monitoring tools; and providing continual interest, encouragement, and support for physical activity. Some of these behavioural strategies have been used in studies in which primary care doctors have been trained to deliver brief advice and counselling on physical activity, with encouraging results in the short term.[4 5] In one study, for example, a written goal oriented exercise prescription from general practitioners, in addition to verbal advice, was particularly effective in promoting increased physical activity over a six week period.[6] More discrepant results obtained from longer term multiple risk factor programmes, however, suggest that more intensive interventions may be needed to obtain longer term effects in at least some segments of the population. Such interventions could include the use of health educators and professionals in addition to the doctor. Health educators and other allied health professionals can provide a level of advice and counselling beyond that which doctors, constrained by time and similar barriers, are typically able to deliver.[7] One promising approach awaiting more extensive investigation involves using brief advice from the doctor as a means of setting the stage for physical activity change in conjunction with specific referral to other health care based or community based health educators or providers. In this way, the perceived credibility and authority of the doctor can be harnessed as a catalyst for change, while the very real time constraints facing many doctors are recognised.[8] The challenge remains to structure the referral network effectively such that patients will successfully follow through with the referral. To maximise the potential benefits of this type of referral network, continuing communication between the doctor and referral source is essential.

In addition, the studies targeting primary care providers have focused almost exclusively on doctors involved in family practice and internal medicine. Yet, other primary care specialties, such as paediatrics and obstetrics-gynaecology, reach important segments of the population for whom physical activity information and messages are particularly relevant. Future research should target the full range of primary care practice.

While face to face instruction and counselling for physical activity have traditionally been the norm in most countries, a growing scientific literature has underscored the utility of mediated channels for delivering physical activity advice and information in an efficient, effective, and potentially less costly fashion. For instance, in the United States, at least 13 randomised controlled investigations have systematically evaluated the use of telephone based physical activity advice and support, either in conjunction with or independent of advice from the doctor.[7 9 10] The telephone supervised physical activity approach has been shown to be effective in both older and younger adult populations, women as well as men, cardiac patients, older family carers of relatives with dementia, and overweight patients. It has been found to be effective in promoting physical activity of various types—for example, endurance, strength, flexibility, general conditioning—intensities—for example, moderate intensity exercise, more vigorous exercise—and formats—for example, home based, group based, combinations of home based and group based exercise. Telephone and similar mediated approaches allow both the health professional and the patient a level of convenience and flexibility that is often diminished or lacking in group based physical activity regimens.

In summary, to reach the public health goals on physical activity in the United Kingdom, United States, Australia, and other countries continued efforts to involve primary care providers and other health professionals as active facilitators of the physical activity message are strongly indicated. Primary care advice in conjunction with referral to appropriate community organisations may help to facilitate the long term increases in physical activity participation that are critical for health promotion and disease prevention. Telephone and other mediated approaches to physical activity promotion provide a promising avenue for programme delivery, in primary care as well as other community settings.

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Where is the pain coming from in tendinopathy? It may be biochemical, not only structural, in origin

Traditional dogma would have it that pain in tendinopathy arises through one of two mechanisms. Firstly, it may result from inflammation in "tendinitis". Secondly, it may be due to separation of collagen fibres in more severe forms of tendinopathy. The latter situation parallels the mechanism of pain with collagen separation after an acute grade I or II ligament injury (fig 1).

Despite the wide acceptance of these two classical models of pain production, a number of studies provide data inconsistent with either theory. Consider first the inflammation mechanism. Histopathological examination of surgical specimens from patients with chronic tendon pain are devoid of inflammatory cells.[1] This applies to tissue from the Achilles, patellar, lateral elbow, medial elbow, and rotator cuff tendons. Furthermore, prostaglandin E2 (a marker of the inflammatory process) is no more abundant in patients with Achilles tendon pain than in normal controls.[2]

Unfortunately, the collagen separation theory does not hold up under scrutiny either. The following five observations about pain and collagen in the patellar tendon are inexplicable. (*a*) Patients who have patellar tendon allograft anterior cruciate ligament reconstruction have minimal donor site knee pain, yet collagen has been excised. (*b*) Such patients are generally pain-free (and back at sport) despite the persistence of abnormal collagen for two or more years.[3 4] (*c*) Similarly, after open surgery for jumper's knee, the imaging appearance of the tendon—that is, collagen status—does not correlate consistently with knee pain.[5] (*d*) Patients with jumper's knee can also be treated by an arthroscopic debridement of the infrapatellar fat pad and the posterior border of the patellar tendon without operation on the collagen defect in the tendon itself.[6] (*e*) Large asymptomatic ultrasonographic hypoechoic regions (abnormal collagen) can be found in patellar tendons of some athletes who have never had a history of jumper's knee.[7 8]

Such discrepancy between collagen structure and pain is not confined to the patellar tendon. Patients with partial (non-perforated) rotator cuff tears were found to have more pain than those with complete perforations[9] despite the former having less collagen damage. Clearly there is more to tendon pain than discontinuity of collagen per se.

