group (28,35,36).

Our findings indicate that certain groups of the population may be more at risk of developing TMD than others (i.e. females, dental students and those with high stress manifestations and low SOC). These findings corroborate the need for a biopsychosocial model of care, with a multidisciplinary response (29). As pain or other symptoms of bodily dysfunction varies among individuals, so too does our idea of what is threatening or what is innocuous (i.e. what will produce a stress response or what will not) (45). This could be what causes a varying progression of TMD and a varying response to symptoms in the individual seen in this study. These findings mirror the study conducted by Edwards et al. (41) on rheumatic diseases that concluded that the presence of depression had a bigger correlation with a patient's subjective pain score than did objective radiographic evidence of disease. As such, these findings could support decision- making in treating TMD (40).

A biopsychosocial model of care could help combat this response by identifying early stress and including stress management education into the treatment plan, with the hope of reducing pain and other symptoms involved in TMD (45). There is also important considerations of stress on pain management, for example, high levels of anxiety and stress can reduce a patient's analgesic response to opioids something worth considering when treating patients with such drugs (41). The effects of sex on TMD in this study and many others are contradictory and confusing, more research needs to be done to understand more clearly the link between sex, stress, sex hormones and their relation with TMD. Despite this, it is an important consideration to understand that females could have increased prevalence to certain types of TMD during certain periods of their life or of their menstrual cycle. Dentists and other healthcare professionals diagnosing/ treating TMD should consider these results to prevent them making erroneous generalisations and to open up the possibility of sex-specific potential treatment pathways under a biopsychosocial care treatment plan for our female patients (3,17,49).

This study design was improved from the study conducted by Sójka et al. (36) as laid out in this study's justification. However, limitations are still present. One limitation of this study is the possible skewing of results due to omitted variable bias. One such emission are genetical factors. Meloto et al. discovered that the effect of stress on pain is modified by its interaction with the HPS haplotype (causing low COMT activity) and sex. So that, in males with high stress with more number of copies of the HPS haplotype, pain sensitivity decreased. However, in females they noted the exact opposite, females with high stress showed no change in pain sensitivity regardless of number of copies of the HPS haplotype. This is an important consideration when reviewing our results as it suggests that some correlations may be skewed by factors that we cannot take into consideration (e.g. genetic factors) (48).

Another limitation is small sample size. The sample size was 47 which, was greater than the 41 that was calculated that we needed for this study using a formula and the software Fisterra (38). In spite of that, the prevalence of TMD that was inputted according to our research was 5% (8). However, it seems that this prevalence was too modest (perhaps as it was taken from suspected TMD prevalence in the general population and the population of medical students have a much higher prevalence of TMD (this study showed a prevalence of 61.70%).

Going forward, to improve this study design, I would recommend the inclusion of more variables so that we can understand the different mechanisms involved in TMD better. In particular I would ideally want to measure oestrogen in participants to understand better its correlation with TMD. On top of this, obtaining genotypic data from purified blood samples from each participant so that we can study the participant's COMT haplotypes and their relation with stress manifestations and TMD prevalence.

I would also recommend a larger sample size. I would set my prevalence to 61.70% (as found in this research). If the population size was the same as this study's (92 students), I would conduct the study on minimum 74 people (87 adjusted for losses). The larger the sample, the more precise the results would be.

It would also be helpful to conduct the same study on a similar population group with a similar demographic (age, sex and social standing) but who are not studying dentistry. This could help us understand better the predilection for stress manifestations and TMD within dental students.

As this was a cross-sectional study design, recording status at only one point in time, there could be incidence-biased. For this reason a prospective study should be designed to follow this research further.

Despite all of the recent research into this field, there is still gaps in our knowledge and more experimental research with a prospective study design are required to better illustrate the definitive mechanism between psychosocial factors, sex and TMD-pain disorders.

Conclusion

The primary objective of this study was to evaluate the relationship between intensity of stress manifestations, SOC and probability of TMD in a group of dental students in UCV. After having followed this study, we can conclude that a relationship between intensity of somatisation, distress, depression, SOC and TMD-pain disorders exist. Somatisation and SOC were found to be able to best predict

the presence of a TMD-pain disorder. Distress and depression were also able to predict the presence of a TMD-pain disorder but, with less accuracy.

On the other hand, this study showed no relationship between intensity of anxiety and TMD-pain disorders and no evidence to support a relationship between any psychosocial variable tested and TMD- joint disorders.

Results showed that females in this population group were more likely than men to have a TMD-joint disorder. Males with TMD-joint had a much lower somatisation than females with TMD-joint. However, there was insignificant differences found in male and female prevalence of TMD-pain disorders.

There was an effect of TMD-pain on distress, somatisation and SOC. This result draws the conclusion that experience of TMD-pain can perpetuate or contribute to the onset or intensity of some stress manifestations (distress and somatisation) the effect of which, can be buffered by a high SOC. Moreover, the research indicated that females with TMD-joint experience higher levels of somatisation than males with TMD-joint.

Our results highlight dental students as an at risk group for intense stress manifestations and a heightened risk of TMD (risk increases towards the later years of study). This research calls on the need for a support system and stress management education to help offload these burdens.

Finally, our results support the implementation of a biopsychosocial model of care so that dentists and other healthcare providers can be better equipped to single out populations at risk of developing TMD and offer them prompt, individualised interventions that is supported by a multidisciplinary team.

Conflict of interest

None declared.

