

explained by the greater tendency to central deposition of fat in South Asian children.¹⁰ Important observations, such as those of Whincup et al and Patel et al, made on cross sectional data, need to be verified in cohort studies. But none of the many cardiovascular cohort studies in the United Kingdom can yield risk-outcome data by ethnic group.⁴

New risk factors—The third explanation is that specific risk factors, not yet established or discovered, may explain high risk. The search for a specific cause has led to many hypotheses, including the use of ghee and other cooking oils, subclinical hypothyroidism, central obesity, stress, racism, insulin resistance, a thrifty genotype, a thrifty phenotype, low vitamin C, high homocysteine, endothelial dysfunction, high levels of lipoprotein a, and other specific lipid abnormalities. No “South Asian cause” of coronary heart disease has been proved, though each new idea has diverted attention from established risk factors. The best studied hypothesis is that the high prevalence of insulin resistance, independent of diabetes, underlies the high rates of coronary heart disease in South Asians.¹ Rigorous tests of this hypothesis, based on prospective studies, are awaited, but Whincup et al provide data of interest on children. Though South Asian children were no more obese than those of European origin, fasting and 30 minute post load insulin were about 50% higher.

Competing causes—The fourth, rarely considered explanation, is that there are fewer competing causes of death in middle aged South Asians, particularly as cancer rates are comparatively low. Whincup et al do not touch on this concept.

Whincup et al have paved the way to paying more attention to young South Asians, mostly born in the United Kingdom. They show that if insulin and insulin resistance do turn out to be causally related to coronary heart disease in South Asians then preventive action will need to take place early. Simmons reported from New Zealand that Indian babies had less insulin in cord blood than European, Maori, and Pacific Islander babies.¹¹ Further studies are needed to corroborate these findings; to confirm that findings in Pakistanis apply to other South Asian groups—as is likely; and to establish exactly when the tendency to insulin resistance emerges and why. Even if insulin

resistance is not directly causative of coronary heart disease, it is predictive of diabetes, a key and highly prevalent risk factor in South Asians. This work emphasises that the prevention of diabetes must start in early life.

This study has policy and service implications. South Asians’ poor knowledge and understanding of coronary heart disease and diabetes are shocking, particularly in Bangladeshis and Pakistanis.¹² In addition to conveying effective and accurate messages about coronary heart disease prevention in adults we must weave in the key message that children are at risk. As all the established risk factors are important in South Asians, the health promotion challenge is formidable.

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- 1 McKeigue P, Sevak, L. *Coronary heart disease in South Asian communities*. London: Health Education Authority, 1994.
- 2 Yusuf S, Reddy S, Ounpuu S, Anand S. Global burden of cardiovascular diseases: part II: variations in cardiovascular disease by specific ethnic groups and geographic regions and prevention strategies. *Circulation* 2001;104:2855-64.
- 3 Whincup PH, Gilg J, Papacosta O, Seymour C, Miller GJ, Alberti KGMM, et al. Early evidence of ethnic differences in cardiovascular risk: cross sectional comparison of British South Asian and white children *BMJ* 2002;324:635-8.
- 4 Bhopal R. What is the risk of coronary heart disease in South Asians? A review of UK research. *J Pub Health Med* 2000;22:375-85.
- 5 Williams R, Bhopal R, Hunt K. Coronary risk in a British Punjabi population: comparative profile of non-biochemical factors. *Int J Epidemiol* 1994;23:28-37.
- 6 Bhopal R, Unwin N, White M, Yallop J, Walker L, Alberti KGM, et al. Heterogeneity of coronary heart disease risk factors in Indian, Pakistani, Bangladeshi and European origin populations: cross sectional study. *BMJ* 1999;319:215-20.
- 7 Joint Health Services Unit. *Health survey for England. The health of minority ethnic groups '99*. London: Stationery Office, 2001.
- 8 Bhatnagar D, Anand IS, Durrington PN, Patel DJ, Wander GS, Mackness MI, et al. “Coronary risk factors in people from the Indian subcontinent living in west London and their siblings in India. *Lancet* 1995;345:405-9.
- 9 Patel S, Unwin N, Bhopal R, White M, Harland J, Ayis SA, et al. A comparison of proxy measures of abdominal obesity in Chinese, European and South Asian adults. *Diabetic Med* 1999;16:853-60.
- 10 Peters J, Ulijaszek SJ. Population and sex differences in arm circumference among Indo-Pakistani children living in the East Midlands of Britain. *Ann Hum Biol* 1992;19:17-22.
- 11 Simmons D. Differences in umbilical cord insulin and birth weight in non-diabetic pregnancies of women from different ethnic groups in New Zealand. *Diabetologia* 1994;37:930-6.
- 12 Rankin, J, Bhopal R. Understanding of heart disease and diabetes in a South Asian community: cross sectional study testing the ‘snowball’ sample method. *Pub Health* 2001;115:253-60.

