Lipids, adiposity and tendinopathy: is there a mechanistic link? Critical review

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ABSTRACT
Being overweight or obese is associated with an elevated risk of tendon pathology. However, for sportspeople the epidemiological data linking weight or adiposity on one hand, and risk of tendon pathology on the other, are less consistent. Indeed, the mechanistic links between diet, adiposity and tendon pathology remain largely unexamined. Recent studies have begun to examine the effects of dietary interventions on outcomes such as tendon biomechanics or pain. Oxidised low-density lipoprotein has been shown to accumulate in the tendon tissues of mice that eat a fatty diet and induce a pathological phenotype in human tendon cells. This paper addresses the current debate: is excessive body mass index (causing increased load and strain on tendon tissue) per se the underlying mechanism? Or do local or systemic influences of fat on tendons predispose to tendon pathology? This narrative review argues that excessive blood lipids may be an important avenue for clinical investigations.

INTRODUCTION
Minimising the impact of tendon pathology is an important clinical goal for those treating elite or recreational sportspeople. The same goal applies when prescribing exercise as medicine for weight reduction in sedentary individuals, who may be prone to acute or overuse injury when beginning a training programme.

Individualised load management strategies for patients undergoing rehabilitation for tendinopathy or tendon rupture (including identification and correction of biomechanical faults, and attention to training intensity and volume) are well recognised components of clinical practice. Although the load management strategy is not based on level 1 evidence, the approach emerged from clinical experience and close observation of patient characteristics associated with treatment success or failure. In a similar manner, Cook and co-workers reported their clinical impression that individuals with thicker waists were more likely to suffer from patellar tendinopathy. This impression was followed up with a small case–control study, which suggested that larger waist measurement may be a significant risk factor for patellar tendon pathology. This observation prompted further research into (A) the strength of this association in various populations that experience tendon pathology (including tendinopathy, tendon rupture and asymptomatic lesions identified on imaging) and (B) the underlying mechanism. This research can help the clinician decide whether adiposity needs to be accounted for as part of a holistic tendon injury management programme in active individuals (most of whom have body mass index (BMI) and lipid values within normal limits).

In this paper, we first examine key epidemiological studies that have been conducted in primarily sedentary populations. Although tendon pathology may present quite differently in the general population than in sportspeople, there appear to be some common themes. This is followed by a review of the available epidemiological evidence in sportspeople. Finally, we highlight potential mechanisms underlying the observed associations between adiposity and tendon pathology and identify some directions for future research.

TERMINOLOGY
Pathology may be defined as ‘The anatomic or functional manifestations of a disease’. It may be symptomatic or asymptomatic. Tendon pain, rupture and abnormal imaging may share an underlying pathology (tendinosis), but this cannot be assumed in the absence of histological samples. In this paper, we therefore use the term ‘tendon pathology’ to refer to tendinopathy, rupture or asymptomatic lesions.

EPIDEMIOLOGICAL EVIDENCE—POPULATION DATA
One of the largest studies that examined the risk factors of tendinopathy was a 2014 case–control study conducted in the UK. Five thousand individuals in the general population who had received a diagnosis of rotator cuff disease (tendinopathy, bursitis, impingement, tears and calcific tendinopathy) were randomly selected from a health registry. This resulted in a sample with equal sex distribution (ie, gender was not a risk factor) and the patients were matched by age, sex and general practice with non-rotator cuff cases from the same registry. The average BMI of people with rotator cuff disease (26.5 kg/m²) was significantly higher than that of the control group (25.9 kg/m², p<0.01). Furthermore, the incidence of rotator cuff disease was higher in those who were overweight (BMI 25.1–30 kg/m²) or obese (BMI 30.1–40 kg/m²). BMI data were only available for 67% of those with tendon pathology and 61% of controls.

In this study, people who consulted a doctor more frequently (as the overweight and obese do) were more likely to receive a diagnosis of rotator cuff disease. Is it the case that people who are overweight or obese are more likely to receive a diagnosis of tendon pathology because they are more frequently in contact with doctors? Or is it that they actually experience more tendon pathology, and are also in contact with doctors about other problems (in which case, controlling for

A longitudinal study also examined, reported. In 81 healthy individuals who were either runners or non-runners with no history of Achilles tendon pain, sonographic abnormalities (indicating the presence of asymptomatic tendon pathology) were more frequently observed in the Achilles tendons of overweight sedentary individuals (24% vs 8%). In this study, the Achilles tendons of overweight sedentary individuals were thicker than the tendons of normal weight sedentary individuals (5.2 vs 4.4 mm, p=0.002), and overweight runners with sonographic alterations had a higher body weight than overweight runners without sonographic abnormalities. Frey and Zamora surveyed 1411 patients in an orthopaedic foot and ankle clinic and found that a BMI $>25$ kg/m² significantly increased the risk (OR 1.9, 95% CI 1.39 to 2.66) of tendinopathy (Achilles, posterior tibial or peroneal). Similar findings, but specifically with regard to the increased risk of Achilles tendinopathy in overweight individuals, were reported in two other case–control studies. Finally, MRI evidence of patellar tendon pathology is associated with a history of elevated BMI or elevated body weight.

