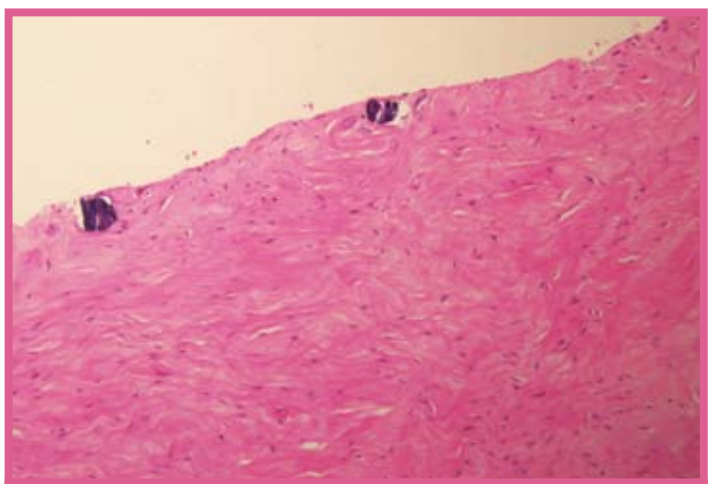
Vancouver tendinop

Alex Scott is an assistant professor at the University of British Columbia (UBC) Department of Physical Therapy and lead investigator of the Tendinopathy Research Group in the Centre for Hip Health and Mobility. Here he summarises current research conducted within the group and, to complement this, Australian postdoctoral researcher Angie Fearon, APAM, describes her latest research into the diagnosis of greater trochanteric pain syndrome.



Calcification of tendon is a recognised pathology. This tendon specimen demonstrates two calcium deposits embedded on the edge of a tendon specimen. (H&E x100)

The research interests of the Tendinopathy Research Group are diverse, but centre around the issue of tendon overuse pathology. The team is part of the Centre for Hip Health and Mobility, a multidisciplinary research centre whose vision is to conduct innovative research to enhance the hip health and mobility of Canadians across the lifespan. Through research, our mission is to prevent, detect and treat bone and joint problems, and to translate research outcomes into effective programs, policy or practice. The Centre is home to surgeons, engineers, physical therapists, radiologists, sports medicine doctors, exercise specialists and sociologists, who collaborate on a range of projects.

I lead the Tendinopathy Research Group within the Centre and I obtained funding to launch this research program from the Canadian Foundation for Innovation, the Canadian Institutes of Health Research, the Michael Smith Foundation for Health Research, and the National Sciences and Engineering Research Council. The research group comprises both PhD and MSc students, a research associate (Dr Hayedeh Behzad) and a postdoctoral fellow (Dr Angie Fearon). Each team member manages their own project, and research goals are set collaboratively.

The Tendinopathy Research Group attracts many international collaborations. In the past few months, we have hosted visiting PhD students from Copenhagen, Umea, Indiana, and Norway, leading to interesting published contributions on tendon basic or clinical science. I also teach in the professional Master of Physical Therapy program at UBC, where students are trained in, among other skills, how to read and interpret scientific studies.

As a whole, the group focuses on: (1) studying chronic pathology and discovering linkages with clinical parameters; and (2) conducting experiments with potential biological or physical therapy treatments. Current studies include: cell biological and biomechanical studies of tendons, clinical interventions for chronic tendinopathy, the influence of mechanical movement on tenocyte biology, the impact of local neuropeptide production in tendons, and the link between lifestyle factors including hypercholesterolemia and the mechanisms of tendon injury. The following is an overview of the research themes and projects currently being implemented by the Vancouver Tendinopathy Research Group.

The impact of neuropeptides on chronically overused tendons

One of our unique contributions includes the discovery that substance P (SP) production by tenocytes is upregulated by mechanical loading, and that SP stimulates collagen remodelling (submitted). PhD trainee Gloria Fong (co-supervised by Patrik Danielson of Umea University) is pursuing this finding. SP may be a key player in tendinopathies, causing pain, collagen remodelling, and neurogenic inflammation.

The role of mast cells in tendinopathy

Another of our recent findings is that mast cells play a modulatory role in tendon healing, as well as in chronic tendon pathology, perhaps by increasing the levels of TGF-beta at the site of injury. We are trying to study the underlying mechanisms and roles of this understudied cell type in tendon pathology, and each

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day brings new surprises as we observe mast cells and tenocytes interacting and signalling to one another *in vitro*.