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Annexes

ESTIMAR UNA PROPORCIÓN

Total de la población (N) (Si la población es infinita, dejar la casilla en blanco)	92
Nivel de confianza o seguridad (1-α)	95%
Precisión (d)	5%
Proporción (valor aproximado del parámetro que queremos medir) (Si no tenemos dicha información p=0.5 que maximiza el tamaño muestral)	5%
TAMAÑO MUESTRAL (n)	41

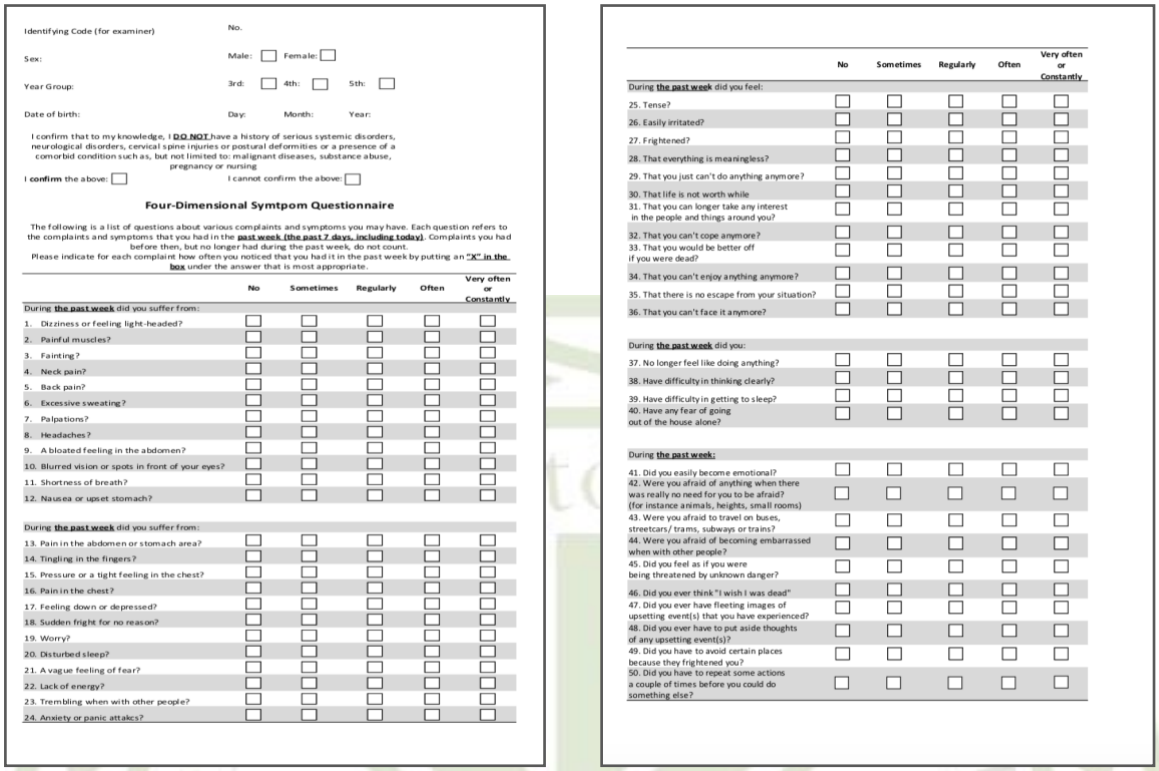
EL TAMAÑO MUESTRAL AJUSTADO A PÉRDIDAS

Proporcion esperada de pérdidas (R)	15%
MUESTRA AJUSTADA A LAS PÉRDIDAS	48

Beatriz López Calviño
Salvador Pita Fernández
Sonia Pértega Díaz
Teresa Seoane Pillado
Unidad de epidemiología clínica y bioestadística
Complexo Hospitalario Universitario A Coruña

Annex 1. Estimation of the sample size using software "Fisterra"

Annex 2. Envelope supplied to each participant containing 6 pages of questionnaires



Identifying Code (for examiner) No.

Sex: Male: ☐ Female: ☐

Year Group: 3rd: ☐ 4th: ☐ 5th: ☐

Date of birth: Day: Month: Year:

I confirm that to my knowledge, I **DO NOT** have a history of serious systemic disorders, neurological disorders, cervical spine injuries or postural deformities or a presence of a comorbid condition such as, but not limited to: malignant diseases, substance abuse, pregnancy or nursing

