

A pilot study using ultrasound imaging to compare fascial thickness between chronic neck pain and control groups

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Declaration

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This thesis **entitled:** ‘A pilot study using ultrasound imaging to compare fascial thickness between chronic neck pain and control groups’, is submitted in partial fulfillment for the requirements for the Unitec degree of Master of Osteopathy

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Candidate’s declaration

I confirm that:

- This Thesis represents my own work
- The contribution of supervisors and others to this work was consistent with the Unitec Regulations and Policies
- Research for this work has been conducted in accordance with the Unitec Research Ethics

Committee Policy and Procedures, and has fulfilled any requirements set for this project by the Unitec Research Ethics Committee.

Research Ethics Committee Approval Number: 2018 1001

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Introduction to Thesis

Chronic neck pain (CNP) is considered to be one of the most common musculoskeletal complaints and is increasing in prevalence (Ghamkhar & Kahlaee, 2017). The aetiology of CNP is both multifactorial and complex, and includes ergonomic factors (e.g. inadequate postures, inappropriate physical demands, repetitive movements) (Genebra et al., 2017), psychosocial influences (e.g. psychological stress, depression, anxiety, work satisfaction) (Cimmino, Ferrone, & Cutolo, 2011), behavioural elements (e.g. fitness levels and smoking) and individual specific variables (e.g. genome, body mass index, age, and histories of pain complaints), (Genebra et al., 2017; Malchaire et al., 2001). ‘Myofascial’ dysfunction is one aetiological consideration thought to contribute to the development of CNP (Gerwin, 2001). Although the term ‘myofascial pain’ suggests pain related to both muscular and fascial tissue, a substantial proportion of research investigating myofascial dysfunction is focused on the ‘myo’ or muscle aspect (Stecco, Meneghini, Stern, Stecco, & Imamura, 2014).

Findings reported within a pre-clinical study of a randomised controlled trial (RCT) highlight a potential dysfunction of fascia in CNP (Stecco et al., 2014). The pre-clinical study used ultrasound imaging (USI) to obtain thickness measurements of the deep cervical fascia enveloping SCM and middle scalene muscles in healthy individuals (n = 25). The thickness values obtained from the asymptomatic participants were later compared to the CNP group (n = 28), who were involved in the main clinical study which aimed to assess the effect of different fascial treatment modalities on neck disability index scores and pain levels (visual analogue scale of pain) in people with CNP. Within the pre-clinical study, Stecco et al. (2014) reported a statistically significant increase in fascial thickness enveloping the SCM in the CNP group (n = 28) compared with the controls (n = 25). In their study, the observed fascial thickening occurred in the loose connective tissue (LCT) sub-layers of the deep cervical fascia. These layers normally provide lubrication to permit fascial gliding during movement (Roman, Chaudhry, Bukiet, Stecco, & Findley, 2013). Stecco and colleagues speculated that the observed expansion of these LCT layers, which they termed ‘densification’, was due to an increased viscosity of hyaluronic acid (HA), which is a fundamental composite of LCT (Stecco et al., 2014). This is thought to occur as a result of the interaction between lactate and HA (Pavan, Stecco, Stern, & Stecco, 2014; Stecco et al., 2014) that would theoretically occur in over-active muscles. The densification of SCM fascia in CNP are of particular interest given that over-activity

of SCM has previously been linked to CNP (Boudreau & Falla, 2014; Elliott et al., 2014; Falla, Bilenkij, & Jull, 2004a; Falla, Jull, & Hodges, 2004b; Jull, O’Leary, & Falla, 2008).

Stecco et al. (2014) concluded that their observation of thickened fascia encasing SCM in CNP, specifically with a minimum thickness of 0.15cm, may be used to diagnose myofascial CNP (Stecco et al., 2014). It remains unclear whether this observed association is a valid causal association between CNP and densification. Findings of thicker fascia encasing a muscle in people with CNP highlights a potential role that fascia may have in myofascial pain. Further research appears warranted to explore such findings by investigating whether densification may occur in fascia encasing other cervical muscles.

It appears that the only study comparing the deep cervical fascia thickness values in individuals with CNP against asymptomatic controls was conducted by Stecco et al. (2014). However, due to several limitations associated with the reporting of the study, the findings can only be regarded as preliminary in nature. Further validation of Stecco et al.’s. (2014) findings of thickened fascia in CNP is required before additional research can be conducted to investigate densified fascia. For example, one significant limitation within Stecco et al.’s. (2014) study was incomplete reporting of results. This makes it difficult to estimate effect sizes and presents a challenge in designing future studies with sample sizes that confer adequate statistical power. Given this limitation, a pilot study is justified in order to report findings on the fascial thickness in people with and without CNP. The results of such a pilot study would be useful for estimating a between group effect size in planning a large scale study. This would enable the adequate powering of future studies by appropriate sample sizes and offer a potential validation of the findings reported by Stecco et al. (2014). At the time Stecco et al. (2014) was published, there appeared to be no other studies reporting on the reliability of USI as a tool for measuring the deep cervical fascia. Subsequently, USI has been established as a reliable tool for measuring the deep cervical fascia encasing SCM (Harley, 2016), and upper Trapezius (uTrap) (Salavati, Akhbari, Ebrahimi Takamjani, Ezzati, & Haghighatkah, 2017). Similar to SCM, uTrap has been shown to exhibit increased electromyographic (EMG) activity (Nederhand, Hermens, IJzerman, Turk, & Zilvold, 2002; Nederhand, IJzerman, Hermens, Baten, & Zilvold, 2000) in addition to higher amounts of interstitial lactic acid in people with CNP (Rosendal et al., 2004). To date, there are no studies investigating differences in the deep cervical fascial thickness between people with CNP and asymptomatic controls, despite research indicating that USI is reliable for measuring the thickness of the deep cervical fascia.

The aim of the study reported in this thesis was to investigate the thickness of the deep cervical fascia encasing SCM and uTrap in individuals with and without CNP.

The primary aims of this pilot study were:

1. To report and compare the thickness measurements of the deep cervical fascia encasing SCM and uTrap between people with CNP and asymptomatic controls.
2. To estimate an effect size for detecting potential differences in thickness of the deep cervical fascia encasing SCM and uTrap in order to inform the design and planning of future, more definitive, investigations.

Secondary aims were to report issues relating to the feasibility of using USI as an instrument to investigate fascial thickness in people with, and without, CNP.

This thesis is presented in three sections. Section I contains a literature review; Section II contains a manuscript reporting the pilot study; and Section III contains the Appendices including ethics documentation.

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Abbreviations

C7	Seventh cervical vertebrae
CNP	Chronic neck pain
DF	The deep aspect (underlying the muscle) of the superficial lamina of the deep cervical fascia
EMG	Electromyography
HA	Hyaluronic acid
ICC	Intraclass correlation coefficient
IPAQ	International Physical Activity Questionnaire
LCT	Loose connective tissue
MRI	Magnetic resonance imaging
NDI	Neck disability index questionnaire
RCT	Randomised controlled trial
SF	The superficial aspect (overlying the muscle) of the superficial lamina of the deep cervical fascia
SCM	Sternocleidomastoid
T12	The twelfth thoracic vertebrae
USI	Ultrasound imaging
uTrap	Upper trapezius
VAS	Visual analogue scale

Section I – Literature Review

1 Introduction to the literature review

The aim of Section I is to review background research relevant to the hypothesis that ‘densification’ of fascia occurs in individuals with CNP compared with controls. The first part of the literature review will provide an anatomical overview of SCM, trapezius and fascia as these are the tissues of interest in the study reported in Section II. As fascia is the main subject of this thesis, this will be discussed with additional detail including a review of its varying definitions, morphology, and function. The second part of the review focuses on CNP and reviews the salient literature that applies to the potential muscular and fascial components of myofascial pain and the physiology of densification. The third section discusses ultrasound and its utility for measuring the deep cervical fascia; the feasibility of this is a secondary aim of the manuscript. The literature review will conclude with a summary that highlights the rationale for a research project.

2 Sternocleidomastoid, trapezius and the deep cervical fascia; an overview of anatomy, morphology and definitions

Anatomical knowledge is required when using USI. Therefore it is important that this research thesis describes the anatomy of the measured tissues; SCM, trapezius, and the deep cervical fascia. This is to avoid boundary misplacement which, as discussed by Harley (2016) in his thesis, could occur when similar tissues with the same echogenicity are in close proximity. Harley (2016) developed a sonographic protocol for measuring the SCM and established excellent intra-rater reliability when this protocol was used. Nevertheless, the morphological descriptions of cervical fascia appear to vary within research (Guidera, Dawes, Fong, & Stringer, 2014), which could potentially complicate accurate boundary identification. This literature review will discuss the varying descriptions of cervical fascia to highlight its obscure morphology.

For the purposes of this thesis, the literature review will also discuss the various definitions and classifications of fascia. The variety of fascial terminology displays an evolution from traditional to current perspectives regarding its structure and function. However, the various definitions of fascia still lack consensus, increasing the difficulty of correctly interpreting research within the field. The advantages and disadvantages of each definition of fascia will be discussed to both inform the reader and ensure that the current study uses a description best suited to the research objective. The current section is organised in the following order; SCM anatomy, trapezius anatomy and fascia. The section discussing fascia includes a number of subsections; definitions; fascial layer morphology; the functions of fascia.

2.1 Sternocleidomastoid: anatomical considerations

The SCM is a superficial neck muscle that spans from the occipito-temporal region (mastoid) to the manubrium of the sternum (see figure 1) (Bordoni & Varacallo, 2018). As suggested by the name, the SCM muscle originates from two sites of attachment. The tendinous sternal head arises from the superior anterior edge of the manubrium, the muscular clavicular head arises from the superior aspect of the medial third of the clavicle (Saha, Mandal, Chakraborty, & Bandyopadhyay, 2014). Prior to its insertion at the mastoid process of the temporal bone and lateral half of the superior nuchal line, the muscular clavicular head spirals behind the tendinous sternal head merging with its deep fibres to form a single muscle belly (Saha et al., 2014). The SCM has a parallel fibre arrangement and is reported to have a greater thickness and strength in males (Bordoni & Varacallo, 2018).

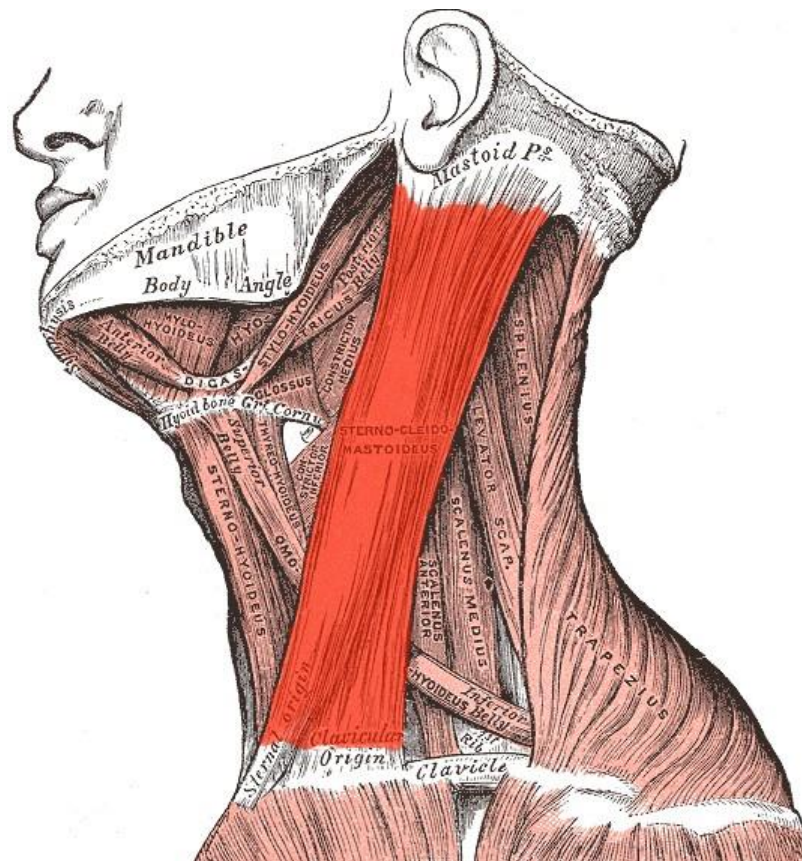


Figure 1: An image of the SCM muscle showing its attachments and relation to the upper trapezius positioned posterolaterally (Gille, 2007).

Unilateral contraction of the SCM produces a combination of three movements, contralateral cervical spine axial rotation, ipsilateral lateroflexion, and cervical spine extension (Saha et al., 2014). Bordoni and Varacallo (2018) explain that the actions produced by bilateral contraction will vary depending on the initial position of the cervical spine and the co-activity of other neck muscles. Assuming a rigid neck fixed by contracted paravertebral muscles, bilateral SCM contraction will result in cervical spine flexion on the thoracic spine (Bordoni & Varacallo, 2018). In instances where paravertebral muscles are not active, bilateral contraction will result in cervical extension. The SCM may also function as an accessory inspiratory muscle by elevating the sternum and clavicle (Bordoni & Varacallo, 2018; Kohan & Wirth, 2014).

A number of physiological variants of SCM have been described (Bordoni & Varacallo, 2018; Kohan & Wirth, 2014). For example, congenital unilateral agenesis (absence), narrowed or widened origins, and multiple muscle heads (in some instances, four) (Kim, Jang, Kim, & Yoon, 2014). Additionally, variations of innervation to SCM have been reported with usual innervation being cutaneous branches from the cervical plexus and motor supply by the accessory nerve (cranial XI). Documented variations include additional

innervation from a branch of the ansa cervicalis (Blythe, Matharu, Reuther, & Brennan, 2015), and an anomalous branch of the facial nerve (Cvetko, 2015; Paraskevas, Lazaridis, Spyridakis, Koutsouflianiotis, & Kitsoulis, 2015).

2.2 Trapezius: anatomical considerations

The trapezius is a superficial trapezoid-shaped muscle that spans the posterior neck, the shoulder and upper thorax (see figure 2). Bilaterally, the paired trapezius forms the shape of a diamond, the lateral aspects being the acromion process of each scapula, the superior aspect at the external occipital protuberances and inferior aspect at T12 (Ourieff & Agarwal, 2018). Both left and right muscles are attached to the medial third of the superior nuchal line, the ligamentum nuchae, the spinous processes of C7 to T12 and the related supraspinous ligaments, the lateral third of the clavicle, the acromion process, and the superior lip of the crest of the scapula (Ourieff & Agarwal, 2018).

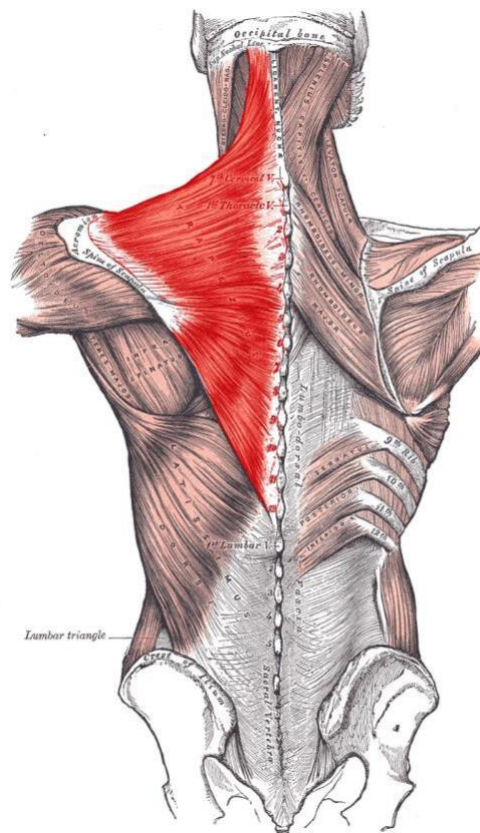


Figure 2: An image showing the attachment points and varying fibre directions of the trapezius muscle (Häggström, 2014).

The trapezius has three functional regions (upper, middle and lower) (Wallden, 2014). These regions are described by fibre orientation as distinct to sites of attachments. The upper trapezius (uTrap) fibres descend from the occiput to the lateral aspect of the clavicle, and are responsible for shoulder elevation and superior rotation of the scapula (using the glenoid as a reference) (Wallden, 2014). The middle region refers to the horizontal fibres that span between the upper thoracic spine and the medial border of the scapula enabling scapular retraction. The lower region describes the ascending fibres that inferiorly rotate the scapula laterally and depress the shoulder (Ourieff & Agarwal, 2018).

The motor innervation of the muscle, like SCM, is provided by the spinal accessory nerve (cranial XI), with sensory innervation arising from the ventral rami of C3 and C4 (Ourieff & Agarwal, 2018). As with SCM, modern imaging has revealed significant anatomical variations in trapezius. Documented variations include an absence of the muscle either in part or entirely, accessory parts of the muscle and variations in nerve supply (Noussios, 2014).