Nociceptors provide significant afferent pain pathways. In the knee, they are located in the retinaculum, fat pad, synovium, and periosteum,[10] and all these structures may play a role in the tendon pain pathway. Biochemical irritants may include extravasation of glycosamines, especially chondroitin sulphate,[11 12] from damaged tendon.

The five observations listed above can be explained with what we term a "biochemical" hypothesis (fig 2). We speculate that the pain of patellar tendinopathy is largely due to biochemical agents irritating nociceptors located in the fat pad immediately posterior to the patellar tendon. In 39 cadaver dissections of the proximal patellar tendon,[13] we consistently identified a thin layer of fat adherent to the posterior portion of the patellar tendon. In the corresponding tissue specimens from patients operated on for chronic jumper's knee, this fat tissue contained increased Alcian blue stain (and thus glycosaminoglycans), presumably leaked from the adjacent region of tendinosis.

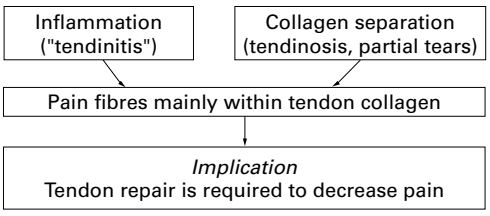

Figure 1 The classical "inflammatory" and "structural" tendon pain models.

To our knowledge, the key irritant biochemical agent has not yet been identified, and this presents a challenge for tendon biochemists. Using microdialysis, Alfredson recently identified an abnormal amount of the excitatory neurotransmitter, glutamate, in subjects with painful Achilles tendinopathy.[2] Until these histopathological and biochemical findings are correlated with some measure of pain, we can only speculate as to whether they are causative, or merely byproducts of nearby tendinosis.

Of interest, in the rotator cuff pain and pathology study quoted above,[9] collagen damage was inversely related to pain, but the presence of substance P (a nociceptive neurotransmitter) was significantly associated with pain. Nerve fibres immunoreactive to substance P were localised around vessels in the subacromial bursa and in the non-perforated rotator cuff.[9]

Although the data presented may suggest a biochemical cause of pain, other workers consider mechanical impingement of the fat pad as a cause of anterior knee pain. The Australian physiotherapist, Jenny McConnell, recognised fat pad impingement as a cause of anterior knee pain (not necessarily tendon pain) over 10 years ago. Johnson proposed that impingement caused the pain of patellar tendinopathy.[14] The infrapatellar fat pad is an extremely sensitive region[15] and contains a large number of nociceptors, but as tendon pain occurs at many anatomical sites, it does not appear logical that a structure related to only one tendon—that is, the patellar fat pad—would necessarily play a unique role in a problem as widespread as tendinopathy. Further, the clinical observation that the pain of jumper's knee does not disappear and may actually increase when palpation is performed with the knee in full extension would appear to argue more for a biochemical than a mechanical cause of pain in tendinopathy. Nevertheless, the jury requires more evidence.

If our biochemical hypothesis proves to have some validity, it would have significant clinical and research implications. In clinical management, the aim of treatment would be to modify the biochemical milieu, rather than to focus on reducing inflammation or necessarily augmenting collagen repair. Collagen repair may, of course, improve the biochemical milieu and thus explain why eccentric strengthening programmes can help.[16] Researchers would be encouraged to pursue a pharmaceutical approach focused on reducing the irritant (but not necessarily inflammatory) biochemical compounds around the tendon. Surgery may play a role through denervation. Thus, if sports medicine researchers collaborate with basic scientists who understand pain physiology, knowledge will be

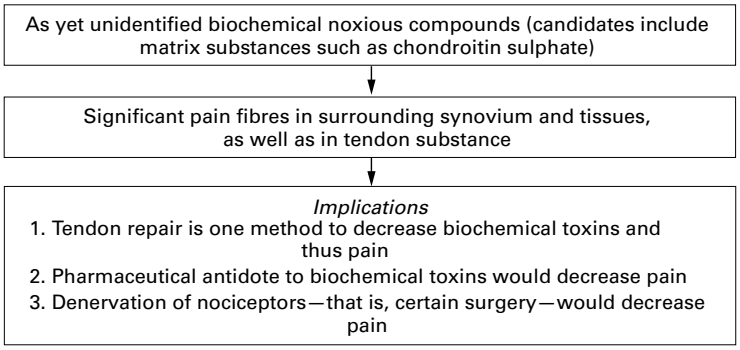

Figure 2 Contemporary "biochemical" tendon pain model.

advanced in both fields, and we will progress toward the goal of alleviating the pain of what is often structurally rather a trivial problem.