I **confirm** the above: ☐ I cannot confirm the above: ☐

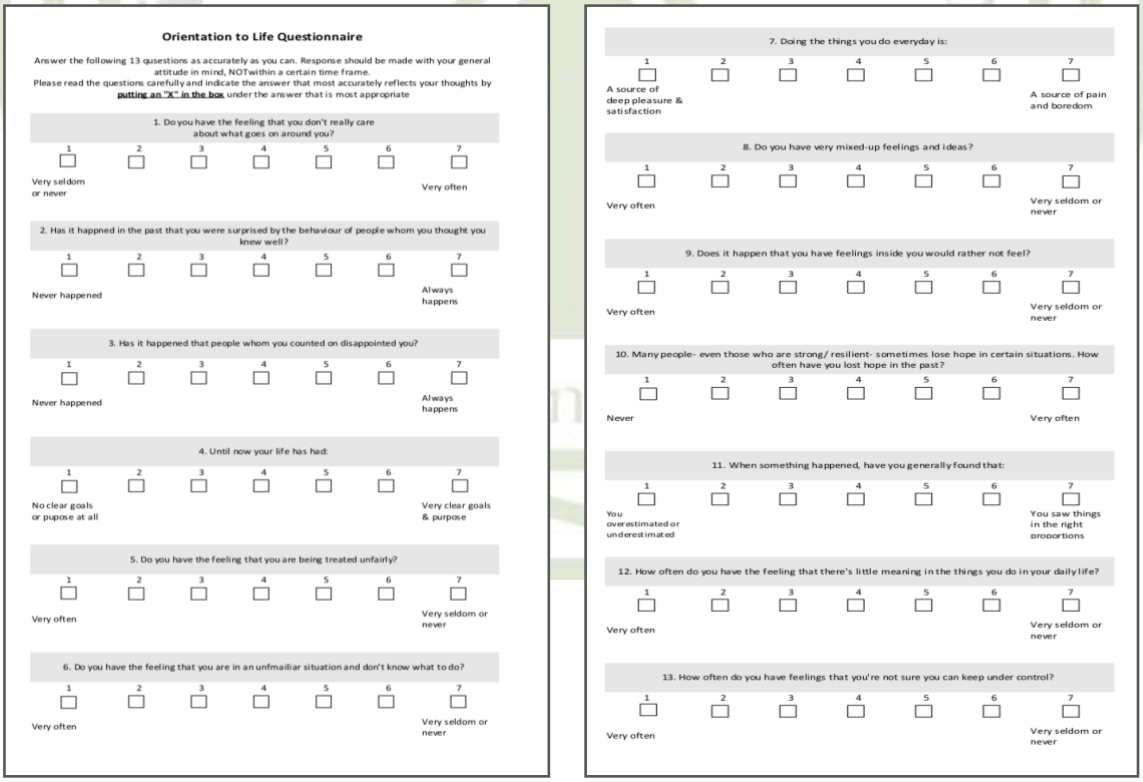
Four-Dimensional Symtpom Questionnaire

The following is a list of questions about various complaints and symptoms you may have. Each question refers to the complaints and symptoms that you had in the **past week (the past 7 days, including today)**. Complaints you had before then, but no longer had during the past week, do not count.
Please indicate for each complaint how often you noticed that you had it in the past week by putting an **"X" in the box** under the answer that is most appropriate.

	No	Sometimes	Regularly	Often	Very often or Constantly
During **the past week** did you suffer from:					
1. Dizziness or feeling light-headed?	☐	☐	☐	☐	☐
2. Painful muscles?	☐	☐	☐	☐	☐
3. Fainting?	☐	☐	☐	☐	☐
4. Neck pain?	☐	☐	☐	☐	☐
5. Back pain?	☐	☐	☐	☐	☐
6. Excessive sweating?	☐	☐	☐	☐	☐
7. Palpations?	☐	☐	☐	☐	☐
8. Headaches?	☐	☐	☐	☐	☐
9. A bloated feeling in the abdomen?	☐	☐	☐	☐	☐
10. Blurred vision or spots in front of your eyes?	☐	☐	☐	☐	☐
11. Shortness of breath?	☐	☐	☐	☐	☐
12. Nausea or upset stomach?	☐	☐	☐	☐	☐
During **the past week** did you suffer from:					
13. Pain in the abdomen or stomach area?	☐	☐	☐	☐	☐
14. Tingling in the fingers?	☐	☐	☐	☐	☐
15. Pressure or a tight feeling in the chest?	☐	☐	☐	☐	☐
16. Pain in the chest?	☐	☐	☐	☐	☐
17. Feeling down or depressed?	☐	☐	☐	☐	☐
18. Sudden fright for no reason?	☐	☐	☐	☐	☐
19. Worry?	☐	☐	☐	☐	☐
20. Disturbed sleep?	☐	☐	☐	☐	☐
21. A vague feeling of fear?	☐	☐	☐	☐	☐
22. Lack of energy?	☐	☐	☐	☐	☐
23. Trembling when with other people?	☐	☐	☐	☐	☐
24. Anxiety or panic attakcs?	☐	☐	☐	☐	☐

	No	Sometimes	Regularly	Often	Very often or Constantly
During **the past week** did you feel:					
25. Tense?	☐	☐	☐	☐	☐
26. Easily irritated?	☐	☐	☐	☐	☐
27. Frightened?	☐	☐	☐	☐	☐
28. That everything is meaningless?	☐	☐	☐	☐	☐
29. That you just can't do anything anymore?	☐	☐	☐	☐	☐
30. That life is not worth while	☐	☐	☐	☐	☐
31. That you can longer take any interest in the people and things around you?	☐	☐	☐	☐	☐
32. That you can't cope anymore?	☐	☐	☐	☐	☐
33. That you would be better off if you were dead?	☐	☐	☐	☐	☐
34. That you can't enjoy anything anymore?	☐	☐	☐	☐	☐
35. That there is no escape from your situation?	☐	☐	☐	☐	☐
36. That you can't face it anymore?	☐	☐	☐	☐	☐
During **the past week** did you:					
37. No longer feel like doing anything?	☐	☐	☐	☐	☐
38. Have difficulty in thinking clearly?	☐	☐	☐	☐	☐
39. Have difficulty in getting to sleep?	☐	☐	☐	☐	☐
40. Have any fear of going out of the house alone?	☐	☐	☐	☐	☐
During **the past week:**					
41. Did you easily become emotional?	☐	☐	☐	☐	☐
42. Were you afraid of anything when there was really no need for you to be afraid? (for instance animals, heights, small rooms)	☐	☐	☐	☐	☐
43. Were you afraid to travel on buses, streetcars/ trams, subways or trains?	☐	☐	☐	☐	☐
44. Were you afraid of becoming embarrassed when with other people?	☐	☐	☐	☐	☐
45. Did you feel as if you were being threatened by unknown danger?	☐	☐	☐	☐	☐
46. Did you ever think "I wish I was dead"	☐	☐	☐	☐	☐
47. Did you ever have fleeting images of upsetting event(s) that you have experienced?	☐	☐	☐	☐	☐
48. Did you ever have to put aside thoughts of any upsetting event(s)?	☐	☐	☐	☐	☐
49. Did you have to avoid certain places because they frightened you?	☐	☐	☐	☐	☐
50. Did you have to repeat some actions a couple of times before you could do something else?	☐	☐	☐	☐	☐

Annex 3. Pages 1 & 2 of questionnaires: general data & 4DSQ (22).