2.3 Fascia – an overview highlighting variable definitions, anatomical and histological descriptions and functions

2.3.1 Defining fascia – a review of various definitions, highlighting the pitfalls and advantages of each.

The term ‘fascia’ is widely used, yet definitions tend to be confusingly variable and ambiguous in the literature (Adstrum, Hedley, Schleip, Stecco, & Yucesoy, 2017; Langevin, 2014; Mirkin, 2008). Tozzi, (2014) critiques traditional definitions of fascia and argues they are based on cadaveric studies where connective tissue is often dissected and separated causing fascia to be viewed as a separate entity. Fascial definitions of this segregated nature are evidenced by terminology such as ‘thoracolumbar fascia’, ‘crural fascia’ and ‘antebrachial fascia’. Although these regional classifications of fascia are useful for specific anatomical reference, the terminology fails to represent the body-wide continuity and function of fascia (Findley & Shalwala, 2013; Tozzi, 2014).

In contrast to the segregated nomenclature of fascia, the International Fascia Research Congress defined fascia as the “soft tissue component of the connective tissue system that

permeates the human body” and goes on to describe fascia as, “all fibrous connective tissues, including aponeuroses, ligaments, tendons, retinaculae, joint capsules, organ and vessel tunics, the epineurium, the meninges, the periosteal, and all the endomysial and intermuscular fibres of the myofasciae.” (Findley, 2009). Although this current definition recognises the continuous body-wide architecture of fascia, due to the broad nature of, and many structures encompassed by this definition, it lacks specificity and therefore inevitably causes difficulty for interpretation of research.

Stecco and Schleip (2016) explain that the variability in definitions occurs because fascia is primarily perceived in two ways: functional, and morphological (Adstrum et al., 2017).

Adstrum et al. (2017) suggested that definitions of fascia must be selected that are appropriate to the research subject and recommend that research investigating functional properties of fascia, the term ‘fascial system’ is used, which includes:

“adipose tissue, adventitia, neurovascular sheaths, aponeuroses, deep and superficial fasciae, dermis, epineurium, joint capsules, ligaments, membranes, meninges, myofascial expansions, periosteal, retinacula, septa, tendons (including endotendon/peritendon/ epitendon/paratendon), visceral fasciae, and all the intramuscular and intermuscular connective tissues, including endomysium/perimysium/epimysium”

Comparatively, histological and morphological descriptions define fascia as, “*sheet, or any other dissectible aggregations of connective tissue that forms beneath the skin to attach, enclose, and separate muscles and other internal organs*”(Adstrum et al., 2017). The present study aims to investigate the thickness of fascia, therefore this latter definition will be adopted for the investigation reported in this thesis.

2.3.2 Anatomical and histological considerations of Fascia

Given the substantial variation in fascial purpose and structure, the classification pedagogy of fascia is a developing field. As with most regions in the body, fascia in the neck has been described as possessing ‘superficial’ and ‘deep’ layers. The following section will first outline the general histology and anatomy, followed by more specific anatomy relating to fascia in the neck. This is of particular importance to the research project because the study

reported in this thesis aims to measure the thickness of the superficial lamina of the deep cervical fascia.

Superficial Fascia:

The superficial layer of fascia is a fibrous sheet of connective tissue composed of loosely packed interwoven collagen fibres mixed with elastic fibres (Stecco, Macchi, Porzionato, Duparc, & De Caro, 2011a). It lies in the subcutaneous space dividing superficial and deep layers of adipose tissue which are dissected by fibrous septa attaching from the superficial fascia to the overlying dermis and to the underlying deep fascia (Stecco et al., 2014).

Deep fascia:

The term ‘deep fascia’ describes a dense fibrous layer of connective tissue that spans through the body forming a network which envelops muscles and forms sheaths for the passage of vessels (Stecco et al., 2011a). While there appears to be a smaller number of histological studies of fascia, it has been found to contain primarily Type I collagen (Casanova, Trindade, & Trindade, 2009). Benetazzo et al., (2011) have shown that in the deep crural fascia, collagen accounted for 18% of total volume with less than 1% being elastin. There appear to be no specific histological studies of the deep cervical fascia *per se*. While more general histological descriptions of the histology of fascia do exist in the literature, it is acknowledged that fascial histology is dependent upon and reflective of specialised function (Kumka & Bonar, 2012).

Three dimensional models of the thoracolumbar (a bilaminar layer) and crural fascia, demonstrate the deep fascia to be a multi-layered system comprised of two or three lamina (Benetazzo et al., 2011). These layers are separated by LCT that lubricate and permit gliding between fascial layers during movement. According to Stecco et al., (2011a) this morphology, identified in the deep fascia of the limbs, is also apparent in the neck. These authors’ go on to state that the fascia identified in trapezius resembles a completely different structure (Stecco et al., 2011a). Unfortunately, there is no apparent evidence for this statement. According to previous histological investigations, trapezius, as well as pectoralis major, deltoid, and latissimus dorsi, appear to lack a true epimysial layer encasing the muscle (Stecco et al., 2009). Instead, the deep fascia strongly adheres to the muscle via intramuscular septa acting as a surrogate epimysium (Stecco et al., 2009). Importantly, it is unclear whether

these studies analysed the uTrap located at the neck, previously described by Stecco et al. (2011a) as having a three-layered fascial morphology.

2.3.3 Cervical fascia anatomy

The cervical fascia is typically described in two parts: the superficial, and the deep cervical fascia. Throughout the available literature, anatomical descriptions vary and the exact fascial morphology does not appear well established.

Superficial Cervical Fascia:

As the name suggests, the superficial cervical fascia describes the most superficial layer of fascia in the neck. Variable descriptions describe the attachments and morphology of the superficial cervical fascia (Guidera et al., 2014). These descriptions describe the superficial fascia as a thin continuous sheet spanning from the head, to the neck, thorax and axilla, whereas additional sources report this layer to be indistinguishable from subcutaneous adipose tissue (Guidera et al., 2014; Linder, 1986; Paonessa & Goldstein, 1976). In the neck, fascia is described as having a loose arrangement to facilitate movement (Guidera et al., 2014).

The Deep Cervical Fascia:

The deep cervical fascia is subdivided into three distinct sections (Guidera et al., 2014). The superficial lamina of the deep fascia is described as surrounding the entire neck enveloping SCM and uTrap. The middle lamina lies beneath the superficial layer surrounding the trachea and the omohyoid muscles. The deep lamina refers to the deepest layer of fascia encasing the vertebral column. The study reported in this thesis (Section 2) involves measuring the thickness of the superficial lamina of the deep cervical fascia, however morphological descriptions are highly variable, which may make the sonographic measurement of the thickness difficult. Therefore the following subsection will discuss the superficial lamina of the deep cervical fascia and highlight the variability in morphological descriptions.

The superficial lamina of the deep cervical fascia:

The superficial lamina of the deep fascia has been described as encasing the entire neck beneath the skin. However, studies report that in the posterior cervical triangle, the superficial lamina may be incomplete or indistinguishable from the surrounding LCT (Nash, Nicholson, & Zhang, 2005; Zhang & Lee, 2002). Natale and Stecco (2015) conducted a literature review with the intention of establishing a definition for the cervical fascia and concluded that despite the reported absence of fascia between SCM and uTrap (Nash, Nicholson & Zhang, 2005; Zhang & Lee, 2002), there appeared limited evidence suggesting that this observation represented the wider population (Natale et al., 2015). There are additional discrepancies around the morphological relationship between the middle and superficial lamina of the deep cervical fascia. Guidera et al. (2014) cite various dissection studies and anatomical reviews, reporting that the superficial and middle lamina may be fused at different regions, namely: where the middle lamina covers thyrohyoid and sternohyoid (Grodinsky & Holyoke, 1938), at the buccopharyngeal fascia (Kostrubala, 1945), at the hyoid and along the superolateral border of the anterior aspect of omohyoid, and then continuing along the posterior aspect to the posterior triangle (Guidera et al., 2014). The anatomical boundaries of the cranial aspect of the superficial lamina are also debated. There are claims that it attaches to the superior temporal line (Sinnatamby, 2011; Standring, 2005), or that it extends into the aponeurotic layer of fascia in the scalp (Kostrubala, 1945). In contradistinction, some authors describe it to be limited superiorly at the zygoma (Berkovitz & Moxham, 1988; Standring, 2005).

2.3.4 Functional considerations of fascia – a review outlining the proposed function of fascia with an aim of providing insight into the potential implications of densification

Tensegrity:

Traditional perspectives on the function of fascia commonly refer to its anatomical relationship with muscle, of a role that encases and provides a surrounding structure to muscles and their fibres (Benjamin, 2009; Klingler, Velders, Hoppe, Pedro, & Schleip, 2014). However, these traditional perspectives do not necessarily reflect the body-wide continuity of fascia. More recently, a model of tensegrity has been used to highlight the functional considerations of fascial architecture (Bordoni, Lintonbon, & Morabito, 2018; Swanson, 2013). Tensegrity is an architectural model, first described by Buckminster Fuller, that can be used to describe how a perfect balance between tension and compression

in the human body results in stability (Swanson, 2013). Using the ‘lens’ of tensegrity, fascia has been described as a body-wide web of tensioned tissue suspended by bones under compression (Scarr, 2011).

Fascia contributes to the tensional solution required by tensegrity and may possess a large role in allowing the body to function as a self-stabilising structure (Scarr, 2011; Swanson, 2013). The model of fascial densification proposes a structural thickening of fascia which reportedly causes increased stiffness (Stecco et al., 2014). It is not unreasonable to theorise that a change to fascial architecture and its tensional elements, could affect the tensional contributions of fascia and disturb the bodies tension/ compression balance that permits tensegrity. The inevitable question arises from this, whether fascial change leads or lags functional change. It is hoped this research may also add light to this question.

Force transmission:

Conventionally, force transmission from muscle contraction was thought to be transmitted sequentially from aponeurosis, to the tendon, terminating at the enthesis and bone (Bojsen-Møller, Schwartz, Kalliokoski, Finni, & Magnusson, 2010). More recent evidence suggests that transmission of contractile forces may also occur in absence of myotendinous connections. Huijing and colleagues utilised magnetic resonance imaging (MRI), to conduct an *in vivo* analysis of myofascial force transmission and reported muscular strain in the lower extremity following passively induced knee movements (Huijing, Yaman, Ozturk, & Yucesoy, 2011). Of particular interest, strain was evident in the soleus, a muscle that does not cross the knee joint, which appears to offer support for theories of non-myotendinous force transmission (Huijing et al., 2011). Findley, Chaudhry and Dhar (2015) postulate a radially directed transmission of force from muscle to its surrounding fascia. While it appears understandable that a contracted muscle might stretch its surrounding fascia, a recent review explains that the anatomical continuity of fascia may imply a larger effect (Wilke, Schleip, Yucesoy, & Banzer, 2018). Wilke et al, (2018) explain that if fascia surrounding muscle has a role in force transmission, by anatomical connections it is plausible that this transmits to deeper fascia such as intramuscular fascial tissue including endomysium, perimysium and epimysium. Additionally, these fascial structures are continuous with collagen reinforced tissue such as intermuscular septa and interosseous membranes (Wilke et al., 2018). A similar phenomenon is described as the posterior hydraulic amplifying mechanism (Macintosh,

Bogduk, & Gracovetski, 1987) and is seen in the epaxial muscles of the thoracolumbar spine, wrapped as they are by the posterior and middle layers of the thoracolumbar fascia (Norris, 1995).

The magnitude of force transmission and contribution of myofascial pathways is believed to be dependent on the properties of myofascial tissue (Bernabei, van Dieën, & Maas, 2016; Zügel et al., 2018). Stiffer or excessively compliant myofascial tissues have been shown to affect the magnitude of force transmission between muscles (Zügel et al., 2018). However, findings were documented in pathological states of tissue such as Ehlers Danlos syndrome (in mice) (Huijing et al., 2010) and in human muscle in states of spastic paresis (Yucesoy & Huijing, 2007). The state of myofascial tissue related to Ehlers Danlos syndrome and spastic paresis cannot be strictly compared to the theory of densification due to the differences in pathology and the exploratory nature of densification. Nevertheless, these findings suggest that alterations in fascia affect force transmission. Therefore, it is possible that densification could affect the functional capacity of force transmission in fascia.

Viscoelasticity:

Evidence has shown that the deep fascia can demonstrate viscoelastic behaviours (Stecco, 2015; Yahia, Pigeon, & DesRosiers, 1993). Yahia and colleagues investigated the viscoelastic properties of whole sheets of thoracolumbar fascia from three human cadavers by assessing the effect of an induced stretch (Yahia et al., 1993). Findings included an increased stiffness of the thoracolumbar fascia following an induced stretch. The authors termed this finding the strain-hardening theory. On the basis that myofibroblasts are present within fascia, they hypothesised that the observed stiffening was due to a myofibroblastic contraction (Yahia et al., 1993). On the other hand Schleip et al. (2012) demonstrated an *in vitro* increased stiffness and a fluid-induced thickening in thoracolumbar fascia following an isometric stretch and suggest that their findings provide an alternative cellular basis for Yahia et al's. (1993) strain-hardening hypothesis, arguing that an increase in matrix fluid content causes fascial stiffening. The study by Schleip et al. (2012) is not without its limitations. For example, the fascia used to measure stiffness was taken from rodents (n=16) and compared with findings of increased tissue hydration in the thoracolumbar fascia of pigs (n=24).

Proprioception:

Fascia has been described as having a potential role in proprioception due to the presence of proprioceptors within its tissue (Yahia, Rhalmi, Newman, & Isler, 1992). Yahia et al. (1992) demonstrated the presence of free nerve endings and mechano-receptors within the tensor fascia lata, and postulated that the fascia may have neurosensory functions. Other connective tissues such as ligamentous and fibrous tissue have demonstrated the presence of mechanoreceptors as well as an established role in proprioception (Proske & Gandevia, 2012; Relph, Herrington, & Tyson, 2013). Therefore, it appears plausible that fascia may contribute to proprioception and that fascial stiffness or thickening could influence this.

However, research has yet to confirm and quantify the extent to which fascia may contribute to proprioception.

3 An introduction to chronic neck pain

Myofascial dysfunction is one factor involved in the aetiology of CNP (Dommerholt, Chou, Finnegan, & Hooks, 2017). A significant body of myofascial CNP research focuses on myofascial pain syndrome a condition characterised by the presence of trigger points (Dommerholt et al., 2017). Despite this, myofascial pain syndrome has poor diagnostic validity due to the absence of an objective diagnostic criteria (Quintner, Bove, & Cohen, 2015). Yet, Stecco and her colleagues use the terms myofascial pain syndrome and myofascial pain interchangeably (Stecco et al., 2014). Due to the contentious nature of myofascial pain syndrome as a diagnosis and the seemingly synonymous use of the two terms, the following section will highlight distinctions between myofascial pain syndrome and myofascial pain. Literature investigating myofascial pain appears to focus largely on muscle dysfunction opposed to fascia as a potential source of CNP. Although fascial dysfunction is a main subject within this thesis, research investigating cervical muscle activity in CNP may coincide with the proposed pathogenesis of the observed densification in CNP. Therefore, the following section will include a subheading involving a review of studies investigating the EMG of uTrap and SCM in states of CNP.

Chronic neck pain has recently been associated with the theory of fascial ‘densification’ (Stecco et al., 2014). Based on sonographic findings including that of thickened fascia overlying the SCM, Stecco et al. (2014) conjectured that densification of fascia is diagnostic of myofascial related CNP. A review of fascial dysfunction will be provided in the section below. This will be subdivided into two main sections describing the biology of fibrosis and densification. Whilst densification is the main topic in the research thesis, Stecco et al. (2014) discuss that it is important to make a distinction between the two processes. Densification is a novel theory with little known about aetiological factors, so it is important to also provide an overview of factors thought to contribute to thickening seen in fibrosis, as they may also play a role in densification.

3.1 Chronic neck pain – an overview including definition, epidemiology and aetiological factors

Chronic pain is sometimes defined as pain that has exceeded an expected time frame for normal tissue healing (Tompkins, Hobelmann, & Compton, 2017). Chronicity is generally defined as pain lasting beyond 3 months, however, a 6 month time period has also been described (Khosrokiani et al., 2018; Merskey & Bogduk, 1994; Tompkins et al., 2017). Neck pain describes pain perceived as arising from within the anatomical borders of the neck, a region that is “bounded superiorly by the superior nuchal line, inferiorly by the tip of the spinous process of the first thoracic vertebrae and laterally by the lateral borders of the neck” (Merskey & Bogduk, 1994). The Bone and Joint Decade Task Force on Neck Pain elaborated that neck pain can occur “with, or without pain referral into the head trunk or upper limbs” (Guzman et al., 2010; Macdermid, Walton, Bobos, Lomotan, & Carlesso, 2016). Neck pain has been described as one of the major musculoskeletal disorders in adult populations with a worldwide prevalence ranging between 16.7% to 75.1% (Fejer, Kyvik, & Hartvigsen, 2006; Genebra et al., 2017). Approximately 50% of adults will develop neck pain lasting beyond 6 months. The prevalence of one-year incidence in neck pain is higher in women (27.2%) than in men (17.4%) (Cheng, Su, Yen, Lui, & Cheng, 2015; Khosrokiani et al., 2018).