The role of neovascularisation in tendinopathy

This series of studies examines the mechanisms by which angiogenesis occurs in tendons, leading to 'neovessels' which have been implicated in chronic tendon pathology. For this project, we repetitively strain tendons or tendon cells in the laboratory and track their production of angiogenic substances. In the future, we hope to find a way of blocking this process. The presence of 'neovessels' on power Doppler ultrasound was recently found to be a predictor of Achilles tendinopathy in runners (*Med Sci Sports Exercise* 2012).

Is there a tendinitis phase which precedes the development of tendinosis?

We are currently working with the classic model of Achilles overuse developed at Umea University but focusing on the changing cell populations—particularly in the paratendon—which occur in the early phases of tendon overuse. Many athletes self-medicate with anti-inflammatories as a strategy for preventing tendinopathies, despite the lack of evidence—both clinical and biological—to support this practice.

Tendinopathy: an overuse or underuse injury?

We are also studying the connection between high-fat diet, hypercholesterolemia and tendon pathology. For this project, we are using a mouse model of hypercholesterolemia. We are examining changes in the structure and fat content of tendon using histology and ultrasound, as well as testing biomechanics—not an easy task considering the size of an average mouse tendon. Additionally, we are tracking the cholesterol levels of patients with Achilles tendon ruptures to see if, as others report, tendon rupture patients are more likely to be dyslipidemic. Should we be sending tendinopathy patients for blood tests? These studies will potentially provide some new insights into why tendinopathies are so prevalent in sedentary individuals with no obvious history of mechanical loading.

Tendinopathy Research Group

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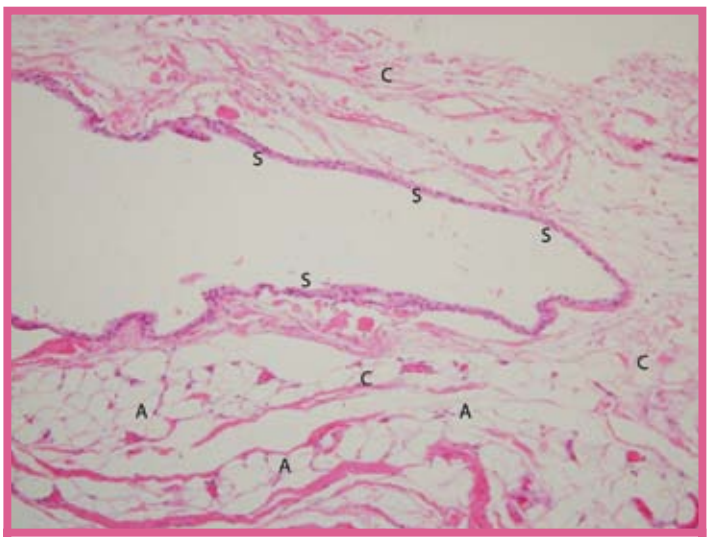


Bursa are traditionally described as synovium-lined fluid-filled sacks. This image illustrates not only the synovium (S), but also the underlying stroma that the synovium rests on. The stroma is composed on collagen fibres (C) and adipocytes (A). Subachromical bursa pathology has been demonstrated to be in the stroma as well as the synovium, thus therapies (physical and otherwise) need to bear this in mind. (H&E x100)

Assistant Professor Alex Scott is Chair of the Second International Scientific Tendinopathy Symposium, to be held on 27–29 September 2012 in Vancouver, Canada. Following the Symposium there will be a two-day clinical course led by Jill Cook, Professor in Musculoskeletal Health at Monash University and Bill Vicenzino, APAM, Professor in Sports Physiotherapy at the University of Queensland. For more information on these events, check the conference website at ists2012.com/.

Understanding GTPS

Greater trochanteric pain syndrome (GTPS) is a common, sometimes disabling, hip condition that affects older women in higher proportions than any other demographic. The aim of my thesis was to address a number of issues relating to this condition.

Reports that the diagnosis of GTPS is straightforward are misleading, with misdiagnosis and under-diagnosis frequently reported. A definitive set of diagnostic criteria has not been established. Other than being a woman over the age of 50, no risk factors have been established for the development of GTPS. In addition, despite the high prevalence of GTPS, the impact on quality of life and function has only been measured in people representing a narrow spectrum of the condition; there is no Australian data. Finally, although greater trochanteric pain syndrome is thought to be due to local tendon and bursa pathology, this has not been established.

A battery of clinical tests and functional outcome tools were used to address the above questions. Participants included a group with GTPS, a group with GTPS who underwent gluteal tendon reconstruction (GTR), a group with severe hip osteoarthritis (OA), and an asymptomatic group. The following paragraphs describe findings that should be helpful to clinicians.