Orientation to Life Questionnaire

Answer the following 13 qusestions as accurately as you can. Response should be made with your general attitude in mind, NOT within a certain time frame.
Please read the questions carefully and indicate the answer that most accurately reflects your thoughts by **putting an "X" in the box** under the answer that is most appropriate

1. Do you have the feeling that you don't really care about what goes on around you?
1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐ 7 ☐
Very seldom or never — Very often

2. Has it happned in the past that you were surprised by the behaviour of people whom you thought you knew well?
1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐ 7 ☐
Never happened — Always happens

3. Has it happened that people whom you counted on disappointed you?
1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐ 7 ☐
Never happened — Always happens

4. Until now your life has had:
1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐ 7 ☐
No clear goals or pupose at all — Very clear goals & purpose

5. Do you have the feeling that you are being treated unfairly?
1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐ 7 ☐
Very often — Very seldom or never

6. Do you have the feeling that you are in an unfmailiar situation and don't know what to do?
1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐ 7 ☐
Very often — Very seldom or never

7. Doing the things you do everyday is:
1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐ 7 ☐
A source of deep pleasure & satisfaction — A source of pain and boredom

8. Do you have very mixed-up feelings and ideas?
1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐ 7 ☐
Very often — Very seldom or never

9. Does it happen that you have feelings inside you would rather not feel?
1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐ 7 ☐
Very often — Very seldom or never

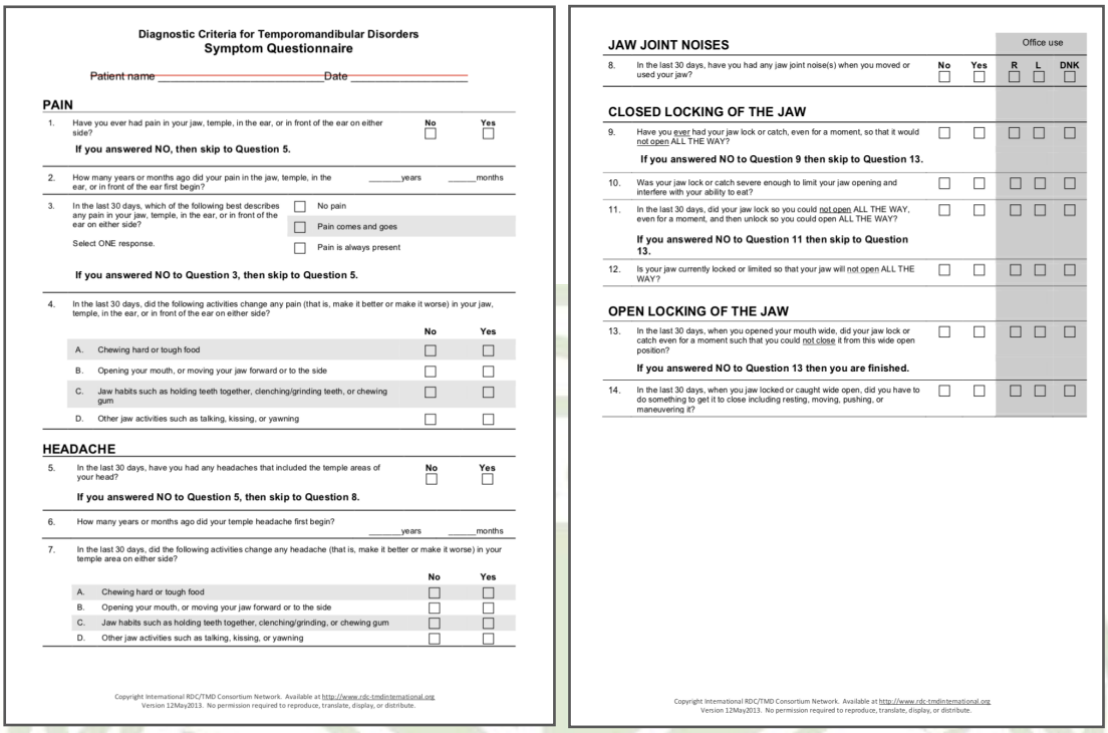
10. Many people- even those who are strong/ resilient- sometimes lose hope in certain situations. How often have you lost hope in the past?
1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐ 7 ☐
Never — Very often

11. When something happened, have you generally found that:
1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐ 7 ☐
You overestimated or underestimated — You saw things in the right proportions

12. How often do you have the feeling that there's little meaning in the things you do in your daily life?
1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐ 7 ☐
Very often — Very seldom or never

13. How often do you have feelings that you're not sure you can keep under control?
1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐ 7 ☐
Very often — Very seldom or never

Annex 4. Pages 3 & 4 of questionnaires: SOC-13 (23).



Diagnostic Criteria for Temporomandibular Disorders
Symptom Questionnaire

Patient name _____ Date _____

PAIN

1. Have you ever had pain in your jaw, temple, in the ear, or in front of the ear on either side? — No ☐ Yes ☐
   If you answered NO, then skip to Question 5.
2. How many years or months ago did your pain in the jaw, temple, in the ear, or in front of the ear first begin? _____years _____months
3. In the last 30 days, which of the following best describes any pain in your jaw, temple, in the ear, or in front of the ear on either side? Select ONE response.
   ☐ No pain
   ☐ Pain comes and goes
   ☐ Pain is always present
   If you answered NO to Question 3, then skip to Question 5.
4. In the last 30 days, did the following activities change any pain (that is, make it better or make it worse) in your jaw, temple, in the ear, or in front of the ear on either side?

		No	Yes
A.	Chewing hard or tough food	☐	☐
B.	Opening your mouth, or moving your jaw forward or to the side	☐	☐
C.	Jaw habits such as holding teeth together, clenching/grinding teeth, or chewing gum	☐	☐
D.	Other jaw activities such as talking, kissing, or yawning	☐	☐

HEADACHE

5. In the last 30 days, have you had any headaches that included the temple areas of your head? — No ☐ Yes ☐
   If you answered NO to Question 5, then skip to Question 8.
6. How many years or months ago did your temple headache first begin? _____years _____months
7. In the last 30 days, did the following activities change any headache (that is, make it better or make it worse) in your temple area on either side?