Chronic neck pain is considered to have a complex multifactorial aetiology that includes ergonomic factors (e.g. inadequate postures, strenuous exercise, repetitive movements), psychosocial influences (e.g. increased stress, depression, anxiety, work satisfaction), behavioural elements (e.g. fitness levels and smoking) and individual specific variables (e.g. genome, body mass index, age, and histories of pain complaints), (Cimmino et al., 2011; Genebra et al., 2017; Malchaire et al., 2001). Following the exclusion of pathological conditions, an underlying pathoanatomical cause may be difficult to identify. This has resulted in a substantial growth of research into CNP, a large proportion of which focuses on muscular dysfunction (Blomgren, Strandell, Jull, Vikman, & Røijezon, 2018; Dommerholt et al., 2017; Jull & Falla, 2016)

3.2 Myofascial pain: distinctions between myofascial pain and myofascial pain syndrome

Myofascial pain is a term that describes pain relating to muscle and fascial tissue and as a topic, occupies a large proportion of CNP literature (Dommerholt et al., 2017). However, similar to fascial definitions, the interpretation of research appears complicated by synonymous use of myofascial pain and myofascial pain syndrome. For example, these terms are used interchangeably by Stecco et al. (2014), so it is important to outline the distinctions between these two conditions.

Myofascial pain syndrome has been described as a sub-category of musculoskeletal pain, and despite the absence of an objective diagnostic criteria, it is reported that the condition is prevalent in 85% of people with chronic pain (Adigozali, Shadmehr, Ebrahimi, Rezasoltani, & Naderi, 2017; Maher, Hayes, & Shinohara, 2013; Müller, Aranha, & Gavião, 2015; Simons, 1996; Tekin et al., 2013). Myofascial pain syndrome is a clinically popular concept that proposes the presence of local nociceptive trigger points in muscle and fascia, or ‘myofascial’ tissue (Quintner et al., 2015; Simons, 1996). The described signs and symptoms of myofascial pain syndrome mirror those associated with CNP and include local and referred pain, hyperalgesia, reduced range of motion, increased muscle tension and weakness (Adigozali et al., 2017; Müller et al., 2015).

Despite substantial research interest, the diagnosis of myofascial pain syndrome remains a topic of contentious debate (Dommerholt et al., 2017; Quintner et al., 2015). Quintner et al. (2015) conducted a critical review and highlighted that myofascial pain syndrome lacked a reliable diagnostic criteria coupled with an absence of compelling objective evidence supporting the existence of myofascial trigger points. Whilst Quintner et al. (2015) acknowledge the plausibility of trigger points, they contest the validity of myofascial pain syndrome as a diagnosis due to a lack of reliable objective evidence demonstrating the existence of a trigger point. To date, there appear to have been no studies that provide incontestable evidence proving the existence of a trigger point. The current thesis acknowledges the putative distinctions between the two conditions of myofascial pain and myofascial pain syndrome. Nevertheless, it is difficult to dismiss outright a large body of research

investigating muscular dysfunction of the neck that persistently refers to myofascial pain syndrome (Dommerholt et al., 2017). Therefore, for the purposes of this review, CNP research involving myofascial pain syndrome will be referred to using the phrase ‘myofascial pain’.

3.3 Myofascial chronic neck pain and implications in the cervical motor system; a possible rationale for densification

In the past 20 years, there has been an increase in the research investigating the effects of CNP on the functionally related muscle groups. (Blomgren, Strandell, Jull, Vikman, & Røijezon, 2018; Jull & Falla, 2016). Cervical musculature are instrumental in providing the neck with stability and mobility (Panjabi, Cholewicki, Nibu, Babat, & Dvorak, 1988). Muscles in the neck can be divided into deep and superficial muscle groups. Superficial neck muscles (erectores spinae, SCM, and upper trapezius) exert more torque than the deep neck stabilisers due to larger cross sectional areas, and the greater distance between attachment sites that gives rise to long lever arms and mechanical moments. The deep neck stabilisers (multifidus, semispinalis cervicis, longus capitis and longus colli) contribute to the control and stabilisation of vertebral motion segments by virtue of their close attachments to the cervical spine and small moment arms (Peterson et al., 2015).

Evidence suggests that in states of CNP, the functional relationship between superficial and deep cervical muscles is altered. Specifically, several studies have shown that in states of CNP, the superficial cervical muscles may exhibit altered levels of activity and recruitment patterns, whereas the deep muscles show reduced activity (Boudreau & Falla, 2014; Elliott et al., 2014; Falla, Bilenkij, & Jull, 2004a; Falla, Jull, & Hodges, 2004b; Jull, O’Leary, & Falla, 2008; Jull & Falla, 2016). Nederhand et al. (2000) used surface EMG to examine activity of uTrap in participants with whiplash associated disorder (n=19) against asymptomatic controls (n=18) during different functional movement tasks. The whiplash group showed increased co-activation of uTrap during physical exercise as well as a decreased ability to relax following the prescribed task. A later study reported similar findings of increased uTrap EMG activity in people with insidious neck pain in addition to whiplash groups (Nederhand et al, 2002). However, a recent systematic review aimed to summarise the results of scapular EMG activity in patients with CNP in comparison with asymptomatic controls (Castelein et

al., 2015). Castelein et al. (2015) examined 25 studies, and concluded that uTrap EMG findings of people with CNP were variable across literature, and that these could depend unsurprisingly, on different functional tasks.

Findings of increased EMG levels in SCM in states of CNP are more consistent across literature than those investigating uTrap. Early work from Falla et al. (2004a) documented increased SCM EMG signal in individuals with CNP during a cranio-cervical flexion test. The same authors also identified an under-activity of the deep cervical flexors (longus colli and longus capitis) in participants with CNP (Falla et al., 2004b). It was postulated that the observed overactivity of the superficial cervical muscles was to compensate for the impaired stabilising function typically carried out by the inhibited deep cervical flexors (Blomgren et al., 2018; Boudreau & Falla, 2014; Falla et al., 2004b; Jull & Falla, 2016).

Theories of fascial densification, which will be discussed in sections below, involve lactic acid accumulation causing structural changes in the LCT (Stecco et al., 2011b). Hence, when considering findings of altered EMG in states of CNP, the existence and pathology of densification appears plausible. Rosendale et al. (2004) used micro-dialysis to investigate the metabolic status of uTrap in people with chronic work-related myalgia and asymptomatic controls. Their findings showed higher levels of interstitial muscle pyruvate (180 ± 15 vs. 135 ± 12 $\mu\text{mol/l}$) and lactate (4.4 ± 0.3 vs. 3.1 ± 0.3 mmol/l) in participants with myalgia compared with controls ($P < 0.001$) (Rosendal et al., 2004). These findings are further supported by similar studies in CNP populations showing increased pyruvate and lactate in uTrap (Gerdle et al., 2014; Gerdle, Söderberg, Puigvert, Rosendal, & Larsson, 2010).

3.4 Fascial contributions of chronic neck pain

The term ‘myofascial’ pain suggests pain relating to muscular and fascial tissue although research in this field seems to focus almost entirely on muscular dysfunction. While there appears limited research investigating fascial dysfunction as a contributor to myofascial pain (Stecco, Gesi, Stecco, & Stern, 2013), fibrosis and densification are identified as two possible pathologies that may affect fascia. Whilst little is known about the aetiology of densification, diabetes, hormones and age are thought to affect fascia and contribute to fibrosis and therefore will be discussed in the fibrosis section below.

3.4.1 Fibrosis

Fibrosis refers to changes in the collagenous component of fascial architecture (Pavan et al., 2014). The involved mechanisms are similar to scarring, where reparative processes occur with excessive collagen deposition (Pavan et al., 2014). This may result from an abnormal healing response following injury or pathology.

Fascia is a tissue that has a high capacity for regeneration, that appears strongly dependent on mechanical loading (Zullo et al., 2017). When fascia is injured, an inflammatory response occurs followed by regenerative phases involving proliferation and remodelling (Klingler, Velders, Hoppe, Pedro, & Schleip, 2014). During these processes, new collagen fibres aggregate and orientate in directions of mechanical load to ensure a better functional response. Immobilisation will prompt disorganised collagen deposition and result in a decreased ability to withstand mechanical loads whilst rehabilitative loading may achieve adequate fibre alignment (Kinger et al., 2014).

Aging, Diabetes and hormones; potential influencers of fascial fibrosis and dysfunction

Aging has also been associated with reduced flexibility and increased stiffness in connective tissue. Trinade et al. (2012) conducted an experimental cadaveric study of deep temporal fascia samples with the intention of reporting findings that might inform temporomandibular joint dysfunction. Trinade and colleagues found higher levels of stiffness and significantly higher stress-strain values in older individuals compared with a younger group (Trindade et al., 2012). Wojtysiak (2013) studied porcine connective tissue, which is a common analogue for human tissue used by research, in the longissimus lumborum muscle. Findings included a wavy disposition of collagen fibres in new-borns and a denser, regular arrangement of collagen in older ages (Wojtysiak, 2013). Overall, fascia in older age groups appeared to exhibit less elasticity and higher levels of stiffness that could potentially predispose to injury.

Multiple studies have identified a relationship between diabetes mellitus and named conditions involving fascial fibrosis such as Dupuytren's contracture (Broekstra, Groen, Molenkamp, Weker, & van den Heuvel, 2018; Johnson, Pavano, & Rodner, 2018), however there appears to be no clear evidence of the pathophysiology. Whilst underlying mechanisms

are yet to be identified it is thought to involve non-enzymatic glycation of proteins giving rise to abnormal cross link formation (Pavan et al., 2014).

Hormonal factors, specifically oestrogen are also thought to contribute to fascial fibrosis. States of oestrogen deficiency and use of the oral contraceptive pill have been associated with fascial dysfunction and decreased connective tissue elasticity (Beeson, 2014; Pavan et al., 2014; Rajasekaran & Finnoff, 2015), although the specific iatrogenesis and physiology relating to this is unknown.

3.4.2 Densification

‘Densification’ when applied to fascia, is a term attributable to Stecco et al. (2014) that describes a thickening of LCT sublayers of fascia (Pavan et al., 2014). Stecco et al. (2014) postulates that LCT thickening occurs as a result of an accumulation and acidification of HA. Hyaluronin is a glycosaminoglycan monomer that is highly prevalent in the LCT of deep fascia (Cowman, Schmidt, Raghavan, & Stecco, 2015; Roman, Chaudhry, Bukiet, Stecco, & Findley, 2013; Stecco et al., 2011b). While it usually functions as a lubricant for musculoskeletal structures, HA-rich solutions have potential to be highly viscous and demonstrate characteristics of non-Newtonian fluids (Cowman et al., 2015; Morley & Traum, 2016). Changes in molecular weight and decrease pH can lead to an increase in HA viscosity, which is thought to diminish the sliding capacity of the deep fascia (Cowman et al., 2015). This is thought to occur for two potential reasons, firstly, an accumulation of lactic acid reacting with HA; and secondly, an increased production of HA. Stecco et al., (2011b) explain the LCT can accumulate waste products from surrounding tissues and on this basis suggest excessive lactic acid could interact with HA causing viscosity and densified fascia. Considering that individuals with uTrap myalgia have been demonstrated to show higher levels of lactic acid at rest (Rosendal et al., 2004), the theory of densified fascia in people with CNP is logical. Whilst an increased production of HA is thought to occur to improve lubrication (Stecco et al., 2011b), Matteini et al., (2009) observed that HA in high concentrations (obtained from bacteria and tested in physiological saline) behaves as a non-newton fluid and increases in viscosities.

Stecco and her colleagues state that a thickness value of 0.15cm (measured using USI) of the deep cervical fascia encasing SCM can help aid in the diagnosis densification and myofascial neck pain (Stecco et al., 2014). These findings arose from a preclinical study within Stecco et al. (2014) primary study, which assessed the effect of different treatment modalities on the fascial thickness in people with CNP. The findings of thickened fascia offer new ideas into the underlying mechanisms of CNP, although several limitations emerged that hinder the validity of these results. The limitations of the study by Stecco et al. (2014) will be discussed in the following section.

4 Ultrasound imaging

This section is of relevance to the research study as it reviews the validity of USI as a diagnostic tool. Ultrasound is a safe and reliable tool commonly used to analyse soft tissue structures. Three studies have been conducted using USI to measure the thickness of the deep cervical fascia. This section will provide a general overview of USI in relation to diagnostic imaging followed by a critical review of the studies measuring the thickness of the deep cervical fascia.

4.1 Ultrasound imaging – a general overview of its application in musculoskeletal diagnostics

Ultrasound is a non-ionizing, non-invasive, safe and accessible form of diagnostic imaging (Øverås, Myhrvold, Røsok, & Magnesen, 2017). As with other forms of imaging, USI is described as operator dependant therefore its validity and accuracy can depend on the experience and skill of the sonographer (Chiou, Chiou, Chiou, Liu, & Chang, 2003). In New Zealand, USI is typically used to support the diagnosis of pathology as opposed to research or experimental purposes (Nazarian, 2008; Øverås et al., 2017). Ultrasound is becoming more readily available in office practice settings, and is frequently advocated for the speedy, low cost imaging of soft tissues (Chiou et al., 2003; Øverås et al., 2017; Whittaker et al., 2007)

Within clinical musculoskeletal practice, USI has been employed for diagnostic, exploratory and therapeutic purposes (Øverås et al., 2017). There are several different types of USI techniques such as A-mode ('amplitude' mode), B-mode ('brightness' mode), M-mode (time motion mode), Doppler (to assess blood flow), and elastography to assess tissue stiffness (Carovac, Smajlovic, & Junuzovic, 2011). Of particular interest, B-mode is usually advocated for diagnostic imaging particularly of musculoskeletal structures (Whittaker et al., 2007). The transducer generates high-frequency sound waves that propagate through tissue and are reflected back to the transducer. The time interval between pulse generation and detection is used to calculate the depth of the reflector (Bakhru & Schweickert, 2013). Signal processing is used to generate an image of the tissue reflector with the intensity of the reflection

represented by shades of grey (Whittiker et al., 2007). Muscle tissue is comprised of highly organised proteins and cytoplasm and therefore, generates few echoes (i.e. is hypoechoic) compared to connective tissue like fascia. In B-mode, muscle fibres are hypoechoic (dark) between hyperechoic (bright) perimysium, and are therefore visualised as a striated and textured appearance when the transducer is configured longitudinally (Walker, 2004). Comparatively, fascia is highly reflective of sound waves and therefore has a hyperechoic appearance (Stecco et al., 2014; Walker, 2004).

Whilst USI is a useful form of imaging, it is not without limitations (Serafin-Król & Maliborski, 2017) and there are several assumptions made in the process of generating images. Ultrasound assumes that echoes are detected by a straight sound beam, and when this deviates the images are susceptible to contamination by artefacts (Li & Lee, 2015), which can cause incorrect interpretation of images. Anisotropy is an artefact commonly encountered when imaging fibrillar tissues such as tendons and muscles. Anisotropy occurs when sound beams are not perpendicular to the tissue which results in refraction of sound waves away from the transducer, and therefore no echoes are detected and no image is generated (Li & Lee, 2015). On sonographic images anisotropy appears as decreased echogenicity, and in musculoskeletal USI of tendons and muscles is of particular importance because it can mimic tendon tears (Bickle & Morgan, 2019). Altering the angle of the transducer when encountering an apparent ‘tear’ can help to identify anisotropy (Bickle & Morgan, 2019). The quality of USI images can be variable depending on the skill and experience of the sonographer and the quality of the equipment (Chiou et al., 2003). Given these limitations, it is important that in research investigating musculoskeletal structures using USI, an experienced sonographer and good quality equipment are utilised.

4.2 Ultrasound as an instrument for measuring the thickness of the deep cervical fascia; a review of three studies

Ultrasound has been used to investigate and measure the thickness of fascia in a number of body regions (Fede et al., 2018). However, there appears to be only a few studies investigating cervical fascia with USI, with only three apparent studies that have used USI to examine the thickness of the deep cervical fascia (Harley, 2016; Salavati et al., 2017; Stecco

et al., 2014). Each of these will be considered as their findings and methodology may help to inform the design of this study (reported in Section II) as well as future research.

4.2.1 Study one: ultrasonography in myofascial neck pain: randomised clinical trial for diagnosis and follow-up (Stecco et al., 2014)

Stecco et al. (2014) conducted a randomised control trial using USI to assess the effects of fascial manipulation modalities on the thickness of the deep cervical fascia over SCM and middle scalene. This involved a pre-clinical study assessing fascial thickness in asymptomatic individuals ($n = 25$), to later be used as a comparison for thickness values against the CNP group ($n = 28$). Sonographic images were taken in B-mode, using a 38mm linear array probe to measure fascial thickness. Measured tissues included the SCM muscle, and the related superficial and deep layers of the deep cervical fascia, overlying and underlying the muscle. Averages were calculated from three thickness values for each tissue (SCM muscle, superficial layer and deep layer).