The clinical diagnostic criteria for GTPS should be:

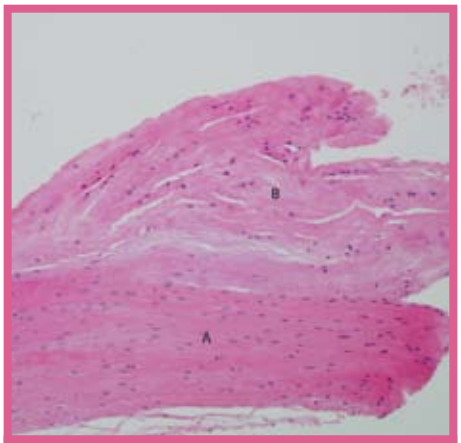
a) a history of lateral hip pain
b) having no difficulty manipulating shoes and socks on and off
c) pain on palpation of the greater trochanter
d) a FABER test that reproduces lateral hip pain. The reproduction of lateral hip pain with a FABER test provides an odds ratio of 43.7, a sensitivity of 82.9 per cent, a specificity of 90.0 per cent, a positive predicative value of 94.4 per cent and a negative predicative value of 72.0 per cent.

Of note, in people who can not localise their hip pain, comorbidities should be considered and further tests undertaken.

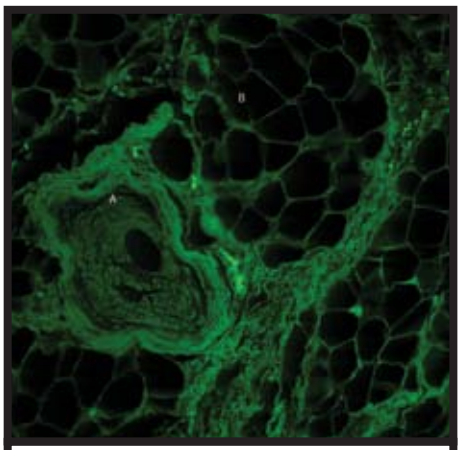
The risk factors for GTPS were confirmed to be multi-factorial, with bony and higher levels of adiposity implicated. The odds ratio of having a neck shaft angle of less than 134 degrees, relative to a pain-free group was highest for the GTR group at 3.33, 1.4 for the GTPS, and 0.85 for the OA group. The odds ratio of the GTR group relative to GTPS was 2.4 (Fearon et al 2012). It was found that women with GTPS appear wider due to increased gynoid adiposity.

People with GTPS were found to have levels of pain and disability almost indistinguishable from those experienced by people with severe hip osteoarthritis. This GTPS population had lower levels of full-time work and exercise participation than age-matched pain-free participants. These data highlight the role physiotherapists can play in assisting people with ongoing musculoskeletal disorders to manage their conditions, thus ensuring that levels of fitness and work participation are maintained.

Histopathologically, the tendon and bursa are both implicated in GTPS (Fearon et al 2010). Interadipose septae were seen within the bursa stroma, possibly explaining US reports of bursae thickening. To further the understanding of tendon and bursa pathology in general, and in relation to GTPS in particular, I was invited to the University of British Columbia and the Centre for Hip Health and Mobility, to work with Assistant Professor Alex Scott.



Tendinosis is characterised by degeneration of the tendon. Tendinosis frequently varies in severity within a small area of tendon; fairly normal tendon (A), and degenerative tendon (B). The first area (A) has slightly increased cell numbers, good collagen alignment and fairly normal tendon cells. The second area (B) has areas with increased and decreased tendon cell numbers, poor collagen alignment, and some tendon cells that are more rounded than others. Such variation can make the histological assessment of tendons difficult. (H&E x200) (Bursa x100)



Pacinian corpuscles (A) were identified within the bursa stroma (B). Pacinian corpuscles are mechanio-receptors which respond to mechanical pressure. This raises the intriguing possibility of the role of bursa in responding to pressure in addition to their role in reducing friction between tissue planes. (Second harmonic generation microscopy x650)

The current research is aimed at elucidating the relationship between bursitis and tendinopathy. Using the trochantic bursa and gluteal tendons as a model, I am examining the tissue morphology, inflammatory cell markers, neuropeptides and the regulation of matrix metalloproteinase. I am examining some cellular mechanisms in each of the tissues that likely result in some adverse interaction and/or result in further degeneration of the tissues.

Angie Fearon—B(App)Sc(Phty), M(Phty), PhD(MedSc)—has more than 25 years of clinical experience and has worked in both public and private practice, including as a partner in the latter. She is currently a Postdoctoral Research Fellow in the Physical Therapy Department at the University of British Columbia, Vancouver, Canada.

For a list of references email ngeditor@physiotherapy.asn.au.