		No	Yes
A.	Chewing hard or tough food	☐	☐
B.	Opening your mouth, or moving your jaw forward or to the side	☐	☐
C.	Jaw habits such as holding teeth together, clenching/grinding, or chewing gum	☐	☐
D.	Other jaw activities such as talking, kissing, or yawning	☐	☐

JAW JOINT NOISES

		No	Yes	Office use R	L	DNK
8.	In the last 30 days, have you had any jaw joint noise(s) when you moved or used your jaw?	☐	☐	☐	☐	☐

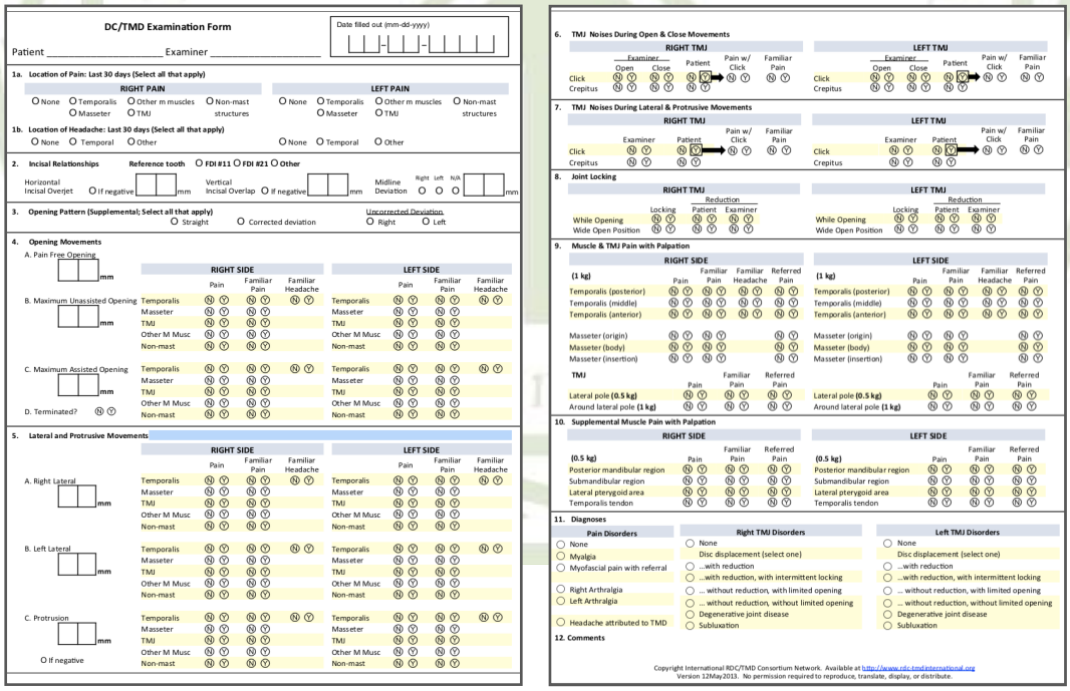
CLOSED LOCKING OF THE JAW

9.	Have you ever had your jaw lock or catch, even for a moment, so that it would not open ALL THE WAY? **If you answered NO to Question 9 then skip to Question 13.**	☐	☐	☐	☐	☐
10.	Was your jaw lock or catch severe enough to limit your jaw opening and interfere with your ability to eat?	☐	☐	☐	☐	☐
11.	In the last 30 days, did your jaw lock so you could not open ALL THE WAY, even for a moment, and then unlock so you could open ALL THE WAY? **If you answered NO to Question 11 then skip to Question 13.**	☐	☐	☐	☐	☐
12.	Is your jaw currently locked or limited so that your jaw will not open ALL THE WAY?	☐	☐	☐	☐	☐

OPEN LOCKING OF THE JAW

13.	In the last 30 days, when you opened your mouth wide, did your jaw lock or catch even for a moment such that you could not close it from this wide open position? **If you answered NO to Question 13 then you are finished.**	☐	☐	☐	☐	☐
14.	In the last 30 days, when you jaw locked or caught wide open, did you have to do something to get it to close including resting, moving, pushing, or maneuvering it?	☐	☐	☐	☐	☐

Annex 5. Pages 5 & 6 of questionnaires: DC/ TMD Symptoms Questionnaire (8).



DC/TMD Examination Form

Patient _____ Examiner _____ Date filled out (mm-dd-yyyy) ☐☐-☐☐-☐☐☐☐

1a. Location of Pain: Last 30 days (Select all that apply)
RIGHT PAIN: O None O Temporalis O Other m muscles O Non-mast structures O Masseter O TMJ
LEFT PAIN: O None O Temporalis O Other m muscles O Non-mast structures O Masseter O TMJ

1b. Location of Headache: Last 30 days (Select all that apply)
O None O Temporal O Other O None O Temporal O Other

2. Incisal Relationships — Reference tooth O FDI #11 O FDI #21 O Other
Horizontal Incisal Overjet O If negative ☐☐ mm — Vertical Incisal Overlap O If negative ☐☐ mm — Midline Deviation Right O Left O N/A O ☐☐ mm

3. Opening Pattern (Supplemental; Select all that apply) O Straight O Corrected deviation — Uncorrected Deviation O Right O Left

4. Opening Movements

		RIGHT SIDE Pain	Familiar Pain	Familiar Headache	LEFT SIDE Pain	Familiar Pain	Familiar Headache
A. Pain Free Opening ☐☐ mm							
B. Maximum Unassisted Opening ☐☐ mm	Temporalis	N Y	N Y	N Y	N Y	N Y	N Y
	Masseter	N Y	N Y		N Y	N Y	
	TMJ	N Y	N Y		N Y	N Y	
	Other M Musc	N Y	N Y		N Y	N Y	
	Non-mast	N Y	N Y		N Y	N Y	
C. Maximum Assisted Opening ☐☐ mm	Temporalis	N Y	N Y	N Y	N Y	N Y	N Y
	Masseter	N Y	N Y		N Y	N Y	
	TMJ	N Y	N Y		N Y	N Y	
	Other M Musc	N Y	N Y		N Y	N Y	
	Non-mast	N Y	N Y		N Y	N Y	
D. Terminated? N Y							