Within the study, Stecco and colleagues reported higher SCM mean thickness values in participants with CNP (superficial layer = 0.19cm left, 0.18cm right; deep layer = 0.16cm left, 0.11cm right) compared with asymptomatic controls (superficial layer = 0.11cm bilaterally; deep layer = 0.12cm left, 0.11cm right) (Stecco et al., 2014). The increase in thickness of the superficial layer of SCM cervical fascia was statistically significant between CNP and controls ($P = < 0.05$). The study reported a moderate positive correlation ($r = 0.44$) between CNP groups and increased fascial thickness (> 0.05 cm). They concluded that USI imaging may be used to assist a diagnosis of myofascial disease in CNP and they suggested that a thickness of at least 0.15cm of SCM fascia may be used as a diagnostic value.

Harley (2016) used an online PEDro checklist (Maher, Sherrington, Herbert, Moseley, & Elkins, 2003) to assess the methodological quality of the study by Stecco and colleagues and reported a score of 5/10 explaining that marks were lost due to inconsistent blinding of operators. Although this checklist provides an effective assessment tool, it fails to address limitations that may exist within the preclinical study, which forms a basis for this thesis. Limitations in the preclinical study are primarily related to poor reporting that constrain the

validity of the results. First, the study did not describe the level of experience of the sonographer. It is established that sonography is highly operator dependent, with the quality and measurements known to exhibit high dependency upon the skill and experience of the sonographer (Chiou et al., 2003). Second, it appears unlikely that the asymptomatic sample size was large enough to adequately represent normal thickness values in the deep fascia. The adequacy of the sample size cannot be determined because the study does not report a calculated effect or the data required to independently calculate one. Third, Stecco et al. (2014) state a correlation between CNP, decreased cervical range of motion and increased fascial thickness without reporting a correlation coefficient. Given these limitations, their results need to be treated with caution, in particular the putative relationship between a stated value for fascial thickness and CNP. At the time of this study, there was no research establishing the reliability of USI for the assessment of deep cervical fascia thickness (Harley, 2016). Since this time however, there have been two studies establishing USI as a reliable tool for measuring fascial thickness of uTrap (Salavati et al., 2017) and SCM (Harley, 2016).

4.2.2 Study two: reliability of deep cervical fascia and sternocleidomastoid muscle thickness measurements using ultrasound imaging (Harley, 2016)

Harley (2016) investigated the reliability of deep cervical fascia measurements using USI. A convenience sample of 10 asymptomatic participants, 5 females and 5 males took part in the study. Utilising B-mode, a high frequency (17-5 MHz) ‘hockey stick’ transducer for SCM (muscle and fascial) measurements following a standardised USI protocol developed by the author. Harley (2016) reported moderate to excellent reliability ($ICC = 0.312 - 0.821$) for intra-operator cervical fascia thickness measurements obtained by an experienced sonographer, whereas in contrast, it was shown that the novice (unregistered) operator demonstrated poor reliability.

Sonographic images were taken of SCM at the mid-point between the mastoid process and the medial head of the clavicle (located using a clear plastic ruler). The experienced operator mean thickness values were 0.045cm (superficial layer of deep cervical fascia) and 0.055cm (deep layer of deep cervical fascia). These measurements are approximately half the thickness values observed in Stecco et al’s. (2014) control group. Harley (2016) reported low

to moderate ($ICC = 0.033 - 0.428$) inter-operator reliability for cervical fascia measurements, and acknowledged that this may explain the discrepancy between results, as well as the described difficulties in fascial border identification.

4.2.3 Study three: Reliability of the upper trapezius muscle and fascia thickness and strain ratio measures by ultrasonography and sonoelastography in participants with myofascial pain syndrome (Salavati et al., 2017)

Salavati et al. (2017) appears the only other published study to investigate the reliability of USI in the measurements of deep cervical fascia. In contrast to Harley (2016), Salavati et al. (2017) measured the thickness of cervical fascia enveloping the mid belly of the uTrap, in people with neck pain ($n = 22$) for at least 3 months. Measurements of uTrap were obtained at a mid-point between the spinous process of C7 and the acromion process using a linear 5-14MHz transducer (Salavati et al., 2017). This study concluded USI was a reliable method for measuring uTrap fascial thickness, reporting good to excellent reliability for both within intra-examiner ($ICC = 0.78-0.96$) and between intra-examiner reliability ($ICC = 0.75-0.98$).

Mean thickness values were 0.123 cm and 0.125cm for the first and second operators. Whilst this provides important information about uTrap fascial thickness, no normative thickness values have been reported. Therefore, it is unknown whether uTrap fascia thickens in states of CNP, or whether this finding is exclusive to the SCM and middle scalene reported by Stecco et al. (2014).

5 Summary and statement of research question:

Section I has conducted a review of literature relating to the potential relationship between densified fascia and CNP, with a particular focus on the feasibility of sonographic investigation of cervical fascia thickness. In summary, two studies have reported good reliability using sonographic protocols for measuring the deep cervical fascia encasing uTrap and SCM. Nevertheless, cervical fascial morphology appears to be highly variable, which could potentiate inaccurate measurements or cause difficulty establishing normative values for fascial thickness. The literature also reveals that research into fascial function and dysfunction is a developing field where the majority of the proposed physiology is still unknown due to a lack of supportive evidence in human tissue. Concerning densification, the literature highlights that SCM and uTrap show findings of increased EMG in CNP, which suggests support for the proposed mechanisms of lactic acid accumulation causing a fluid-induced thickening of LCT. While the theory of densification seems plausible, the findings of Stecco et al. (2014) remain exploratory and require validation by future research.

Based on the current literature review, it appears that Stecco et al. (2014) is the only study investigating fascial thickness between CNP and asymptomatic control groups. However, inadequate reporting of results prevent power calculations from informing an appropriate sample size. For example, Stecco et al. (2014) report statistically significant differences between a fascial thickness of CNP and control groups without describing a standard deviation. Without a standard deviation, an effect size to inform a power analysis cannot be undertaken. The concept of densification is apparent in fascial literature with many studies utilising this theory as an established cause of myofascial pain (Fede et al., 2018; Luomala & Pihlman, 2016; Pavan et al., 2014; Shah et al., 2015). Given this, it is essential that densification is researched further, which first requires a pilot study to establish an effect size and to report limitations concerning feasibility.

Having addressed the relevant literature relating to fascial densification and CNP in section I, section II will present the current study. This pilot study utilises USI to measure the thickness of fascia enveloping SCM and uTrap in participants with CNP compared with matched

controls. It is intended to provide results sufficient to estimate an effect size for the difference in thickness of the deep cervical fascia between individuals with CNP and controls, to inform the design and planning of future, more definitive investigations.

6 References

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Section II – Manuscript

The manuscript presented in this section follows the Instruction to Authors for *Surgical and Radiologic Anatomy*, available for viewing here: <http://goo.gl/tnxfVL>. To facilitate thesis marking, referencing is styled using the APA 6th conventions rather than those of the Vancouver style required by the journal.

A pilot study using ultrasound imaging to compare fascial thickness between chronic neck pain and control groups

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Abstract

Background: ‘Densification’, defined as a thickening in the loose connective tissue (LCT) of fascia, is a recently proposed theory that is thought to be associated with chronic neck pain (CNP). Stecco and colleagues reported that individuals with CNP have a greater thickness of deep cervical fascia enveloping the sternocleidomastoid (SCM) when compared to asymptomatic controls. Due to a number of limitations these findings are exploratory and require further validation. Before larger more definitive studies can take place, a pilot study is required to estimate an effect size between groups (symptomatic and control) and inform an appropriate sample size.

Purpose: The current pilot study aims to use ultrasound imaging (USI) to measure the thickness of the deep cervical fascia enveloping SCM and upper Trapezius (uTrap) in controls and people with CNP.

Methods: In each of the 18 participants, pre-established USI protocols were used to measure the fascial thickness of SCM and uTrap in CNP (n = 9) and control (n = 9) groups.

Results: Between groups there were no significant differences in fascial thickness for SCM and uTrap. The effect for detecting differences in fascial thickness between CNP and controls was ‘small’ for SCM and ‘medium’ for uTrap.

Conclusion: Overall, there were no differences in fascial thickness between CNP and controls. Using these results and the identified effect sizes, almost 800 participants would be required to sufficiently power a study examining fascial thickness between people with CNP and asymptomatic controls. This pilot study identified additional limitations, such as variations in fascial morphology, that should be considered by future studies into fascial thickness.

Keywords: Fascia, chronic neck pain, densification, ultrasound imaging

Introduction

Chronic neck pain is a common condition with an increasing prevalence (Ghamkhar & Kahlaee, 2017). Chronic neck pain is thought to have a multifactorial aetiology that includes ergonomic factors (e.g. inadequate postures, inappropriate physical demands, repetitive movements) (Genebra et al., 2017), psychosocial influences (e.g. psychological stress, depression, anxiety, work satisfaction) (Cimmino, Ferrone, & Cutolo, 2011), behavioural elements (e.g. fitness levels and smoking) and individual specific variables (e.g. genome, body mass index, age, and histories of pain complaints), (Genebra et al., 2017; Malchaire et al., 2001). Following the exclusion of known pathological conditions, an underlying pathoanatomical cause may be difficult to identify. This has resulted in a substantial growth of research into CNP, a large proportion of which focuses on muscular dysfunction, often referred to as myofascial CNP (Dommerholt, 2017).

‘Densification’ is a theory recently proposed as having a role in the development of CNP (Stecco, Meneghini, Stern, Stecco, & Imamura, 2014). When applied to fascia, the term ‘densification’ refers to a fluid induced thickening within the LCT sublayers of fascial lamina, which normally provide lubrication to promote fascial gliding during movement (Pavan, Stecco, Stern, & Stecco, 2014). The LCT is highly abundant in hyaluronic acid (HA), which can become more viscous when interacting with lactic acid (Stecco et al., 2014; Stecco et al., 2011b). Stecco et al. (2014) propose that an increase in HA viscosity following interactions with lactic acid is responsible for densification. The LCT acts as an important reservoir for water and salts, and can also accumulate waste products such as lactic acid (Pavan et al., 2014; Stecco, 2015). Based on these proposed mechanisms of densification, this would theoretically occur in overactive muscles. In states of CNP, superficial cervical muscles such as SCM and uTrap are known to exhibit altered or increased electromyography activity (Falla, Bilenkij, & Jull, 2004; Jull & Falla, 2016; Nederhand, Hermens, IJzerman, Turk, & Zilvold, 2002; Nederhand, IJzerman, Hermens, Baten, & Zilvold, 2000). Additionally, individuals with work related uTrap myalgia (lasting at least 3 months) have been shown to have higher interstitial lactate within the uTrap muscle (Rosendal et al., 2004).

Stecco et al. (2014) discuss that the increase in viscosity of HA could explain symptoms of stiffness and decrease cervical range of motion in states of CNP.

The model of densification was proposed following observations of thickened fascia in people with CNP, observed in a pre-clinical study reported within Stecco et al's. (2014) randomised controlled trial (RCT). The pre-clinical study used USI to obtain thickness measurements of the deep cervical fascia enveloping SCM and middle scalene muscles in healthy individuals ($n = 25$) to later be compared to the CNP group ($n = 28$), who were involved in the main clinical study. The main clinical study aimed to assess the effect of different fascial treatment modalities on neck disability index scores and pain levels (using the visual analogue scale of pain) in people with CNP. For the pre-clinical study, B-mode sonographic images were taken using a 38mm linear array probe to measure the thickness of the SCM and middle scalene muscles, and the related deep cervical fascia (including the superficial layer (SF) of fascia overlying the muscle, and the deep layer (DF) underlying the muscle). Stecco et al. (2014) reported higher SCM mean thickness values in values in participants with CNP compared to the control group. Within the study, Stecco et al. (2014) reported a moderate positive correlation between an increase in SCM fascial thickness ($> 0.05\text{cm}$) and people with CNP. Of particular interest, they conclude that a minimum thickness of 0.15cm in the fascia encasing SCM can be used to diagnose myofascial CNP.

The findings of increased thickness suggest that fascia might have a potential role in CNP. Therefore, further research is warranted to explore these findings in fascia encasing other muscles implicated in CNP. However, several limitations within the study render these findings exploratory at best, and they require validation before additional research investigating fascial densification can occur. A notable limitation within Stecco et al's. (2014) study is the inadequate reporting of results which prevents future research from conducting power calculations to inform an appropriately powered sample size. Stecco et al. (2014) is the only study investigating fascial thickness in CNP and asymptomatic controls, meaning that currently, no power analysis can be conducted to inform a sample size. Therefore, a pilot study is required to report findings on the fascial thickness in people with and without CNP. These results will provide appropriate data to estimate an effect size to inform future studies aiming to validate the findings reported by Stecco et al. (2014). During the time of Stecco et al's. (2014) study, there was no research reporting on the reliability of USI as an instrument to measure the thickness of the deep cervical fascia. Recently, two reliability

studies have reported USI to be of acceptable reliability for measuring the deep cervical fascia encasing SCM (Harley, 2016) and uTrap (Salavati et al., 2017) however, a pilot study is yet to be conducted. The experimental aim of this pilot study was to report and compare the sonographic thickness measurements of the deep cervical fascia encasing SCM and uTrap between CNP groups and matched asymptomatic controls.

Methods

Study Design

This pilot study is reported in accord with the CONSORT 2010 checklist for information to include when reporting a pilot or feasibility trial (see Appendix A). The design was a cross-sectional comparison of the deep cervical fascial thickness between people with CNP (n=9) and asymptomatic controls (n=9). The groups were matched for age, sex, height, weight, and physical activity levels. Following pilot trial commencement, there were no changes to eligibility criteria. This study was approved by the institutional ethics committee (UREC Approval 2018-1001) (see Appendix B for ethics approval).

Participants

Participant recruitment took place between August 2018 until October 2018, and participants were invited to a single sonographic scanning session towards the end of November.

Initially, 28 individuals for the CNP group (n=18) and asymptomatic group (n=10) were recruited by word of mouth, and through an online newsletter (see Appendix C) emailed to all academic and administrative staff of a tertiary education institution. Individuals responding to the advertisements were provided with information sheets to explain the study requirements. The primary researcher engaged in conversations over email and in person with each participant to screen for eligibility. Following this screen, 9 CNP participants and 1 asymptomatic control were excluded from the study due to failing to meet the eligibility criteria. All participants provided written informed consent (see Appendix D and E for participant information sheets and consent forms). Recruited participants who were eligible to take part in the study attended a single sonography scanning session at Sound Experience Ltd, an independent private sonography practice located in Auckland, New Zealand. Participants were eligible if they fulfilled the following criteria:

Inclusion criteria:

All Participants:

- Over 18 years of age

- Under 55 years of age, due to greater risk of the onset of neck arthritis from this age
- Must be able to read and understand an information sheet in English language about the study and sign a consent form
- Agree to complete a neck disability index questionnaire (NDI) (see Appendix F for NDI)

People with CNP:

- Score of 5 or above in the NDI (a score of 5 indicates mild neck pain)
- CNP for most days for at least the previous 3 months
- CNP intensity of at least 20mm on a 100mm visual analogue scale (VAS) (see Appendix F for questionnaire including VAS) on most days over the past 3 months.

People without neck pain (control group):

- No neck pain lasting over 1 month, or recurrent neck pain with episodes adding to greater than 1 month for at least 2 years (Acute neck pain has a high prevalence and typically resolves within days to weeks, whereas CNP is defined by lasting longer than 3 months) (Merskey & Bogduk, 1994).
- Score between 0-4 in the NDI.

Exclusion Criteria:

CNP (symptomatic group):

- Experiencing chronic/ and or acute neck pain due to a known cause such as disk pathology, neural compromise, congenital deformity of the spine, arthritis, neoplasm, inflammatory rheumatoid disease
- Experiencing neck pain for a duration of less than 3 months.
- Suffering from neurological symptoms such as, sensory abnormalities, weakness, altered reflexes.
- Symptoms related to a motor vehicle accident or trauma within the last 6 months.
- A history of chronic headaches or migraines.

People without neck pain (control group):

- Neck pain lasting more than 1 month in the past year
- A current episode of neck pain

Following recruitment of the CNP group, participants for the control group were recruited and matched for age, sex, height, weight, and physical activity levels scores in the short form International Physical Activity Questionnaire (IPAQ). A pre-determined age for matching was within a 2 year age difference. Participants were also required to complete a questionnaire (see Appendix F) designed by the primary researcher which included the NDI, the IPAQ, a VAS of pain, and descriptive questions relating to their neck pain.

Sonographer

A registered sonographer with greater than 20 years experience in musculoskeletal sonography undertook all USI measures. Previously, the sonographer had participated as an operator in Harley's (2016) reliability study, and had demonstrated moderate ($ICC = 0.357$) to excellent ($ICC = 0.821$) reliability in intra-operator reliability for thickness measurements of the deep cervical fascia across two follow-up sessions.

Procedures:

All images were captured using a 2014 GE Logiq USI machine (GE Healthcare, Waukesha, Washington) in B-mode using a 'hockey stick' transducer at 16.0 MHz. Pre-established USI protocols for measuring SCM fascia (Harley, 2016) and uTrap fascia (Salavati, Akhbari, Ebrahimi Takamjani, Ezzati, & Haghighatkhah, 2017) were used. Three scanning sessions (a total of six hours) were prescribed to familiarise the sonographer with procedures and recommend protocol modifications in response to issues encountered in the preliminary practise sessions.