**Epidemiological evidence—sportspeople**

Epidemiological evidence among sportspeople is scarcer than that for the general population. A Union of European Football Associations (UEFA) study tracked Achilles tendon pathology (tendinopathy or rupture) among 1743 male professional footballers. There was no difference in BMI between those who did or did not experience an Achilles tendon injury (23.6±1.3 vs 23.4±1.4 kg/m² respectively, p=0.092), although waist measurements were not reported. The findings were similar in two smaller studies in elite-level volleyball players in that BMI was not related to the presence of sonographic patellar tendon pathology or to the development of symptomatic tendinopathy. However, in the former study, waist circumference was measured, and was a significant risk factor: those with a waist circumference of 83 cm or greater had a 74% chance of patellar tendon pathology, compared with 15% in those with <83 cm waistline. The authors speculated that, in sportspeople, waist circumference may be a better predictor of tendon pathology than BMI per se, but this finding was not confirmed by Visnes and Bahr.

**Epidemiological evidence—systematic review and meta-analysis**

Gaida et al conducted a systematic review of observational studies and included all studies that compared adiposity between individuals with or without tendon pathology. Twenty-eight studies including 19 949 individuals were analysed. The included population was extremely diverse, including asymptomatic cases (defined by imaging), as well as tendinopathy and rupture cases. The authors concluded that elevated adiposity is significantly associated with tendon pathology. Indeed, 81% of studies that recruited patients with tendon pathology (ie, people presenting to a healthcare practitioner for management of tendon pain) indicated a positive association of tendon pathology with adiposity. The authors recommended further research to determine whether interventions to reduce adiposity would improve the results of treatment or injury prevention.

**Evidence regarding mechanical load as a mechanism linking adiposity and tendon pathology**

Does increased body weight predispose to pathology because sportspeople subject their tendons to high or, arguably, excessive loads? There is limited evidence to address this question for upper as well as lower extremity tendons. However, the anatomical and functional differences between upper and lower limb tendons make it difficult to determine the importance of similarities and differences in the data across population and athletic cohorts.

Garner et al examined the impact of body composition on the magnitude moment arms at the shoulder and elbow during throwing. There was a significant relationship between increasing fat content in the arm, and increasing magnitude of injury-related throwing mechanics. Given that the shoulder tendons are frequently injured in sportspeople who throw, a case–control study in a larger population could shed further light on this potential mechanism of injury. One could argue that a heavier arm leads to higher loads through the rotator cuff tendons, however, this has not been directly demonstrated and further research would be required to confirm or refute this hypothesis. It would also be of interest to examine injury-related...
mechanics in a variety of other tasks of relevance to a sedentary population (eg, lifting, reaching). Gupta et al. compared the scapular kinematics in individuals of high (>27 kg/m²) or low (<23 kg/m²) BMI in 20 individuals. Those with higher BMI demonstrated altered scapular kinematics (increased scapular upward rotation), which was suggested to be a strategy to compensate for increased arm mass.

With regard to the lower extremity, Wearing et al. examined the structure (ultrasound imaging) and biomechanical response of the Achilles tendons in individuals of low (<23 kg/m²) or high (>25 kg/m²) BMI. The Achilles tendons of the high BMI group were on average 12% thicker, and this figure was used by the authors to estimate that the cross-sectional area (CSA) of the Achilles tendon was a remarkable 38% larger for overweight individuals. This increased CSA was further estimated to be capable of offsetting (compensating for) the increased tendon load that results from increased body weight. In other words, increased body weight may act as an adaptive stimulus capable of increasing tendon CSA, as has been shown with resistance training programmes. However, the transverse strains of the Achilles tendon were reduced in those with elevated BMI, suggesting that the collagenous matrix was not only larger, but abnormal in structure or composition. Therefore, the observed increase in tendon CSA in individuals with high BMI may represent a pathological as opposed to adaptive response. However, it is unclear whether this response is due to increased load through the tendon (due to increased body weight), or due to a systemic influence.