Time to abandon the “tendinitis” myth

Painful, overuse tendon conditions have a non-inflammatory pathology

Tendinitis such as that of the Achilles, lateral elbow, and rotator cuff tendons is a common presentation to family practitioners and various medical specialists.¹ Most currently practising general practitioners were taught, and many still believe, that patients who present with overuse tendinitis have a largely inflammatory condition and will benefit from anti-inflammatory medication. Unfortunately this dogma is deeply entrenched. Ten of 11 readily available sports medicine texts specifically recommend non-steroidal anti-inflammatory drugs for treating painful conditions like Achilles and patellar tendinitis

despite the lack of a biological rationale or clinical evidence for this approach.^{2 3}

Instead of adhering to the myths above, physicians should acknowledge that painful overuse tendon conditions have a non-inflammatory pathology. Light microscopy of patients operated on for tendon pain reveals collagen separation⁴⁻⁶—thin, frayed, and fragile tendon fibrils, separated from each other lengthwise and disrupted in cross section. There is an apparent increase in tenocytes with myofibroblastic differentiation (tendon repair cells) and classic inflammatory cells are usually absent.⁴ This is tendinosis and it was first

BMJ 2002;324:626-7

described 25 years ago,⁶ but this fundamental of musculoskeletal medicine has not yet replaced the tendinitis myth. Tendinosis is not merely a long term corollary of short term tendinitis. Animal studies show that within two to three weeks of tendon insult tendinosis is present and inflammatory cells are absent.⁷

A critical review of the role of various anti-inflammatory medications in soft tissue conditions found limited evidence of short term pain relief and no evidence of their effectiveness in providing even medium term clinical resolution of clearly diagnosed tendon disorders.² Laboratory studies have not shown a therapeutic role for these medications. Corticosteroid injections provide mixed results in relieving the pain of tendinopathy.⁸⁻⁹

If general practitioners, orthopaedic surgeons, and other members of the healthcare professions treating tendon disorders made a quantum shift from previous flawed teaching about overuse tendinitis and adopted these data there would be immediate ramifications. Nomenclature for the clinical presentation of tendon disorders would reflect the true histopathological basis underlying clinical presentation.¹⁰ The term tendinitis would rarely cross doctors' lips. Numerous authorities²⁻¹⁰ recommend the term tendinopathy (for example, Achilles tendinopathy) as this acknowledges that the condition is not tendinitis. We favour this term for clinical diagnosis. Most importantly, we must acknowledge, at least till contrary data appear, that anti-inflammatory pharmacotherapy does not provide significant long term benefit in tendinopathy.²⁻¹¹ Nevertheless, high quality randomised controlled trials are urgently needed to examine the long term effects of these medications on tendinopathy.

If general practitioners treating musculoskeletal conditions embraced the tendinopathy paradigm, it would provide patients with an accurate description of their condition. It would avoid inappropriate pharmacotherapy with its attendant costs and comorbidity. Furthermore, by accepting need to allow time for collagen turnover and remodelling inherent in the pathology of tendinosis, doctors would be free to provide patients with a realistic prognosis that better reflects the finding of prospective clinical studies.¹² These conditions take months rather than weeks to resolve.

Some pockets of the sports medicine, orthopaedics, and rheumatology specialties have adopted this

paradigm,²⁻⁴⁻¹⁰ but it must no longer remain within that cabal. It is time for medical educators to accept the irrefutable evidence that the term tendinitis must be abandoned to highlight a new perspective on tendon disorders. Adopting the tendinopathy paradigm is essential if general practitioners are to practise evidence based medicine. However, there remain many unanswered questions, particularly with respect to treatment.

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- 1 Bongers PM. The cost of shoulder pain at work. Variation in work tasks and good job opportunities are essential for prevention. *BMJ* 2001;322:64-5.
- 2 Almekinders LC, Temple JD. Etiology, diagnosis, and treatment of tendinitis: an analysis of the literature. *Med Sci Sports Exerc* 1998;30:1183-90.
- 3 Khan KM, Maffulli N. Tendinopathy: an Achilles' heel for athletes and clinicians. *Clin J Sport Med* 1998;8:151-4.
- 4 Khan KM, Cook JL, Bonar F, Harcourt P, Astrom M. Histopathology of common overuse tendon conditions: update and implications for clinical management. *Sports Med* 1999;27:393-408.
- 5 Jozsa L, Kannus P. *Human tendons*. Champaign, Illinois: Human Kinetics, 1997.
- 6 Puddu G, Ippolito E, Postacchini F. A classification of Achilles tendon disease. *Am J Sports Med* 1976;4:145-50.
- 7 Backman C, Boquist L, Friden J, Lorentzon R, Toolanen G. Chronic Achilles paratenonitis with tendinosis: an experimental model in the rabbit. *J Orthop Res* 1990;8:541-7.
- 8 Hay EM, Paterson SM, Lewis M, Hosie G, Croft P. Pragmatic randomised controlled trial of local corticosteroid injection and naproxen for treatment of lateral epicondylitis of elbow in primary care. *BMJ* 1999;319:964-8.
- 9 Stahl S, Kaufman T. The efficacy of an injection of steroids for medial epicondylitis. A prospective study of sixty elbows. *J Bone Joint Surg* 1997;79-A:1648-52.
- 10 Maffulli N, Khan KM, Puddu G. Overuse tendon conditions. Time to change a confusing terminology. *Arthroscopy* 1998;14:840-3.
- 11 Astrom M, Westlin N. No effect of piroxicam on achilles tendinopathy. A randomized study of 70 patients. *Acta Orthop Scand* 1992;63:631-4.
- 12 Paavola M, Kannus P, Paakkala T, Pasanen M, Jarvinen M. Long-term prognosis of patients with achilles tendinopathy. An observational 8-year follow-up study. *Am J Sports Med* 2000;28:634-42.

Deprofessionalising doctors?

The independence of the British medical profession is under unprecedented attack

A patient seeing a doctor professionally in the United Kingdom has expectations of professional conduct that far exceed what is expected of citizens generally or employees of most institutions. This sense of professionalism is important to patients as it motivates doctors. The underpinnings of that professionalism, established over 150 years, have in the last 150 days all been questioned.

The medical profession in the United Kingdom first emerged through the medical royal colleges in 1505. The 1858 Medical Act united the medical profession and, almost 150 years ago, created the General Medical Council—a structure through which the profession could develop an ethical code,¹ systematise education, and punish erring members. The council derives its authority from parliament; its membership includes