The following section describes the imaging protocols designed by Harley (2016) and Salavati et al. (2017) (see Appendix G).

The primary researcher was present during data collection to ensure that the sonographer complied to the protocols outlined in the appendix. Prior to imaging, the primary researcher calculated and marked participants skin overlying uTrap and SCM, to indicate the sites for transducer application. For SCM, participants were required to lie supine. The mastoid process and the sternoclavicular joint were marked and identified using palpation. Using a measuring roll, the mid-point between these anatomical landmarks was calculated and marked with red non-permanent pen to indicate the scanning location. Following this,

participants were required to sit in an arm chair, with their forearms pronated on the arm rests. The scanning site for uTrap was then established by a similar process by calculating (using the measuring roll) and marking (using non-permanent red pen) the mid-point between the seventh cervical vertebra and the acromion process. Participants were scanned in different positions for SCM and uTrap designed to avoid tension or load on the tissues. For SCM, participants lay supine on a massage table with an adjustable head piece, which was altered by the primary researcher to ensure that the neck was in neutral and SCM was parallel to the table. When scanning uTrap, participants were positioned seated in an armchair with their forearms pronated, resting on the arm rests.

The following scanning procedure was the same for both SCM and uTrap. The transducer was placed horizontally at the marked midpoint of the muscle. To capture the thickest aspect of the muscle, slight movements of the transducer in the coronal plane were permitted. The sonographer then rotated the transducer parallel to the muscle to then capture an image for thickness measurements. The thickness of three structures were measured; the muscle, the superficial layer of deep cervical fascia (SF) (overlying the muscle) and the deep layer of the deep cervical fascia (DF) (underlying the muscle). To provide a mean, the sonographer took three measurements which included a centre measurement at the mid-point of the image, and then one centimetre both right and left of the middle measurement. Images were captured of the measured tissues and their corresponding thickness values and saved offline for later analysis. The sonographer was blinded to the group status of all participants (all images for each participant were saved under their allocated number, prescribed by the primary researcher), and opaque self-adhesive paper was used to mask the thickness values displayed on the USI monitor.

Data Analysis:

After the conclusion of data collection, raw measurements were extracted from each saved image and tabulated in a spreadsheet. Values for the mean of each structure of interest were calculated and the assumptions of normality checked using inspection of P-P and Q-Q plots, calculating the Shapiro-Wilk statistic, and checking the z-score for skewness and kurtosis lay within ± 2.58 (Laerd, n.d). All variables were normally distributed. Descriptive statistics were generated for participant characteristics and an independent samples t-test used to check for differences between the variables used for matching (NDI score, height, weight, age). The mean values in fascial thickness for SF and DF in SCM and uTrap bilaterally were contrasted

using independent samples t-test and statistical significance was defined as $\alpha < 0.05$. A 95% confidence interval for the mean difference between groups was also calculated. Cohen's effect size for each contrast was calculated and interpreted using the suggested descriptors offered by Cohen (1988). Observed (*post hoc*) power calculations were made using G*Power v.3.1 (Faul et al., 2009). All other statistical analysis was conducted using SPSS v.25 (IBM SPSS, Armonk, NY).

Results

A total of 18 females (9 with CNP and 9 controls) participated in this study. The descriptive characteristics for all participants are detailed in Table 1.

Table 1: Descriptive Characteristics

	Group	n	Mean	SD	p-value ^a
NDI	Symptomatic	9	8.7	5.9	0.034
	Control	9	3.1	4.1	
Height (cm)	Symptomatic	9	166.1	5.7	0.229
	Control	9	170.0	7.4	
Weight (kg)	Symptomatic	9	69.8	8.5	0.510
	Control	9	67.3	6.8	
Age	Symptomatic	9	25.2	2.8	0.660
	Control	9	24.6	3.5	

Notes: ^a = p-value from independent samples t-test; NDI = Neck Disability Index Questionnaire

The mean values in fascial thickness for SF and DF in SCM and uTrap bilaterally, show no differences between CNP and control groups (see Table 2 and 3). The results provided data required for a power analysis to determine an effect size, these are displayed in Tables 2 and 3 using Cohens descriptors (Cohen, 1988). Based on the effect sizes observed in the current study, a sample size of n=64 for uTrap (based on a 'medium' effect size), and n=394 for SCM (based on a small effect size) is required, per group, to achieve sufficient power (i.e. minimum of 80%).

During scanning there were no allergic reactions to the hypoallergenic ultrasound gel or any harm reported by participants.

Table 2: Results showing thickness values of the deep cervical fascia enveloping SCM in CNP and controls

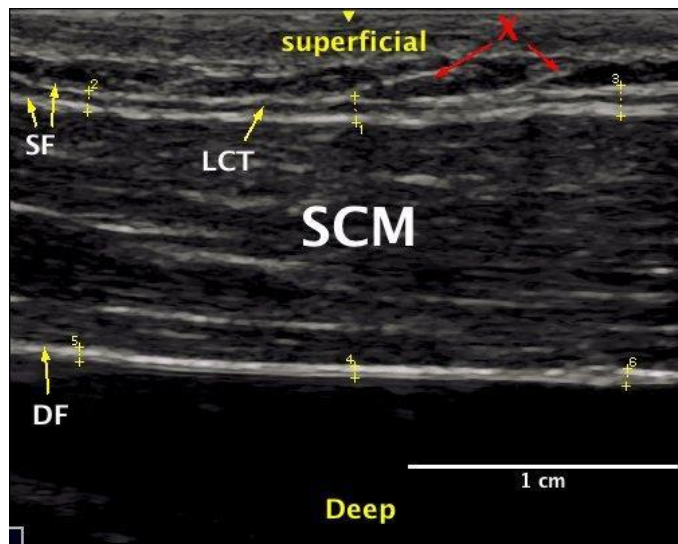
			Mean (cm) n = 9	SD (cm)	95%CI	p-value	Mean difference (cm)	95%CI of mean difference (cm)	Effect Size	Observed Power	Effect descriptor
SCM											
Left	Muscle	Symp	0.758	0.163	0.667 to 0.866	0.992	- 0.027	- 0.133 to 0.133	0.17	0.063	trivial
		Control	0.786	0.158	0.686 to 0.884						
	SF	Symp	0.106	0.533	0.744 to 0.141	0.450	- 0.016	- 0.027 to 0.060	0.39	0.12	small
		Control	0.089	0.032	0.069 to 0.110						
	DF	Symp	0.068	0.018	0.058 to 0.080	0.303	- 0.012	- 0.044 to 0.020	0.41	0.13	small
		Control	0.080	0.413	0.062 to 0.113						
Right	Muscle	Symp	0.729	0.184	0.608 to 0.846	0.759	- 0.035	- 0.202 to 0.133	0.21	0.07	small
		Control	0.764	0.149	0.659 to 0.857						
	SF	Symp	0.099	0.051	0.069 to 0.133	0.363	0.0178	- 0.024 to 0.133	0.44	0.14	small
		Control	0.080	0.029	0.064 to 0.099						
	DF	Symp	0.081	0.034	0.062 to 0.104	0.119	0.148	- 0.011 to 0.041	0.61	0.23	medium
		Control	0.066	0.015	0.057 to 0.075						

Notes: SCM = Sternocleidomastoid, SF = the superficial layer of the deep cervical fascia overlying the muscle, DF = the deep layer of the deep cervical fascia underlying the muscle, Symp = CNP group, Effect size descriptors are those described by Cohen (1988).

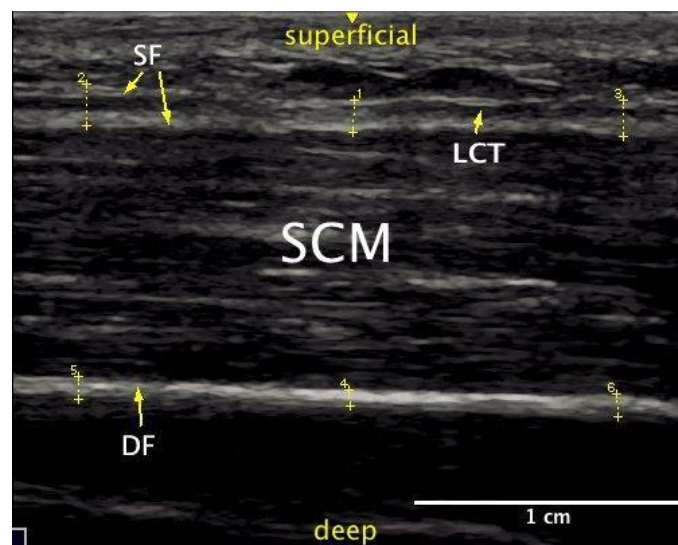
Table 3: Results showing thickness values of the deep cervical fascia enveloping uTrap in CNP and controls

			Mean (cm) n = 9	SD (cm)	95%CI	p-value	Mean difference (cm)	95%CI of mean difference (cm)	Effect Size	Observed Power	Effect descriptor
uTrap											
Left	Muscle	Symp	1.009	0.222	0.865 to 1.157	0.452	0.087	- 0.112 to 0.287	0.44	0.14	small
		Control	0.921	0.173	0.805 to 1.031						
	SF	Symp	0.113	0.043	0.088 to 0.142	0.597	- 0.035	- 0.072 to 0.001	0.99	0.51	large
		Control	0.149	0.028	0.132 to 0.166						
	DF	Symp	0.234	0.164	0.128 to 0.341	0.001	0.038	0.009 to 0.266	1.26	0.71	large
		Control	0.096	0.054	0.063 to 0.132						
Right	Muscle	Symp	1.027	0.236	0.896 to 1.182	0.706	0.278	- 0.112 to 0.365	0.53	0.18	medium
		Control	0.901	0.241	0.734 to 1.061						
	SF	Symp	0.128	0.051	0.099 to 0.164	0.913	- 0.017	- 0.064 to 0.030	0.36	0.11	small
		Control	0.144	0.043	0.118 to 0.170						
	DF	Symp	0.276	0.178	0.178 to 0.411	0.218	0.136	- 0.002 to 0.274	1.05	0.55	large
		Control	0.140	0.081	0.093 to 0.196						

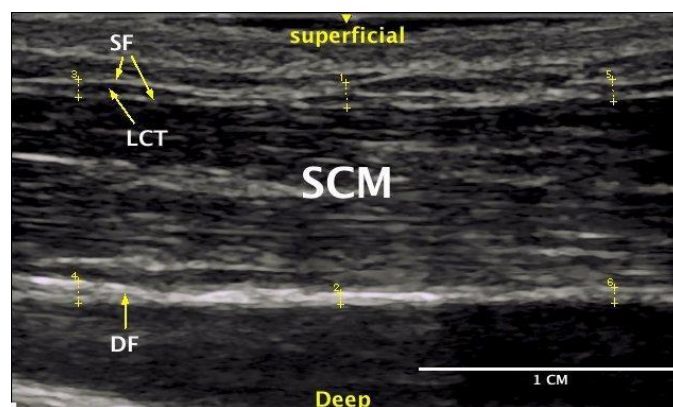
Notes: uTrap = upper Trapezius, SF = the superficial layer of the deep cervical fascia overlying the muscle, DF = the deep layer of the deep cervical fascia underlying the muscle, Symp = CNP group, Effect size descriptors are those described by Cohen (1988).



Panel A: SCM measurements in a control participant. The LCT layer is present between SF as a singular hypoechoic line. Hyperechoic bands appear to branch off the SF and connect with superficial layers (indicated by X). Researchers were unsure if the structures marked by 'X' represented fascial tissue and whether to include it in the measurement.

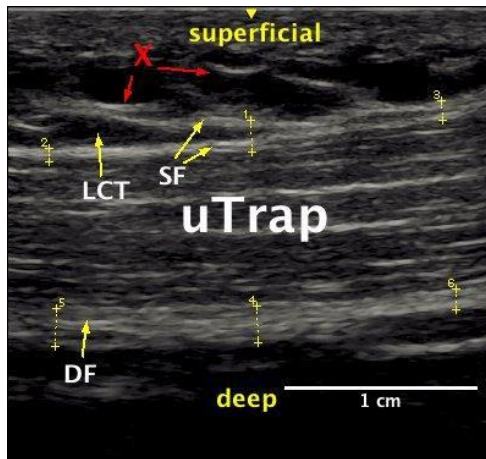


Panel B: SCM measurements in a control participant. A LCT layer is visible at the SF, indicating densification. The left SF thickness measurement (callipers labelled at 2), was 0.15cm which Stecco et al. (2014) report as diagnostic for myofascial pain.

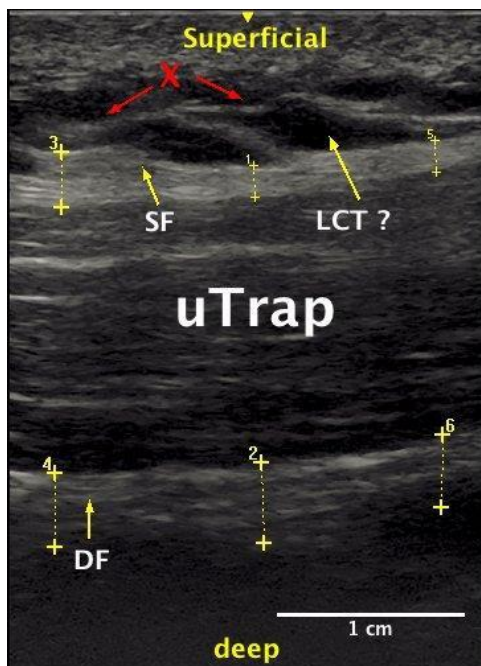


Panel C: SCM measurements in a symptomatic participant. Similar to panel B and A, this image illustrates the presence of a LCT layer at the SF with hyperechoic bands connecting to superficial tissues.

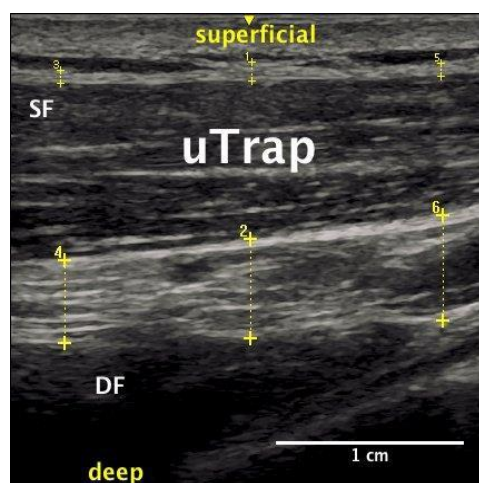
Figure 1. Measurements of SCM fascial thickness showing a varying degree of fascial morphology at the level of SF in images captured from CNP and control groups. Notes: SCM = sternocleidomastoid muscle; SF = superficial layer of fascia; DF = deep layer of fascia; LCT = loose connective tissue.



Panel A: uTrap measurements in a control participant. A frayed appearance of fascia is present at the SF and 'X'. Researchers were unsure whether hyperechoic bands represented in the image by 'X' were fascial tissue.



Panel B: uTrap measurement in a control participant. Similar findings of frayed SF tissue are illustrated in this image (indicated by 'X'). Consequentially, researchers were unsure if the marked hypoechoic regions superior to the SF indeed represented LCT and densification.



Panel C: uTrap measurements in a CNP participant. This image illustrates a large thickness of the DF. Contrast to the asymptomatic images, the SF appears as a linear hyperechoic layer adjacent to the muscle.

Figure 2. Measurements of uTrap fascial thickness showing a varying degree of fascial morphology in images captured from CNP and control groups. Notes: uTrap = upper trapezius muscle; SF = superficial layer of fascia; DF = deep layer of fascia; LCT = loose connective tissue.

Discussion

The aim of this study was to compare fascial thickness between participant groups with, and without, CNP. In this study there were no significant difference in mean fascia thickness values between groups, for SCM. There was one significant difference in mean fascia thickness values between groups for the DF in the left uTrap. However, as a pilot study the aim was also to provide the required data to calculate an effect size for the difference between groups in order to inform the design of future, larger scale, research. To our knowledge, the current study is the first to report the required data to calculate an effect size to detect differences in fascial thickness between CNP and controls.

Three previous studies have investigated the thickness of fascia in the neck using USI (Harley, 2016; Salavati et al., 2017; Stecco et al., 2014), however, for reasons outlined below, this data could not be used to calculate a sample size. A pre-clinical study by Stecco et al. (2014) used USI to measure SCM fascial thickness in people CNP ($n = 28$) and controls ($n = 25$). While Stecco et al reported the mean thickness values, they did not report standard deviations which prevented calculations for an effect size and a power analysis to determine an appropriate sample size. Other studies reported sufficient data but lacked either a control or symptomatic group, for example Harley (2016) investigated intra and inter-rater reliability of USI for measuring SCM fascial thickness in asymptomatic individuals only. Salavati et al., (2017) investigated the reliability of USI investigation used to measure fascial thickness of uTrap but lacked a control group of uTrap. The current study identified effect sizes (Cohen, 1988) for detecting differences in SCM (a small effect) and uTrap (a medium effect) fascia thickness. Based on the results of the current study, a power analysis indicates that a sample size of $n=394$ for SCM, and $n=64$ for uTrap (per group), would be required to detect differences in fascial thickness between CNP and controls (assuming 80% power). Although this informs future research of the required sample size, it also highlights that the research from Stecco et al. (2014) and the current study are likely to be substantially under-powered, therefore caution should be taken when interpreting results.