When interpreting the findings of epidemiological studies, one has to remember that the OR represents a statistical association, not necessarily a causal one. Thus, increased risk for development of tendon pathology with increased BMI or waist girth indicates that the variables (tendon pathology and BMI) are associated, despite the multitude of other genetic and nongenetic risk factors. The epidemiological assessments discussed above provide some impetus to further focus investigations into how the risk is manifested and why it occurs.

**EVIDENCE FOR A SYSTEMIC LINK BETWEEN ADIPOSITY AND TENDON PATHOLOGY—ARE BLOOD LIPIDS THE MISSING LINK?**

Given that the case–control studies reviewed above have generally been consistent in identifying that the increased BMI or waist circumference/waist–hip ratio are risk factors for tendon pathology, it is reasonable to ask whether there may be an ‘upstream’ systemic influence of excessive blood lipid. Indeed, inherited hyperlipidaemia is a well-known cause of tendon pathology, and it is also possible that diet-related or age-related hyperlipidaemia, or mildly elevated total cholesterol, may predispose to tendon pathology in otherwise healthy individuals.

Total cholesterol levels within the blood stream are influenced by diet, and by the efficiency with which cholesterol is transported among tissues by cholesterol-rich lipoproteins. Increases in low-density lipoprotein (LDL) or decreases in high-density lipoprotein (HDL) are associated with the accumulation of cholesterol in non-liver tissues. LDLs are primarily responsible for transporting cholesterol between the liver and the circulation, and HDLs are responsible for returning cholesterol to the liver. A key stage in the initiation and progression of atherosclerosis, which has recently come into the spotlight, is the oxidation of LDL. LDL oxidation is thought to occur in the extravascular tissue spaces, which are less protected by antioxidants than in the plasma. Oxidised LDL (oxLDL) has pathogenic effects on a variety of cell types including smooth muscle cells, macrophages and endothelial cells. OxLDL levels are elevated in the obese and because of its pathogenic role it has been proposed as a potential marker for atherosclerosis risk or severity. The liver is reported to be responsible for the removal of oxLDL from the circulation.

Mathiak et al. measured the total cholesterol level of 31/41 patients (average age 40.2 years, range 24–78) who had undergone surgical repair of the ruptured Achilles tendon. Remarkably, 83% of those sampled had elevated total cholesterol levels (>200 mg/dL) and were referred for further medical management. However, these findings are in contrast with a study by Haacke and Parwaresch, who measured lipid levels in 59 patients with Achilles tendon rupture. In this study, only 15% of those sampled (average age 32 years, range 15–42) had increased total cholesterol levels. In those with increased cholesterol, histopathology demonstrated evidence of lipid accumulation within the Achilles tendons; this finding was not present in the 50 cases with normal cholesterol levels, although the blinding and observation procedures were not detailed. Recent findings by Ozgur et al. are supportive of observations by Mathiak et al., and this study had the advantage of including a control group (figure 1). Of the 47 Achilles tendon rupture cases, 74.5% demonstrated elevated total cholesterol and 70.2% had elevated LDL, compared to 25.5% and 29.8% in controls, respectively. The blood sampling varied between the case and control groups; the patient group was sampled 6–8 h following rupture, while the control group was fasting. However, fasting before lipid testing has been shown to result in less than 2% variation for total cholesterol and less than 10% for LDL. Thus, preliminary data suggest that elevated total cholesterol or LDL may be a more frequent finding among patients with Achilles tendinopathy compared with controls, however, definitive data are not yet available.

Two studies of the rotator cuff present further preliminary data on this potential link between blood lipids and tendon pathology. Longo et al. measured serum triglycerides and total cholesterol in 240 patients, 120 of whom were undergoing arthroscopic rotator cuff repair and 120 of whom were controls (patients undergoing meniscectomy). In the rotator cuff...
patients, 69% of patients had elevated total cholesterol (>5.2 mmol/L) compared with 66% of those undergoing meniscectomy. In contrast, Aboud et al\textsuperscript{36} compared 74 rotator cuff surgical candidates with non-cuff shoulder pain patients and found 63% of those with rotator cuff problems had elevated total cholesterol (>240 mg/dL) compared with 28% in the control group.