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$\dot{V}O_2$ slow component and performance in endurance sports

For almost 80 years, physiological studies have attempted to explain endurance performance and to develop ways of improving it by training. Performance for a runner can be represented by the relation of his/her personal power (velocity) to time to exhaustion (time limit).[1]

There are particular velocities that delineate intensity domains which are determined by oxygen uptake ($\dot{V}O_2$) and blood lactate response versus time.[2 3] We are going to use them to define the slow phase of $\dot{V}O_2$ kinetics $\dot{V}O_2$ slow component) which only appears during intense exercise.

A high range of work can be identified at which there is a sustained increase in blood lactate and a decrease in arterial pH with time. These responses decline back towards a baseline value. Oxygen uptake increases in a monoexponential way and stabilises at about 80% in high level marathon runners for at least an hour and a half of continuous exercise. After that time, it is possible for oxygen consumption to increase because of thermoregulatory constraints, and this increase is called the "$\dot{V}O_2$ drift". This intensity of exercise corresponds to the velocity that can be sustained during a marathon and is equal to about 80% of the velocity associated with $\dot{V}O_2MAX$ determined in an incremental test—that is, $v\dot{V}O_2MAX$.[4] During this type of exercise both lipids and carbohydrate are used as fuel.

At a higher intensity, the maximal lactate steady state occurs[5] when the rate of appearance of blood lactate equals the rate of its disappearance. $\dot{V}O_2$ stabilises after three minutes at about 85% $\dot{V}O_2MAX$. This corresponds to the highest velocity that an athlete can sustain for an hour (85% $v\dot{V}O_2MAX$ for a well trained endurance athlete); carbohydrate (and lactate even) is the main substrate for this exercise.

At a higher intensity, at about 90% $v\dot{V}O_2MAX$, the rate of appearance of blood lactate exceeds the rate of disappearance and therefore blood lactate increases. After the first monoexponential increase in $\dot{V}O_2$, there is a second increase after about three minutes which is defined as the $\dot{V}O_2$ slow component. $\dot{V}O_2$ reaches a delayed steady state which is higher than the $\dot{V}O_2$ requirement estimated from the relation between $\dot{V}O_2$ and moderate work rate. For instance, in this case the athlete can run at 90% $v\dot{V}O_2MAX$ and reaches and stabilises at 95% $\dot{V}O_2MAX$ at the sixth minute of exercise (time to exhaustion at this velocity being about 10–15 minutes). This corresponds to the so called "critical power" which is the vertical asymptote of the hyperbolic relation between power (velocity) and time.[6] Time limit at the critical velocity is reduced to less than 30 minutes because of rapid glycogen depletion.[7 8] The critical velocity is the highest velocity below its maximal level ($\dot{V}O_2MAX$) at which oxygen consumption can reach a steady state.

Above this critical velocity, during high intensity exercise, neither $\dot{V}O_2$ nor blood lactate can be stabilised, and both rise inexorably until fatigue ensues, at which point $\dot{V}O_2$ reaches its maximum value.[9]

The initial very small component (phase 1), resulting from a sudden change in the venous return in combination with a small change in the mixed venous gas tension, is not fitted into the following equation. In fact, the parameters for the oxygen uptake kinetics were obtained from a two component exponential model in which the first component accounted for the fast component (phase 2) and the second component accounted for the slow component (phase 3). The oxygen uptake kinetics are described as a function of time by the following equation[10]: $\dot{V}O_2(t) = A_0$ (baseline) + $A_1(1-e^{-(tTD_1)/\tau_1})$ (fast component) + $A_2(1-e^{-(tTD_2)/\tau_2})$] (slow component)
where A_0 is the resting baseline value, A_1 and A_2 are the amplitudes for the two components, τ_1 and τ_2 are the time constants for the two components, and TD_1 and TD_2 are the time delays from the onset of exercise for the two components.