Our findings showed no significant differences in fascial thickness relating to SCM and the SF of uTrap between individuals with and without CNP. The mean thickness values for SCM fascia are less than 0.15cm, which Stecco et al. (2014) concluded was diagnostic for CNP

relating to densification. There are a few factors that may explain this discrepancy. Firstly, due to small sample sizes, the current study and work by Stecco et al. (2014) appear significantly under-powered to detect significant differences in fascial thickness. Alternatively, differences in operator experience may provide a potential explanation for the differences in the results between both studies. Harley (2016), demonstrated that an experienced sonographer can reliably measure the thickness of the deep cervical fascia, nevertheless reported poor *inter-rater* reliability (comparing to a novice USI operator), which the author suggested was due to the inconsistent measures taken by the novice sonographer. While our study recruited an expert sonographer, Stecco et al. (2014) did not report the experience or qualifications of the sonographer. It appears unclear whether operator experience may have threatened the validity of the results.

Qualitative observations of data images:

Although our findings showed no differences in fascial thickness between groups, the majority of the images for both groups present visible features that suggest densification, as described by Stecco et al. (2014), namely the presence of a black LCT layer between white layers of fibrous tissue (see figure 1 and 2) in the sonographic image. Interestingly, these same features are visible in images taken from a sample of asymptomatic individuals in Harley's (2016) study.

Throughout the study, the sonographer, despite considerable expertise in musculoskeletal scanning including previous measures of fascia, expressed persistent difficulty identifying fascial boundaries for caliper placement. This was largely due to differences in fascial morphology observed between multiple participants. A number of recorded images illustrated variations in the SF where a large proportion of images displayed a 'frayed' appearance rather than a hyperechoic line, that Stecco et al. (2014) described as normal (see Figures 1 and 2). The frayed tissues were continuous with superficial tissues and had a similar echogenicity to SF, which complicated the identification of fascial boundaries (illustrated in Figure 1, panel A and Figure 2, panels A and B). This finding may represent fascial septa that intersect the LCT layers described in fascial anatomy (Stecco, 2015). This appearance was not described in the previous literature investigating CNP and densification (Harley, 2016; Stecco et al., 2014) and it therefore appears less likely that this observation reflects abnormal anatomy.

Features present in our images coincide with Stecco et al's. (2014) sonographic description of densification, namely the presence of black hypoechoic layers between white hyperechoic lines indicating an expansion of LCT. It is possible that the frayed appearance may indicate densification of a differing severity than the images reported in Stecco et al's. (2014) research. However, despite the visible features indicating densification, the mean thickness values still fall beneath 0.15cm. This strengthens the aforementioned suggestion that under-powered sample sizes and differences in operator experience may have caused inconsistencies in measurement inter-rater reliability and therefore produced unreliable fascial thickness values.

Strengths, limitations and future considerations

The current study has notable strengths that contribute to internal validity. First, is the experience of the sonographer that increases the internal validity of the measurements. The sonographer has over 20 years experience specialising in musculoskeletal USI. Additionally, the sonographer also participated in Harley's (2016) reliability study that reported good inter-rater reliability across two follow-up sessions, thus a further reliability study was not considered necessary.

Another strength contributing to internal validity was the close matching of participants by age, sex, physical activity levels, height and weight, that reduced the chances of additional confounding variables. However, the strict matching of the sample may have resulted in gender and age restrictions on the generalisability of our study. With respect to recruitment, this took place chiefly by word-of-mouth within staff and students of a large tertiary institution, and a younger age group could be expected compared to the general population. The female sample group was also unintentional, however, does reflect epidemiological data showing a female predominance in CNP (Cheng, Su, Yen, Lui, & Cheng, 2015; Khosrokiani, Letafatkar & Sokhanguie, 2018).

The sedentary status of our participants may be a confounding factor in the results. In the IPAQ, all participants reported a minimum of 5 hours per day in a chair, which may potentiate the mechanisms proposed to cause densification. A previous meta-analysis and systematic review of studies investigating shoulder, neck and forearm muscle EMG activity in office workers concluded that stimulated work place stressors induce increased levels of

muscle activity (Eijkelhof et al., 2013), hypothesising that over time even small increases in muscle activity could impose a significant burden and damage small muscle fibres.

It is plausible that this overload may result in an accumulation of lactic acid within the LCT, which then interacts with hyaluronic acid leading to densification as a result of lowered pH. Therefore, higher hours spent at a desk could explain why our thickness values for controls are almost double to those reported by Harley (2016) who used the same sonographer as the current study but studied only asymptomatic individuals. Therefore, future researchers might consider the potential biomechanical consequences of the ergonomic environments of participants when designing future studies.

The current study identified a number of factors such as varying fascial morphology, and fascial boundary misclassification. Such observations may threaten the feasibility of using USI to measure fascial thickness in people with CNP. In addition to the small effect size, it is possible that fascial thickness is an inappropriate sole measure of fascial densification. A recent investigation into thoracolumbar fascia morphology used a ten point rating system to grade fascia on a continuum from 'very organised' to 'very disorganised' (De Coninck, Hambly, Dickinson, & Passfield, 2018). This grading approach may possess greater utility than the sole sonographic measurement of fascial thickness, but it should be noted that detailed morphology of the thoracolumbar fascia is well-established. In contrast, comparatively less is known about the cervical fascia.

Conclusion

Densification is a novel theory that may provide a pathoanatomical link and possible underlying mechanism between myofascial pain and CNP. However, within this study, the majority of participants with CNP showed no differences in fascia thickness values compared with asymptomatic controls. While these results may appear to challenge the clinical importance of fascial alterations to CNP, our power analysis indicates that the current study was substantially under-powered and so should be interpreted accordingly. Future studies using larger sample sizes will be required to explore the association between CNP and densification.

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Section III – Appendices

Appendix A: CONSORT checklist in accordance to the current study reported in section II



CONSORT 2010 checklist of information to include when reporting a pilot or feasibility trial*

Section/Topic	Item No	Checklist item	Reported on page No
Title and abstract			
	1a	Identification as a pilot or feasibility randomised trial in the title	50
	1b	Structured summary of pilot trial design, methods, results, and conclusions (for specific guidance see CONSORT abstract extension for pilot trials)	51
Introduction			
Background and objectives	2a	Scientific background and explanation of rationale for future definitive trial, and reasons for randomised pilot trial	52-54
	2b	Specific objectives or research questions for pilot trial	54
Methods			
Trial design	3a	Description of pilot trial design (such as parallel, factorial) including allocation ratio	55
	3b	Important changes to methods after pilot trial commencement (such as eligibility criteria), with reasons	55
Participants	4a	Eligibility criteria for participants	55-56
	4b	Settings and locations where the data were collected	55
	4c	How participants were identified and consented	55
Interventions	5	The interventions for each group with sufficient details to allow replication, including how and when they were actually administered	57-58
Outcomes	6a	Completely defined prespecified assessments or measurements to address each pilot trial objective specified in 2b, including how and when they were assessed	57-58

	6b	Any changes to pilot trial assessments or measurements after the pilot trial commenced, with reasons	55
	6c	If applicable, prespecified criteria used to judge whether, or how, to proceed with future definitive trial	N/A
Sample size	7a	Rationale for numbers in the pilot trial	55
	7b	When applicable, explanation of any interim analyses and stopping guidelines	N/A
Randomisation:			
Sequence generation	8a	Method used to generate the random allocation sequence	N/A
	8b	Type of randomisation(s); details of any restriction (such as blocking and block size)	N/A
Allocation concealment mechanism	9	Mechanism used to implement the random allocation sequence (such as sequentially numbered containers), describing any steps taken to conceal the sequence until interventions were assigned	N/A
Implementation	10	Who generated the random allocation sequence, who enrolled participants, and who assigned participants to interventions	58
Blinding	11a	If done, who was blinded after assignment to interventions (for example, participants, care providers, those assessing outcomes) and how	58
	11b	If relevant, description of the similarity of interventions	N/A
Statistical methods	12	Methods used to address each pilot trial objective whether qualitative or quantitative	59
Results			
Participant flow (a diagram is strongly recommended)	13a	For each group, the numbers of participants who were approached and/or assessed for eligibility, randomly assigned, received intended treatment, and were assessed for each objective	55
	13b	For each group, losses and exclusions after randomisation, together with reasons	55
Recruitment	14a	Dates defining the periods of recruitment and follow-up	55
	14b	Why the pilot trial ended or was stopped	N/A
Baseline data	15	A table showing baseline demographic and clinical characteristics for each group	60
Numbers analysed	16	For each objective, number of participants (denominator) included in each analysis. If relevant, these numbers should be by randomised group	61-62
Outcomes and estimation	17	For each objective, results including expressions of uncertainty (such as 95% confidence interval) for any estimates. If relevant, these results should be by randomised group	61-62
Ancillary analyses	18	Results of any other analyses performed that could be used to inform the future definitive trial	60

Harms	19	All important harms or unintended effects in each group (for specific guidance see CONSORT for harms)	60
	19a	If relevant, other important unintended consequences	N/A
Discussion			
Limitations	20	Pilot trial limitations, addressing sources of potential bias and remaining uncertainty about feasibility	66 & 68
Generalisability	21	Generalisability (applicability) of pilot trial methods and findings to future definitive trial and other studies	68
Interpretation	22	Interpretation consistent with pilot trial objectives and findings, balancing potential benefits and harms, and considering other relevant evidence	68
	22a	Implications for progression from pilot to future definitive trial, including any proposed amendments	68
Other information			
Registration	23	Registration number for pilot trial and name of trial registry	N/A
Protocol	24	Where the pilot trial protocol can be accessed, if available	N/A
Funding	25	Sources of funding and other support (such as supply of drugs), role of funders	N/A
	26	Ethical approval or approval by research review committee, confirmed with reference number	55

Eldridge, S. M., Chan, C. L., Campbell, M. J., Bond, C. M., Hopewell, S., Thabane, L., & Lancaster, G. A. (2016). CONSORT 2010 statement: extension to randomised pilot and feasibility trials. *Pilot and feasibility studies*, 2(1), 64. <https://doi.org/10.1186/s40814-016-0105-8>

*We strongly recommend reading this statement in conjunction with the CONSORT 2010, extension to randomised pilot and feasibility trials, Explanation and Elaboration for important clarifications on all the items. If relevant, we also recommend reading CONSORT extensions for cluster randomised trials, non-inferiority and equivalence trials, non-pharmacological treatments, herbal interventions, and pragmatic trials. Additional extensions are forthcoming: for those and for up to date references relevant to this checklist, see www.consort-statement.org.

Appendix B: Emailed ethics approval

6/1/2018

Gmail - Ethics application 2018-1001 van der Linden



Rob Moran <robertwmoran1@gmail.com>

Ethics application 2018-1001 van der Linden

Nigel Adams <nadams@unitec.ac.nz>

31 May 2018 at 17:58

To: sarah van der Linden <sarah.vanders@hotmail.com>

Cc: Rob <robertwmoran1@gmail.com>, Asher Lewis <alewis@unitec.ac.nz>, Unitec Research Ethics Committee <ethics@unitec.ac.nz>

sarah.vanders@hotmail.com

Dear Sarah

Ethics application number: 2018-1001

Thank you for completing and submitting the amendments requested. As Primary Reader of your application and under delegated authority from the Unitec Research Ethics Committee (UREC) I now authorise you to begin your research.

Please note, if you have not yet done so at the time of receiving this advice; please email one copy of your final amended ethics application and any additional documents to the UREC secretary at: ethics@unitec.ac.nz. You will receive a formal letter of approval after the next UREC meeting. Note meetings are held monthly.

The dates that must be referred to on the Information Sheet AND Consent Forms given to all participants and appear on your documents are as follows:

Start date: 31 May 2018

Finish date: 31 May 2019

Please note, you must inform UREC, in advance of any ethically-relevant modification in the project as this may require additional approval.

Best wishes for your project.

Signed,

Nigel Adams

https://mail.google.com/mail/u/0/?ui=2&ik=150ad61e33&jsver=uCCkUJ8n4pE.en_GB.&cbl=gmail_fe_180516.06_p8&view=pt&msg=163b4c67b2e9ef92&search=inbox&siml

Appendix C: online email advertisement for participant recruitment

<https://thenest.unitec.ac.nz/TheNestWP/calling-staff-with-neck-pain-chance-to-be-part-of-a-masters-osteopathy-study/>

our unitec

working @ unitec

policies & forms

services

teaching & research

our networks

student support

quick links

WellYou

Emergency Management

Ask Us / Self Service

IMS Self Service

Academic Resources

Staff Portal

Resources & Info

News & Events

Staff Directory

Performance Partnering

Report an Injury/hazard

Submit News or Classified Ad

Categories

- Classified Ads
- dept_news
- Feature Articles
- News
- Staff Notices
- Uncategorized

Archives

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The Nest News

« Your cost-saving ideas

\$50 for five weeks at Unitec gym »

Calling staff with neck pain. Chance to be part of a Masters Osteopathy study

Tuesday 31/07/2018

Unitec student Sarah van der Linden, as part of her Masters of Osteopathy, is looking for 15 people who have experienced neck pain over the past three months.

Sarah is conducting a research project using ultrasound to see whether there is a difference in muscle and connective tissue thickness in people with and without neck pain.

Eligible candidates will:

- Be aged 18 to 55
- Have no current diagnosed neck conditions such as tumours, arthritis and nerve impingement.
- Not regularly suffer from chronic headaches, migraines, or have any recent onset of a new/unusual headache
- Be available during August (time to be confirmed with you).

You'll attend a 30 – 45-minute ultrasound session at:


Sound Experience clinic
YMCA Leisure Centre Mt Albert
773 New North Road

The session will involve scanning the front and back of your neck to measure the thickness of particular muscles and connective tissues.


If you're interested in taking part, Sarah will ask you a few questions to ensure your eligibility. Please contact:

Sarah van der Linden: M: 0210440141; E: sarah.vanders@hotmail.com

Post Views: 110



The ultrasound machine being used to scan a patient's neck.



Sarah van der Linden

Appendix D: Participant Information Sheet



Participant Information Sheet

Research Project Title: A comparative investigation of the fascial thickness surrounding sternocleidomastoid and upper trapezius in people with and without non-specific chronic neck pain, using ultrasound imaging.

Localities:

Sound Experience Clinic,
YMCA Leisure Centre Mt Albert
773 New North Road.

Researcher: Sarah van der Linden **Contact phone number:** +64 21 044 0141

You are invited to take part in a study *comparing fascial thickness surrounding certain muscles in the neck in people with non-specific chronic neck pain and people without neck pain.*

Whether or not you take part in this study is your choice. If you do not wish to take part, it will not affect your treatment/medical care at the student osteopathic clinic (Clinic 41). If you choose to take part now, but change your mind later, you can pull out of the study at any time up until 5pm one working day after data is collected. You don't need to explain the reasons for this.

This participation information sheet will inform you on the research project and will help you decide whether you wish to take part in it. It sets out why this research project is being done, what your participation will require, the potential risks and benefits, and whether you are eligible for the study. Please read through this sheet carefully. If you have any further questions or concerns, we will go through them with you. You do not have to decide straight away whether or not you would like to participate in this study. Before you decide, you may want to take a bit of time to talk about the study with your family, whānau, friends or healthcare providers. Feel free to do this.

If you meet the criteria and wish to participate in this study, you will be asked to sign a consent form. A copy of both the participant information sheet and a signed consent form will be given to you to keep. Your name, contact details and any information that might identify you will be kept confidential between you and the researchers of this study. All information from which you could be identified will be stored in a password controlled computer file where only the project researchers will have access to it.

OUTLINE AND PURPOSE OF THIS STUDY

Neck pain is common with the majority cases having unknown causes. This means that managing the condition and reducing the pain can be particularly challenging. A large amount of research, particularly into the muscles of the neck, has been carried out in an attempt to better understand neck pain and inform treatment options. Despite this, the occurrence of neck pain still remains high. Although there has been a large amount of research centred on muscular contributions neck pain, little research has been done on the connective tissue that encases these muscles, called fascia.

Recent researchers have found an increase of the thickness of fascia over particular neck muscles in people with neck pain. They propose that this thickening might be partly responsible in the development of neck pain. If this is correct, it could potentially lead to more informed and effective treatment. This research and the produced results are not reliable and need to be validated.

This research project aims to add to this evidence by using ultrasound to measure the thickness of fascia and the related muscles in the necks of people with and without neck pain.

WHAT WILL MY PARTICIPATION IN THE STUDY INVOLVE?

You will be required to be available for one 30-45-minute scanning session at the Mount Albert, YMCA Leisure Centre in the Sound Experience Clinic. You will be asked to wear a singlet with thin straps, or change into the provided elastic strapless top, so that your shoulders and neck are exposed to scan the sternocleidomastoid and upper Trapezius muscles. If you are a male, you will have the option of removing your shirt if comfortable.