5. Lateral and Protrusive Movements

		RIGHT SIDE Pain	Familiar Pain	Familiar Headache	LEFT SIDE Pain	Familiar Pain	Familiar Headache
A. Right Lateral ☐☐ mm	Temporalis	N Y	N Y	N Y	N Y	N Y	N Y
	Masseter	N Y	N Y		N Y	N Y	
	TMJ	N Y	N Y		N Y	N Y	
	Other M Musc	N Y	N Y		N Y	N Y	
	Non-mast	N Y	N Y		N Y	N Y	
B. Left Lateral ☐☐ mm	Temporalis	N Y	N Y	N Y	N Y	N Y	N Y
	Masseter	N Y	N Y		N Y	N Y	
	TMJ	N Y	N Y		N Y	N Y	
	Other M Musc	N Y	N Y		N Y	N Y	
	Non-mast	N Y	N Y		N Y	N Y	
C. Protrusion ☐☐ mm	Temporalis	N Y	N Y	N Y	N Y	N Y	N Y
	Masseter	N Y	N Y		N Y	N Y	
	TMJ	N Y	N Y		N Y	N Y	
	Other M Musc	N Y	N Y		N Y	N Y	
	Non-mast	N Y	N Y		N Y	N Y	
O If negative							

6. TMJ Noises During Open & Close Movements

	RIGHT TMJ Examiner Open	Close	Patient	Pain w/ Click	Familiar Pain	LEFT TMJ Examiner Open	Close	Patient	Pain w/ Click	Familiar Pain
Click	N Y	N Y	N Y	N Y	N Y	N Y	N Y	N Y	N Y	N Y
Crepitus	N Y	N Y	N Y			N Y	N Y	N Y		

7. TMJ Noises During Lateral & Protrusive Movements

	RIGHT TMJ Examiner	Patient	Pain w/ Click	Familiar Pain	LEFT TMJ Examiner	Patient	Pain w/ Click	Familiar Pain
Click	N Y	N Y	N Y	N Y	N Y	N Y	N Y	N Y
Crepitus	N Y	N Y			N Y	N Y		

8. Joint Locking

	RIGHT TMJ Reduction Locking	Patient	Examiner	LEFT TMJ Reduction Locking	Patient	Examiner
While Opening	N Y	N Y	N Y	N Y	N Y	N Y
Wide Open Position	N Y	N Y	N Y	N Y	N Y	N Y

9. Muscle & TMJ Pain with Palpation

	RIGHT SIDE (1 kg) Pain	Familiar Pain	Familiar Headache	Referred Pain	LEFT SIDE (1 kg) Pain	Familiar Pain	Familiar Headache	Referred Pain
Temporalis (posterior)	N Y	N Y	N Y	N Y	N Y	N Y	N Y	N Y
Temporalis (middle)	N Y	N Y	N Y	N Y	N Y	N Y	N Y	N Y
Temporalis (anterior)	N Y	N Y	N Y	N Y	N Y	N Y	N Y	N Y
Masseter (origin)	N Y	N Y		N Y	N Y	N Y		N Y
Masseter (body)	N Y	N Y		N Y	N Y	N Y		N Y
Masseter (insertion)	N Y	N Y		N Y	N Y	N Y		N Y
TMJ	Pain	Familiar Pain		Referred Pain	Pain	Familiar Pain		Referred Pain
Lateral pole (0.5 kg)	N Y	N Y		N Y	N Y	N Y		N Y
Around lateral pole (1 kg)	N Y	N Y		N Y	N Y	N Y		N Y

10. Supplemental Muscle Pain with Palpation

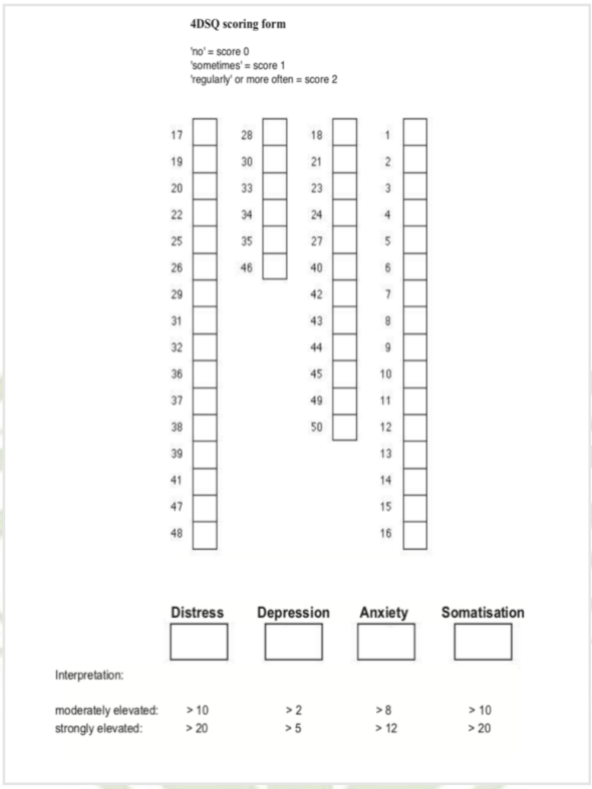
	RIGHT SIDE (0.5 kg) Pain	Familiar Pain	Referred Pain	LEFT SIDE (0.5 kg) Pain	Familiar Pain	Referred Pain
Posterior mandibular region	N Y	N Y	N Y	N Y	N Y	N Y
Submandibular region	N Y	N Y	N Y	N Y	N Y	N Y
Lateral pterygoid area	N Y	N Y	N Y	N Y	N Y	N Y
Temporalis tendon	N Y	N Y	N Y	N Y	N Y	N Y

11. Diagnoses

Pain Disorders	Right TMJ Disorders	Left TMJ Disorders
O None	O None	O None
O Myalgia	Disc displacement (select one)	Disc displacement (select one)
O Myofascial pain with referral	O ...with reduction	O ...with reduction
	O ...with reduction, with intermittent locking	O ...with reduction, with intermittent locking
O Right Arthralgia	O ... without reduction, with limited opening	O ... without reduction, with limited opening
O Left Arthralgia	O ... without reduction, without limited opening	O ... without reduction, without limited opening
	O Degenerative joint disease	O Degenerative joint disease
O Headache attributed to TMD	O Subluxation	O Subluxation

12. Comments

Annex 6. DC/TMD clinical examination form filled out by the investigator (8).