With regard to non-surgical cases of tendinopathy, the results are also preliminary. Gaida et al\textsuperscript{28} measured serum lipids in 60 members of the general population with chronic painful midportion Achilles tendinopathy and in 60 age-matched and BMI-matched controls, and found evidence of underlying dislipidemia including a significant elevation of triglyceride levels. In a population-based study of 5871 Finnish people, Shiri et al\textsuperscript{41} found no significant difference in LDL or HDL levels among those with or without chronic lateral or medial epicondylitis.

**EVIDENCE FOR A LINK BETWEEN BLOOD GLUCOSE AND TENDON RUPTURE OR TENDINOPATHY?**

Elevated blood glucose levels are well known to have a profound influence on tendon health, and this topic has recently been reviewed elsewhere.\textsuperscript{37} Certainly, the link between diagnosed diabetes mellitus and poor tendon health is well established. One could ask whether blood glucose levels in the prediabetic overweight individual could already predispose to tendon injury, and whether this may be another mechanism behind the association of BMI and tendon pathology. This question has not been addressed to our knowledge. Arguing against this line of reasoning, in the large population-based studies cited above, BMI was a stronger risk factor for tendinopathy than a diagnosis of diabetes mellitus.

**EFFECT OF DIETARY INTERVENTIONS TO MITIGATE/PREVENT TENDINOPATHY**

Given that case–control studies have identified that increased BMI or waist circumference and elevated blood lipids are risk factors for tendon pathology, it is reasonable to ask whether an intervention to reduce these would have any effect on the risk of developing tendinopathy or tendon rupture. To the best of our knowledge, this question has hardly been addressed. A recent study in mice demonstrated that mice that were fed a high-fat diet experienced a drop in Achilles tendon modulus compared with mice on a normal diet, and neither exercise nor administration of a nutritional supplement (branched chain amino acid) were able to mitigate this decline in biomechanical function.\textsuperscript{38} Szczurko et al\textsuperscript{39} conducted a patient-blinded and assessor-blinded randomised controlled trial to examine the impact of a naturopathic intervention on rotator cuff tendinopathy; the intervention included individualised dietary counselling, acupuncture and prescribing of a natural supplement (phlo-zenzym). Patients who received the naturopathic intervention experienced a greater improvement in shoulder function than those who received placebo or range of motion exercises. The impact of the dietary intervention in this clinical trial is difficult to gauge, and the exercises used in the trial are not consistent with those with proven clinical efficacy.\textsuperscript{40}

**EFFECT OF STATINS AND DEVELOPMENT OF TENDON PATHOLOGY**

Statins are a class of drugs designed to treat hyperlipidaemia by inhibiting the enzyme HMG-coenzyme A reductase (discussed in ref. 41). While effective in many patients with elevated plasma lipids, they are not without their side effects (discussed in refs. 41 and 42). Individuals with familial hyperlipidaemias are at risk for tendon xanthomas (discussed in refs. 27 and 42), which can impair function. When placed on statins, tendon thickening and the xanthomas are reported to regress.\textsuperscript{27, 43–44} However, a small subset of patients treated with statins has been reported to develop tendinopathies or tendon ruptures.\textsuperscript{41, 42, 45–47}

The mechanism(s) by which statins enhance the risk for tendon ruptures in this subset of patients is not known. However, recently de Oliveira et al\textsuperscript{41} described biochemical changes in the Achilles tendons of rats chronically treated with statins. They report that in the rat studies, an imbalance between catabolic and anabolic activities in the treated tendons was detected. Thus, alterations in metabolism (eg, increased MMP expression and altered matrix expression) were associated with statin treatment.

Such findings raise two important points: (1) questions regarding drug use, particularly of statins, should be included in all clinical and epidemiological studies of tendinopathy and (2) further studies on the influence of statins on tendon repair after injury (eg, tendinopathy, surgery, rupture) should be undertaken to better understand the basis for risk associated with drug treatment.