Prior to the scan, you will be asked to sign and read the consent form, complete a short questionnaire (designed to take between 5-10 minutes to complete) and an investigator will measure and record your height and weight. The investigator will then use a measuring roll and a non-permanent felt-tip pen to calculate and mark your skin to represent the 4 sites to be scanned on the neck and shoulders.

The registered sonographer, Scott Allen from Sound Experience, will use an ultrasound probe with skin gel to scan the marked spots around your neck. The probe transmits and receives

sound waves to create a detailed computer image of the muscles and connective tissues in your neck so that we can measure them. During the scan, you will be required to refrain from talking and adopt a normal/relaxed position. These positions will involve lying on your back on a massage bed and seated in an arm chair.

At the end of the scan, the gel will be wiped clean from your neck with a paper towel and you may get changed into the clothes you arrived in. In consideration of your participation, we would like to offer those who have travelled to the YMCA for scanning, a \$10 voucher to contribute to your travel costs. We will present these to participants after the scanning session at the YMCA.

WHAT ARE THE POTENTIAL RISKS AND BENEFITS OF THIS STUDY?

The procedure of this study poses no risk to your health as a participant. Ultrasound imaging has been highly researched and has no risks associated when performed by experienced practitioners. The examination of your neck should be free of discomfort.

The potential benefits of this study will be an increased knowledge on potential causes of neck pain, which may influence additional research in this area. This may affect treatment choices within healthcare and potentially provide improved clinical results for people with non-specific chronic neck pain.

WHO CAN TAKE PART IN THIS STUDY?

We are looking for people who have non-specific chronic neck pain, and also people who don't have any neck pain. All participants must be between 18 and 55 years of age, be able to read and understand this information sheet and sign a consent form in English. All participants will be required to complete the questionnaire at clinic 41 at the beginning of the scanning session.

If you **HAVE** neck pain, you are eligible to take part in this study if you:

- Agree to complete a neck disability index questionnaire (NDI) and score a particular value.
- Have had neck pain for most days for at least the previous 3 months
- Have neck pain intensity of at least 20mm on a 100mm visual analogue scale (this will be established through the questionnaire on the day of scanning)

If you have **NO** neck pain, you are eligible to take part in this study if you:

- Have had **no** neck pain lasting over 1 month, or recurrent neck pain with episodes adding to greater than 1 month for at least 2 years.

Unfortunately, **you cannot** participate in this study if you

- Are experiencing chronic/ and or acute neck pain due to a **known** cause such as disc pathology, neural compromise, congenital deformity of the spine, arthritis, neoplasm, inflammatory rheumatoid disease, etc.
- Are experiencing neck pain for a duration of **less** than 3 months.
- Are suffering from neurological symptoms in your arms such as, loss of sensation, pins and needles, weakness, paralysis.
- Have neck pain related to a motor vehicle accident or trauma within the last 6 months.
- Experience **chronic** headaches, regular migraines requiring prescription medicine or, if you are suffering from an unusual/unfamiliar headache in the last week.

WHAT HAPPENS AFTER THE STUDY?

The study findings and results will be available to you free of charge. If you wish to receive these, they will be emailed to you upon completion of this research project. It may take up to 18 months for the results and findings to be complete. After the study is finished, data will be kept securely and privately onsite at Unitec for 10 years, after which it will be securely disposed of.

WHO DO I CONTACT FOR MORE INFORMATION OR IF I HAVE CONCERNS?

For additional information, queries or concerns about the study, please contact us using the information provided:

Sarah van der Linden (researcher)

Email: sarah.vanders@hotmail.com

Phone: +64210440141

Rob Moran (Principal supervisor):

Email: rmoran@unitec.ac.nz

If you require Māori cultural support, talk to your whānau in the first instance. Alternatively, you may contact:

He Kamaka Waiora (Māori Health Team): Phone: (09) 486 8324 ext 2324

UREC REGISTRATION NUMBER: 2018-1001

This study has been approved by the UNITEC Research Ethics Committee from 25/05/2018 to 25/05/2019. If you have any complaints or reservations about the ethical conduct of this research, you may contact the Committee through the UREC Secretary (ph: 09 815-4321 ext 8551). Any issues you raise will be treated in confidence and investigated fully, and you will be informed of the outcome.

Appendix E: Participant Consent Form



Participant Consent Form

Project Title: A comparative investigation of the fascial thickness surrounding sternocleidomastoid and upper trapezius in people with and without non-specific chronic neck pain, using ultrasound imaging.

Localities: Osteopathy Clinic 41, Unitec Institute of Technology

Investigator: Sarah van der Linden
0210440141

Contact phone number:

PLEASE TICK TO INDICATE YOU CONSENT TO THE FOLLOWING:

I have had the research project explained to me and I have read and understand the information sheet given to me.

Yes

☐

No

☐

I have the appropriate contact details and know whom I need to speak to if I have questions or concerns related to this project.

Yes

☐

No

☐

I understand that I can withdraw myself from this research study (until 5pm on the following business day after data collection) without having to give justification for doing so and without affecting continuing care I might be receiving.

Yes

☐

No

☐

I understand the information I provide will be kept confidential to the researcher and the supervisor, and any information from which I could be

Yes

☐

No

☐

identified will be stored in password controlled files and/or locked filing cabinets.

I understand that any pictures or video recordings taken through ultrasound imaging will not be personally identifiable in any way.

Yes

☐

No

☐

I understand that ultrasound is a safe form of imaging and that there is a small chance of skin irritation from the gel. Yes ☐ No ☐

I understand that my shoulders and neck need to be exposed in order to complete the scanning procedure involved in this study. Yes ☐ No ☐

I understand that my skin will temporarily be marked with a non-permanent felt-tip pen to represent the sites to be scanned. Yes ☐ No ☐

I understand that overall results from the study may be presented in a Yes ☐ No ☐

publicly accessible thesis, in scientific presentations or reports, but it will not be possible from these to identify me, or any other individual who participates in the study.

Declaration by participant:

I have had time to properly consider all of the provided information, and have had any additional questions answered. I hereby consent to participate in this research project.

Participant Name:

Participant Signature: Date:
.....

Declaration by investigator:

I have given a written and verbal explanation of the research project to the participant, and have answered the participant's questions about it.

I believe that the participant understands the study and has given informed consent to participate.

Researcher Signature: Date:
.....

UREC REGISTRATION NUMBER: 2018-1001

This study has been approved by the UNITEC Research Ethics Committee from 31/05/2018 to 31/05/2019. If you have any complaints or reservations about the ethical conduct of this research, you may contact the Committee through the UREC Secretary (ph: 09 815-4321 ext 8551). Any issues you raise will be treated in confidence and investigated fully, and you will be informed of the outcome.

Appendix F: Questionnaire (including NDI, IPAQ, VAS and descriptive questions)



Neck Pain Questionnaire

Thank you for taking part in this research project. On the following pages, you will answer questions relating to your neck pain. Please carefully read through the questions before answering. Please do not take too long or over-think your answers and instead, choose the option that first comes to mind. This should take no longer than 10 minutes.

This questionnaire has been designed to give your healthcare professional information as to how your neck pain has affected your ability to manage everyday life activities. Please mark in each section **ONE** box that applies to you. We realise that you may consider that two of the statements in any one section relate to you, but please just mark the box that most closely describes your present-day situation.

Pain Intensity

- ☐ I have no pain at the moment
- ☐ The pain is very mild at the moment
- ☐ The pain is moderate at the moment
- ☐ The pain is fairly severe at the moment
- ☐ The pain is very severe at the moment
- ☐ The pain is the worst imaginable at the moment

Lifting

- ☐ I can lift heavy weights without extra pain
- ☐ I can lift heavy weights but it gives extra pain
- ☐ Pain prevents me from lifting heavy weights off the floor, but I can manage if they are conveniently positioned, for example on a table
- ☐ Pain prevents me from lifting heavy weights, but I can manage light to medium weights if they are conveniently positioned
- ☐ I can lift very light weights
- ☐ I cannot lift or carry anything at all

Headaches

- ☐ I have no headaches at all
- ☐ I have slight headaches which come infrequently
- ☐ I have moderate headaches which come infrequently
- ☐ I have moderate headaches which come frequently
- ☐ I have severe headaches which come frequently
- ☐ I have headaches almost all the time

Personal Care

- ☐ I can look after myself without causing extra pain
- ☐ I can look after myself normally but it causes extra pain
- ☐ It is painful to look after myself, I am slow and careful
- ☐ I need some help but manage most of my personal care
- ☐ I need help every day in most aspects of self-care
- ☐ I do not get dressed, I wash with difficulty and stay in bed

Reading

- ☐ I can read as much as I want to with no pain in my neck
- ☐ I can read as much as I want to with slight pain in my neck
- ☐ I can read as much as I want with moderate pain in my neck
- ☐ I can't read as much as I want, because of moderate neck pain
- ☐ I can hardly read at all because of severe pain in my neck
- ☐ I cannot read at all because of the severe pain in my neck

Concentration

- ☐ I can concentrate fully when I want to with no difficulty
- ☐ I can concentrate fully when I want to with slight difficulty
- ☐ I have a fair degree of difficulty concentrating when I want to
- ☐ I have a lot of difficulty concentrating when I want to
- ☐ I have a great deal of difficulty concentrating when I want to
- ☐ I cannot concentrate at all

Work

- ☐ I can do as much work as I want to
- ☐ I can do my usual work
- ☐ I can do my usual work, but no more
- ☐ I can do most of my usual work, but no more
- ☐ I can hardly do any work at all
- ☐ I can't do any work at all

Sleeping

- ☐ I have no trouble sleeping
- ☐ My sleep is slightly disturbed (< 1 hr sleepless)
- ☐ My sleep is mildly disturbed (1-2 hrs sleepless)
- ☐ My sleep is moderately disturbed (2-3 hrs sleepless)
- ☐ My sleep is greatly disturbed (3-5 hrs sleepless)
- ☐ My sleep is completely disturbed (5-7 hrs sleepless)

Driving

- ☐ I can drive my car without any neck pain
- ☐ I can drive my car as long as I want with slight pain in my neck
- ☐ I can drive my car as long as I want with moderate pain in my neck
- ☐ I can't drive my car as long as I want because of moderate pain in my neck
- ☐ I can hardly drive at all because of severe pain in my neck
- ☐ I can't drive my car at all

Recreation

- ☐ I am able to engage in all my recreation activities with no neck pain at all
- ☐ I am able to engage in all my recreation activities, with some pain in my neck
- ☐ I am able to engage in most, but not all my usual recreation activities, because of some pain in my neck
- ☐ I am able to engage in a few of my usual recreation activities because of pain in my neck
- ☐ I can hardly do any recreation activities because of pain in my neck
- ☐ I can't do any recreation activities at all

(McLean, 2011) – derived from Vernon, H., & Mior, S. (1991). The Neck Disability Index: A study of reliability and validity. *Journal of Manipulative and Physiological Therapeutics*, 14, 409-415.

INTERNATIONAL PHYSICAL ACTIVITY QUESTIONNAIRE

We are interested in finding out about the kinds of physical activities that people do as part of their everyday lives. The questions will ask you about the time you spent being physically active in the **last 7 days**. Please answer each question even if you do not consider yourself to be an active person. Please think about the activities you do at work, as part of your house and yard work, to get from place to place, and in your spare time for recreation, exercise or sport.

Think about all the **vigorous** activities that you did in the **last 7 days**. **Vigorous** physical activities refer to activities that take hard physical effort and make you breathe much harder than normal. Think *only* about those physical activities that you did for at least 10 minutes at a time.

1. During the **last 7 days**, on how many days did you do **vigorous** physical activities like heavy lifting, digging, aerobics, or fast bicycling?

_____ **days per week**

☐ No vigorous physical activities → *Skip to question 3*

2. How much time did you usually spend doing **vigorous** physical activities on one of those days?

_____ **hours per day**

_____ **minutes per day**

☐ Don't know/Not sure

Think about all the **moderate** activities that you did in the **last 7 days**. **Moderate** activities refer to activities that take moderate physical effort and make you breathe somewhat harder than normal. Think *only* about those physical activities that you did for at least 10 minutes at a time.

3. During the **last 7 days**, on how many days did you do **moderate** physical activities like carrying light loads, bicycling at a regular pace, or doubles tennis? Do not include walking.

_____ **days per week**

☐ No moderate physical activities → *Skip to question 5*

4. How much time did you usually spend doing **moderate** physical activities on one of those days?

_____ **hours per day**

_____ **minutes per day**

☐ Don't know/Not sure

Think about the time you spent **walking** in the **last 7 days**. This includes at work and at home, walking to travel from place to place, and any other walking that you have done solely for recreation, sport, exercise, or leisure.

5. During the **last 7 days**, on how many days did you **walk** for at least 10 minutes at a time?

_____ **days per week**

☐ No walking → *Skip to question 7*

6. How much time did you usually spend **walking** on one of those days?

_____ **hours per day**

_____ **minutes per day**

☐ Don't know/Not sure

The last question is about the time you spent **sitting** on weekdays during the **last 7 days**. Include time spent at work, at home, while doing course work and during leisure time. This may include time spent sitting at a desk, visiting friends, reading, or sitting or lying down to watch television.

7. During the **last 7 days**, how much time did you spend **sitting** on a **week day**?

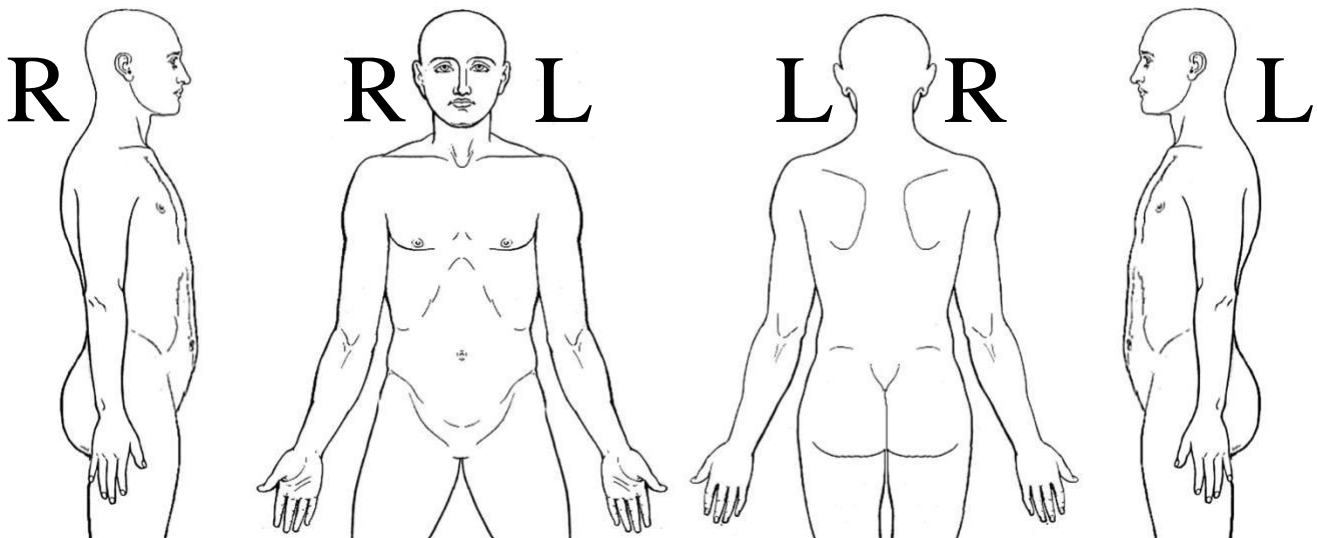
_____ **hours per day**

_____ **minutes per day**

☐ Don't know/Not sure

This is the end of the questionnaire, thank you for participating.

Outline the area on body map that best represents the location of your neck pain.



How long have you been experiencing neck pain for? *Circle the duration that best represents your neck pain.*

3 months

6 months

1 year

1 – 2 years

2 – 5 years

5 + years

How often do you experience neck pain? *Tick **one** of the following boxes that best represents your neck pain.*

- ☐ I rarely have neck pain
- ☐ I sometimes have neck pain
- ☐ Most of the time I have neck pain
- ☐ I always have neck pain

Tick **one** of the following boxes that applies the most to your neck pain:

- ☐ I feel pain more on the left side of my neck

- ☐ I feel pain more on the right side of my neck
- ☐ I feel pain equally on both sides of my neck
- ☐ I feel pain more in the centre of my neck

Rate your **current** level of neck pain by marking the line below:

No pain |-----| Worst pain possible

Rate your **usual** level of neck pain by marking the line below:

No pain |-----| Worst pain possible

Date:/...../.....

Participant Name:

.....

Appendix G: ultrasound protocol

SCM Protocol

The following protocol was produced by Harley (2016). It will be used in for proposed research to measure the thickness SCM and the related deep cervical fascia.