4DSQ scoring form

'no' = score 0
'sometimes' = score 1
'regularly' or more often = score 2

17	28	18	1
19	30	21	2
20	33	23	3
22	34	24	4
25	35	27	5
26	46	40	6
29		42	7
31		43	8
32		44	9
36		45	10
37		49	11
38		50	12
39			13
41			14
47			15
48			16

Distress	Depression	Anxiety	Somatisation

Interpretation:

moderately elevated:	> 10	> 2	> 8	> 10
strongly elevated:	> 20	> 5	> 12	> 20

Annex 7. 4DSQ score form (22).

Universidad Católica de Valencia San Vicente Mártir

INFORME DEL COMITÉ DE ÉTICA DE LA INVESTIGACIÓN DE LA UNIVERSIDAD CATOLICA DE VALENCIA SAN VICENTE MÁRTIR (CEI).

Dra. Mar Aranda Jurado, Secretaria del Comité de Ética de la Investigación de la Universidad Católica de Valencia San Vicente Mártir

Certifico que:

Ha tenido entrada en la reunión del Comité de Ética de la Investigación (CEI) de la Universidad Católica de Valencia San Vicente Mártir en su reunión, de fecha **20 de enero de 2022**, la solicitud de evaluación del proyecto de investigación, relacionado a continuación:

Título: **"Relationship between Psychosocial Factors and TMD in Male and Female Students".**

Código del proyecto: **UCV/2021-2022/058**

Investigador Principal: **Alice Martina Drayne**

El Comité de Ética de la Investigación ha acordado **informar favorablemente el mismo**.

Valencia, 8 de febrero de 2022.

MARIA MAR| ARANDA| JURADO

Firmado digitalmente por MARIA MAR|ARANDA|JURADO
Nombre de reconocimiento (DN): cn=MARIA MAR|ARANDA|JURADO, serialNumber=52648490H, givenName=MARIA MAR, sn=ARANDA JURADO, ou=CIUDADANOS, o=ACCV, c=ES
Fecha: 2022.02.08 12:13:45 +01'00'

Mar Aranda Jurado
Secretaria del Comité de Ética de la Investigación

Annex 8. Certificate of Passing CEI

ID No.	Year study	Sex	Somatisation	Somatisation Range	Distress	Distress Range	Anxiety	Anxiety Range	Depression	Depression Range	SOC	SOC Range	TMD- Pain disorders	TMD- Joint Disorders
1001	5	0	6	0	11	1	2	0	0	0	48	0	1	1
1002	5	0	4	0	2	0	0	0	0	0	75	1	0	0
1003	5	1	12	1	5	0	3	0	1	0	71	1	1	0
1004	5	1	4	0	11	1	6	0	1	0	65	1	0	1
1005	5	0	12	1	8	0	1	0	0	0	64	1	0	1
1006	5	0	18	1	18	1	6	0	2	0	60	0	0	1
1007	5	1	10	0	12	1	4	0	0	0	63	0	1	0
1008	5	1	7	0	20	1	4	0	2	0	51	0	0	0
1009	5	0	12	1	9	0	0	0	0	0	58	0	1	1
1010	5	1	11	1	7	0	0	0	0	0	60	0	1	1
1011	5	1	2	0	1	0	0	0	0	0	63	0	1	0
1012	5	0	23	2	14	1	7	0	1	0	62	0	0	0
1013	5	0	14	1	4	0	0	0	0	0	69	1	1	1
1014	5	0	0	0	1	0	0	0	0	0	73	1	0	1
1015	5	0	13	1	12	1	3	0	0	0	62	0	0	0
1016	3	0	7	0	2	0	1	0	0	0	77	1	1	1
1017	3	0	0	0	1	0	0	0	0	0	85	2	0	0
1018	3	0	0	0	0	0	0	0	0	0	54	0	0	0
1019	3	0	16	1	18	1	8	1	2	0	46	0	1	1
1020	5	0	8	0	8	0	0	0	0	0	59	0	0	1
1022	5	1	5	0	3	0	0	0	0	0	74	1	0	0
1023	5	0	8	0	19	1	11	1	1	0	50	0	1	1
1024	5	1	9	0	10	0	6	0	1	0	61	0	1	0
1025	3	1	0	0	0	0	0	0	0	0	77	1	0	0
1026	3	1	4	0	11	1	2	0	1	0	58	0	0	0
1027	3	1	1	0	3	0	3	0	0	0	70	1	0	1
1028	3	0	7	0	10	0	0	0	0	0	74	1	0	0
1029	3	1	4	0	14	1	12	1	0	0	68	1	0	0
1030	3	1	0	0	0	0	0	0	0	0	87	2	0	1
1031	3	1	1	0	8	0	0	0	0	0	74	1	0	1
1032	3	0	5	0	10	0	0	0	0	0	66	1	0	0
1033	3	1	1	0	2	0	0	0	0	0	74	1	0	0
1034	3	1	0	0	2	0	0	0	0	0	81	2	0	0
1035	4	0	2	0	1	0	0	0	0	0	69	1	0	0
1036	4	0	1	0	2	0	0	0	0	0	82	2	0	0
1037	4	1	1	0	1	0	0	0	0	0	85	2	0	1
1038	4	0	17	1	20	1	6	0	1	0	58	0	1	1
1039	4	1	1	0	9	0	6	0	0	0	63	0	0	1
1040	4	0	2	0	1	0	0	0	0	0	74	1	0	1
1041	4	0	10	0	11	1	1	0	0	0	74	1	0	1
1042	4	1	0	0	0	0	0	0	0	0	83	2	0	1
1043	4	1	6	0	3	0	0	0	0	0	52	0	0	0
1044	4	1	0	0	0	0	0	0	0	0	80	2	0	0
1045	4	1	5	0	11	1	1	0	0	0	59	0	1	0
1046	4	1	20	1	6	0	0	0	0	0	67	1	1	0
1047	4	0	10	0	16	1	2	0	2	0	77	1	1	1
1048	4	0	11	1	18	1	4	0	3	1	62	0	1	1