**INTEGRATED MODEL—THE SYSTEMIC INFLUENCE OF LIPID ON TENDONS AND INFLUENCE ON BIOMECHANICAL FUNCTION**

We recently conducted a study on mice to examine the influences of localised lipid (within or around the tendon tissue) versus systemic lipid (hypercholesterolaemia) on tendon biomechanical properties.\textsuperscript{48} Two strains of mice (normal mice or mice predisposed to developing hypercholesterolaemia, the apo-lipoprotein E knockout (ApoE-KO) model) were fed a normal diet, or a high-fat diet. Interestingly, the accumulation of adipose tissue around tendons occurred in normal mice that were fed a high-fat diet, and their tendons demonstrated a significant reduction in failure load and stress compared with mice on a normal diet. However, an even larger reduction in failure load and stress occurred in the tendons of ApoE-KO mice (particularly when combined with a high-fat diet), even though these mice (A) did not accumulate significant amounts of peritendinous adipose tissue and (B) gained less weight than their normal counterparts. This constellation of findings argues in favour of a systemic, rather than local, influence of dietary lipid or its byproducts on tendon health. This finding is in keeping with data demonstrating impaired tendon healing in a high-fat diet-induced mouse model of obesity and type 2 diabetes.\textsuperscript{39} Furthermore, when we applied oxLDL at physiological levels to human tenocytes, gene expression was altered—the tenocytes adopted an abnormal, proliferative behaviour, reducing their collagen I expression and increasing MMP expression.

Taken together, we propose the following (figure 2). Excessive dietary lipid can do either (or both) of the following: (1) cause accumulation of body weight and/or abdominal adiposity, which places excessive mechanical load on tendon tissues or (2) cause dislipidemia, which exerts a systemic biological effect on tenocytes, thereby weakening the tendons. In the sedentary population more at risk of increasing BMI, either or both mechanisms may be dominant. Excessive load through a tendon previously weakened by metabolically disturbed tenocytes could predispose to injury. In athletic populations with normal (or low) body weight, the second mechanism may play more of a role. Both mechanisms could also interact with all of the other known biomechanical and non-biomechanical risk factors already identified for tendon pathology, such as age, hormonal status, genetics and others.\textsuperscript{10–32}
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group could be an impediment to healthy lifestyle change. In

CONCLUSION
The epidemiology of tendon injuries (ruptures and tendinopa-
thy) has highlighted the association between BMI or adiposity
and the risk of pathology. A potential role of dislipidemia in
predisposing to tendon pathology has been identified in the
general population. Animal studies suggest that dislipidemia
may directly and negatively influence tendons, but this mechan-
ism needs to be confirmed in humans.

Given that exercise is a common prescription for overweight
individuals, the increased risk of tendon injury in this patient
group could be an impediment to healthy lifestyle change. In
the future, larger scale epidemiological studies in a variety of
sedentary and athletic populations could account better and
more specifically for the identified potential risk factors dis-
cussed in this paper (adiposity, diet, blood lipids) to confirm or
refute the hypotheses presented here and, in the future, lead to
holistic exercise prescriptions with consideration for the under-
lying health of musculotendinous tissues.

What are the new findings?

- Large epidemiological studies in the general population
  support the existence of a relationship between body mass
  index (or body weight or waist circumference) and the
  occurrence of tendon pathology, but this relation has not
  been observed in more elite athletic groups.
- There is evidence for a mechanical effect (increased load
  and a systemic effect (eg, effects of circulating lipids), but
  the relative contributions of these two mechanisms remains
  preliminary and hypothetical.
- Data from animal studies suggest that a high-fat diet
  negatively influences tendon health.

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A number of studies have examined the relationship between obesity and musculoskeletal foot disorders. For example, Gaida et al. (2013) found that body mass index (BMI) was positively associated with Achilles tendon rupture. In a cross-sectional study of 1090 athletes, Abat et al. (2012) reported that BMI was significantly higher in athletes who had experienced an Achilles tendon rupture compared to those who had not. Similarly, Scott et al. (2014) reported that BMI was a significant predictor of Achilles tendonitis in a retrospective analysis of 111 athletes with Achilles tendon injuries. These studies suggest that obesity may increase the risk of musculoskeletal foot disorders, particularly Achilles tendon injuries.

In addition to BMI, other obesity-related risk factors such as abdominal obesity and central adiposity have been associated with musculoskeletal foot disorders. For example, a case control study by Scott et al. (2014) found that waist circumference was a significant predictor of Achilles tendonitis. Similarly, a cross-sectional study by Frey et al. (2017) found that abdominal obesity was associated with an increased risk of Achilles tendon rupture.

The association between obesity and musculoskeletal foot disorders is likely due to the increased mechanical stress placed on the musculoskeletal system as a result of increased body weight. Obesity is associated with increased body mass index (BMI) and central adiposity, which can lead to increased mechanical stress on the musculoskeletal system, particularly the foot and ankle. This increased mechanical stress can lead to musculoskeletal injuries such as Achilles tendonitis and Achilles tendon rupture.

In conclusion, obesity is a significant risk factor for musculoskeletal foot disorders, particularly Achilles tendon injuries. Further research is needed to understand the mechanisms by which obesity increases the risk of musculoskeletal foot disorders and to develop interventions to prevent these injuries.
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