USI Machine

- *2014 GE Logiq ultrasound machine (utilised when assembling the following protocol)*

USI Settings

- ***Preset:*** *Small parts thyroid*
- ***Transducer:*** 16.0 MHz ‘hockey stick’
- ***Depth:*** *SCM: Depth is set to the full thickness of the muscle. Fascia: write zoom to focus on the image with increased resolution*
- ***Focus:*** *A single focal zone at the maximum depth of the muscle and fascia measures*

Participant Positioning

The participant should be positioned in supine, with one (flat) head pillow. This will ensure the cervical spine is not unnecessarily flexed. The cervical spine should be in a neutral position, neither flexed nor extended, to avoid changes in length of the SCM muscle and related fascia. The head position should be neutral, and can be checked by ensuring the face is level as demonstrated below (Figure I). The participant should be advised not to speak during measurements, and to breathe normally.



Figure I. Participant positioning. The horizontal lines indicate the plane of the face is parallel to the

plane of the examination table.

Transducer placement and measurements

Begin with the measurement of SCM and related Fascia on the participant's right side

Locate both the inferior aspect of the mastoid process and the superior aspect of the clavicle head (the proximal and distal attachments of SCM muscle). Use a ruler to establish the mid-point between these landmarks.

At this point place the transducer transverse (Figure II) across the anterior neck surface.

Identify the relevant structures including:

- . a) Superficial fascia
- . b) Deep fascia overlying SCM
- . c) SCM muscle
- . d) Deep fascia underlying SCM muscle
- . e) Carotid sheath (moving medially from muscle)

Once all the structures have been identified, rotate the transducer (centre of rotation being SCM) so the SCM muscle can be viewed longitudinally. Align the transducer with the perimysium direction of the SCM muscle. See Figure III with SCM muscle highlighted. Readjust transducer pressure so jugular vein is patent (Figure IV).



Figure II. Transducer initially transverse

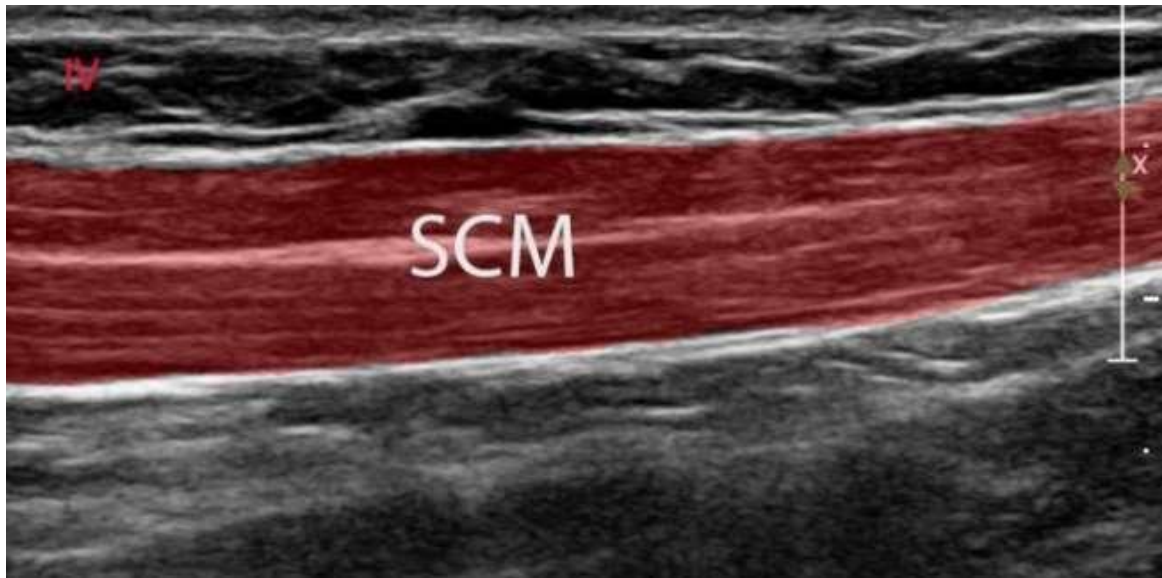


Figure III. Longitudinal view of SCM muscle

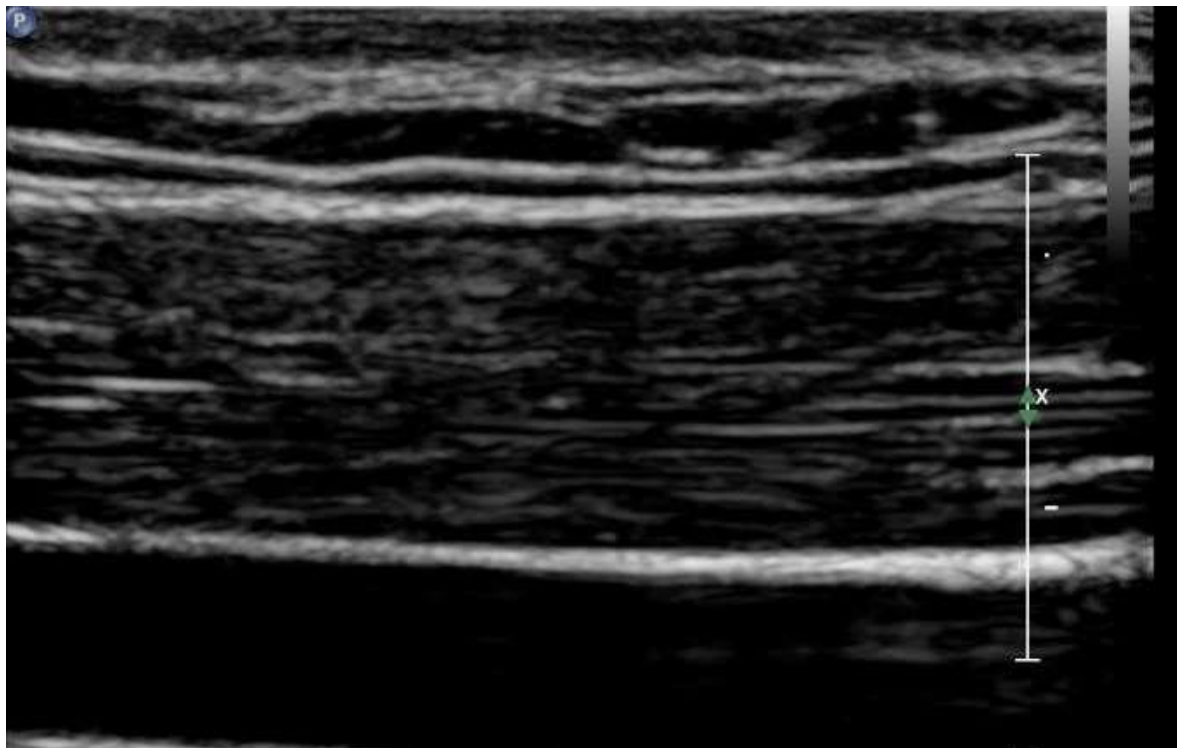


Figure IV. Patent jugular vein – inferior hypoechoic area

Viewing the SCM muscle longitudinally, identify the thickest region of this muscle within this view. Very slight transducer movement in a frontal plane to identify the thickest area of SCM is warranted. Once identified, utilise write zoom function to focus on the full muscle thickness when taking the measurement value. See image V for guidance on borders for measurement relating to SCM. Take three measurements:

- . 1) Centre measurement (thickest aspect of viewable SCM)
- . 2) 1 cm superior from centre measurement (use calipers to measure 1 cm distance)
- . 3) 1 cm inferior from centre measurement

These three measurements will later be averaged for a final measurement.

Fascia Thickness Measurement

Adjust the write zoom to focus on the fascia layer deep to SCM initially, then superficial fascia afterwards.

Fascia thickness includes the dense connective tissue layers and loose connective tissue layers. Measurement should include all layers, as demonstrated by Figure V. In some participants, the loose connective layer may not be visible. Care should be taken to identify the cutaneous and subcutaneous loose connective layers before measurement is taken.

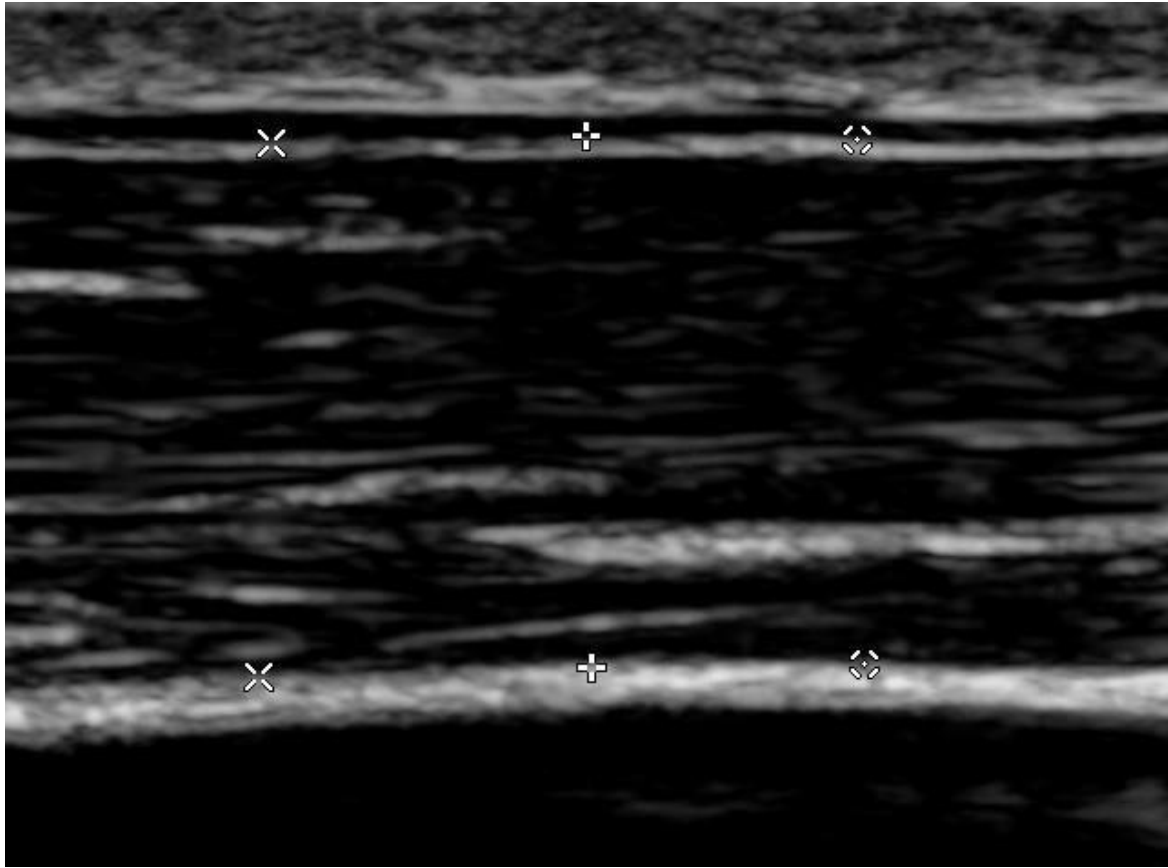


Figure V. The final thickness value will be an average of these three measurements.

Take three measurements for both the superficial aspect and deep aspect of SCM deep fascia as demonstrated in Figure V.

- . a) Centre measurement (thickest aspect of viewable SCM)
- . b) 1cm right from centre measurement
- . c) 1cm left from centre measurement

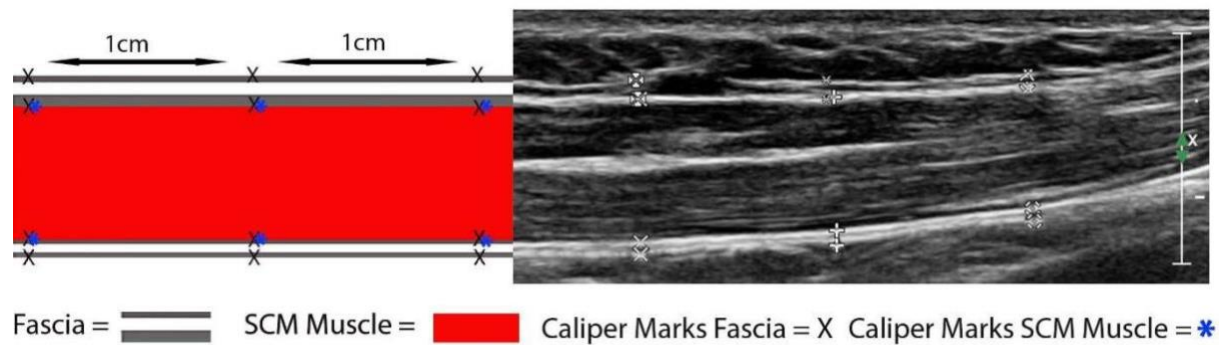


Figure V. *Caliper marking indicate the standardised measurement process utilised within the imaging protocol*

Note: The final thickness value will be an average of these three measurements. The grey layers represent the dense connective tissue layers of fascia. The white layer represents the loose connective tissue layer of fascia, a layer which is not always visible during ultrasound.

Repeat process on the contralateral side for both the SCM and fascia measurements.

Upper Trapezius Protocol

Harley's (2016) SCM scanning protocol was adapted for the scanning of UT and its associated fascia described below. Participant positioning and the scanning site were based off the methodology from Salavati et al. (2017).

USI Machine

- *2014 GE Logiq Ultrasound Machine (utilised when assembling the following protocol)*

USI Settings

- ***Preset:*** *Small parts thyroid*
- ***Transducer:*** 16.0 MHz 'hockey stick'
- ***Depth:*** *uTrap: Depth is set to the full thickness of the muscle. Fascia: write zoom to focus on the image with increased resolution*
- ***Focus:*** *A single focal zone at the maximum depth of the muscle and fascia measures*

Site of scanning and pre-measurement methods.

Participants will be measured five minutes prior to the ultrasound session. Locate the spinous process of C7 by palpating down the spinous processes (SPs) of the neck beginning at the base of the occiput. Once located, use the flexion/extension method to validate the identification of C7. For this, the participants neck will be flexed and the examiner will palpate the two most prominent SPs at the base of the neck. With assisted neck extension, the palpated SP that moves anteriorly is C6 whereas the inferior SP holding its position will be C7. Mark this location with a felt-tip pen. Use palpation to locate and mark the acromion process with a felt-tip pen. Once marked, measure the distance between the two locations to calculate and mark the half-way point using a measuring tape.

Participant position:

The patient will be seated in a chair adopting a relaxed position, with their arms pronated resting on the arms of the chair. This will prevent any postural contraction of the muscle or any excessive stretch that may alter the length of uTrap and its fascia. The patient will be asked to not speak and to breathe normally during scanning.

Transducer placement and measurements

Begin with the measurement of uTrap and its associated fascia on the participant's right side.

Place the transducer over the marked midpoint on the skin. Once applied, rotate the transducer parallel to the transverse fibres of uTrap.

Viewing the uTrap muscle longitudinally, identify the thickest aspect of this muscle within this view. Very slight transducer movement in a frontal plane is permitted to identify the thickest area of uTrap. Once identified, utilise the write zoom function to focus on the full muscle thickness when taking the measurement value. uTrap is defined as the greatest distance between the two hyperechogenic layers of fascia.

Take three measurements:

- 1) Centre measurement (the thickest aspect within the view uTrap).
- 2) 1cm superior from centre measurement (using calipers to measure 1cm distance).
- 3) 1cm inferior from centre measurement (using calipers to measure 1cm distance).

Fascia Thickness Measurement

Alter the write zoom to focus on the fascia layer **deep** to uTrap initially, then the **overlying** superficial layer of fascia afterwards.

Fascia thickness includes the dense connective tissue and LCT sublayers. It is important to note that the LCT layer may not be visible in some participants. Effort and care need to be taken to identify and differentiate between LCT and cutaneous layers before measurements are recorded.

Take 3 measurements

- 1) Centre measurement (the thickest aspect within the view of deep cervical fascia).
- 2) 1cm superior from centre measurement (using callipers to measure 1cm distance).
- 3) 1cm inferior from centre measurement (using calipers to measure 1cm distance).

Repeat this entire process on the contralateral side (left) for uTrap.

Full name of author: Sarah van der Linden

Full title of thesis/dissertation/research project ('the work'):

A pilot study using ultrasound imaging to compare fascial thickness between chronic neck pain and control groups.

Practice Pathway: Community Studies

Degree: Master of Osteopathy

Year of presentation: 2019

Principal Supervisor: Robert Moran

Associate Supervisor: Dr. Christopher McGrath

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Date: 22/03/19



Declaration

Name of candidate: Sarah van der Linden

This Thesis/Dissertation/Research Project entitled: A pilot study using ultrasound imaging to compare fascial thickness between chronic neck pain and control groups
is submitted in partial fulfillment for the requirements for the Unitec degree of Master of Osteopathy.

Principal Supervisor: Robert Moran

Associate Supervisor/s: Dr. Christopher McGrath

CANDIDATE'S DECLARATION

I confirm that:

- This Thesis/Dissertation/Research Project represents my own work;
- The contribution of supervisors and others to this work was consistent with the Unitec Regulations and Policies.
- Research for this work has been conducted in accordance with the Unitec Research Ethics Committee Policy and Procedures, and has fulfilled any requirements set for this project by the Unitec Research Ethics Committee.

Research Ethics Committee Approval Number:

Candidate Signature: [Signature] Date: 09/07/19

Student number: 1399411