KEY

Meaning	Document
Male	1
Female	0
TMD-absent	0
TMD- present	1

SOC KEY

LOW	MODERATE	HIGH
13-63	64-79	80-91

4DSQ- KEY

SCALE	LOW	MODERATELY HIGH	V HIGH
Somatisation	0-10	11-20	21-32
Distress	0-10	11-20	21-32
Anxiety	0-7	8-12	13-24
Depression	0-2	3-5	6-12

Annex 9. Data Collected that was Included

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THESIS

Submitted to
UNIVERSIDAD CATÓLICA DE VALENCIA

Facultad de Medicina y Ciencias de la Salud
in Partial Fulfillment of the Requirements of

DEGREE IN DENTISTRY

Valencia, May 2022

Presented by:
Alice Martina Drayne

Tutor: Dr. Carlos Aguilar González

Influence of Psychosocial Factors in the Risk of Developing Temporomandibular Disorders in Dental Students

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INTRODUCTION

It has been suggested that there is a relationship between sex, sense of coherence (SOC), stress manifestations and Temporomandibular disorders (TMD). This study aims to analyse this proposed relationship in dental students, (a group notoriously susceptible to intense stress manifestations and heightened prevalence of TMD) so dentists can better identify at risk populations and implement a biopsychosocial model of care to manage them more appropriately.

OBJECTIVE

- To evaluate the relationship between intensity of stress manifestations, SOC and probability of TMD in a group of dental students in UCV,
- To study the prevalence of TMD within this population and among males and females,
- To compare the level of stress manifestations and SOC in category (males and females) and category TMD (present or absent).

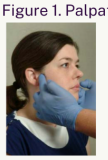
HYPOTHESIS

- There is no relationship between intensity of stress manifestations, SOC and probability of TMD,
- There is no increased prevalence of TMD in students studying dentistry or in females,
- There is no variance in level of stress manifestations and SOC in category (males and females) and category TMD (present or absent).

METHODOLOGY

- 48 dental students attending UCV,
- Questionnaires to evaluate SOC, stress manifestations and TMD symptoms

Figure 1. Palpation of TMJ



- Clinical examination of the temporomandibular joint (TMJ) and diagnosis using the DC/TMD

RESULTS

1. UNIVARIATE LOGISTIC REGRESSION

Table 1: Model Summary TMD- pain disorders

Covariates	Model	Nagelkerke R^2	p-value
Depression	H_1	0.11	0.044*
Anxiety	H_1	0.06	0.158
Distress	H_1	0.16	0.017*
Somatisation	H_1	0.30	< .001*
SOC	H_1	0.20	0.007*

Table 1: shows correlations between TMD-pain disorders and most covariates tested. *(* indicating statistically significant (p < 0.05) result)*

Figure 2: Conditional Estimate Plot showing the variability of TMD-pain disorders as a result of A. Somatisation and B. SOC

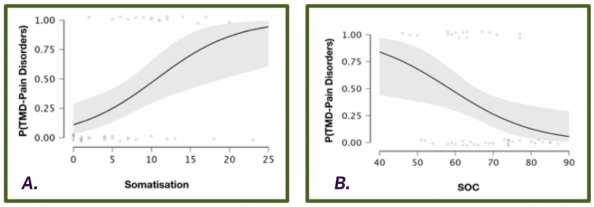


Figure 1 shows somatisation has a positive correlation (A) and SOC has a negative correlation (B) with TMD-pain.

2. PREVALENCE

Figure 3: Donut chart showing prevalence of TMD presence and absence

- 61.70% prevalence of TMD
- No sex predilection for TMD-pain
- Female-to-male ratio of TMD-joint= **1.8:1**

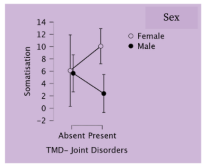
3. ANOVA

Table 2: Sex ∗ TMD- Joint Disorders on Somatisation and SOC

Cases	$F_{[1,43]}$	p
ANOVA- Somatisation	4.56	0.038*
ANOVA- SOC	4.74	0.035*

- *Table 2: Interaction effect between sex and TMD-joint disorders on sex and somatisation. (F= Variation between means)*

Figure 4: Sex ∗ TMD- Joint Disorders on Somatisation



- *Figure 3: post Hoc Bonferroni correction showed a mean difference and 95% CI of 7.69 (1.16 to 14.22), p-value of 0.018 between groups "Females, TMD-Joint present" and "Males TMD-Joint present"*

CONCLUSION

- Relationships between intensity of somatisation, distress, depression, SOC and TMD-pain disorders exist.
- The experience of TMD-pain can perpetuate the onset/intensity of some stress manifestations. Their effect can be buffered by a high SOC.
- Females were more likely than males to have a TMD-joint disorder.
- Males with TMD-joint have a much lower somatisation than females with TMD-joint.
- There is a need for a support system for dental students and a biopsychosocial model of care to manage groups at risk of TMD.

BIBLIOGRAPHY

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