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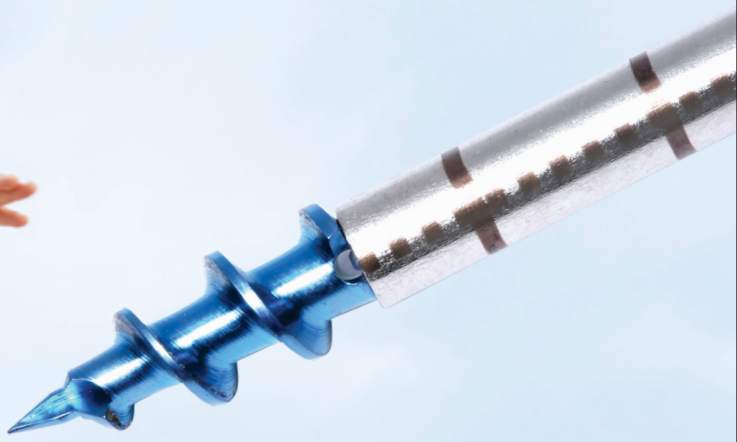

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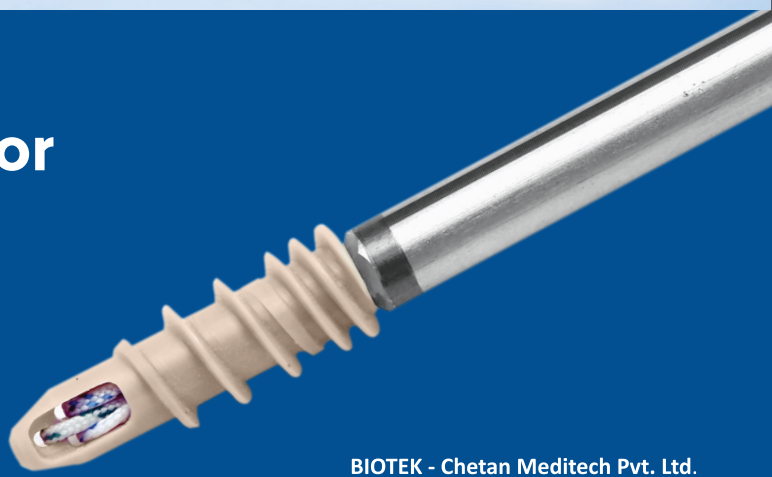

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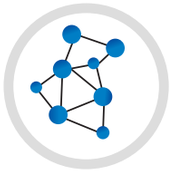

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*Journal of Arthroscopy and Joint Surgery* (JAJS) is committed to bring forth scientific manuscripts in the form of original research articles, current concept reviews, meta-analyses, case reports and letters to the editor. The focus of the Journal is to present wide-ranging, multi-disciplinary perspectives on the problems of the joints that are amenable with Arthroscopy and Arthroplasty. Though Arthroscopy and Arthroplasty entail surgical procedures, the Journal shall not restrict itself to these purely surgical procedures and will also encompass pharmacological, rehabilitative and physical measures that can prevent or postpone the execution of a surgical procedure. The Journal will also publish scientific research related to tissues other than joints that would ultimately have an effect on the joint function.

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Editorial

# Are we Sheep or are we Shepherds?

Knee surgery is one of the fastest advancing fields in Orthopaedic surgery. However, because we don't have the ability to replace like for like, i.e. true anatomical parts our researchers continue to search for the holy grail of "Anatomic Reconstruction" all the while knowing that it is, at this stage, not possible.

I am referring to the incredible number of research papers published on Anterior Cruciate Ligament Reconstruction. "ACL" if put into a search engine will produce more information than almost any other orthopaedic topic.

Why is this? Surely the reason is two fold:

1) ACL injury rate is increasing dramatically as sports participation increases around the world. Over 200,000 ACL reconstructions are done annually in the USA alone. And big profile sport is a massive business. Just look at the earnings of IPL stars; British Soccer players and American Football gladiators.
2) The second reason is that this ligament has actually "got our number"!

We just cannot make it like the creator designed it, plus we cannot always properly diagnose or correct secondary damage that occurred at the time of acute or chronic injury.

Yet the search goes on. Patellar tendon was an early graft used to reconstruct this ligament.[1] Because the results were not so good vascularized grafts were tried.[2] This didn't yield a normal knee and sometimes the complications were significant (arthrofibrosis); so hamstrings were used. Quadriceps tendon is now coming into vogue. Prosthetics and allograft have failed to solve the problem.

Having found no great improvement towards normality with different grafts we started looking at where we placed the graft. Maybe we had it in the wrong place? This seems surprising because the attachments of the natural ACL are quite large and well defined anatomically. Many studies going back decades have taught us this anatomy. Then when "new" anatomic placement didn't seem to make a difference we looked at the anatomy again and re-discovered the "double bundle" nature of the natural ACL. So there was a stampede to produce the best double bundle operation with many techniques on how to fix the various bundles and at what degree of knee flexion. The Pittsburgh group insisted on fixing the Antero-Medial (A-M) bundle in 60° flexion and the Postero-Lateral (P-L) bundle near full extension because their laboratory studies indicated that these were the positions that these bundles took most load.[3] But if one stepped back and looked at what we had been doing when reconstructing mainly the A-M bundle when doing single bundle reconstructions; and we remembered the work of Rosenberg, and that of Larson and Seidles on isometry,[4] one would have realized that their A-M graft with its tunnel position would (in most patients) tighten quite significantly going into extension if fixed at 60°.

It took a number of years and many failed operations of this technique which was "sold" to the world, to realize the error of this fixation position. Double bundle grafts are now being fixed completely differently (and differently by different surgeons as well). And more literature is coming out to again show no benefits to our patients. In amongst this double bundle phase the so-called "anatomic" single bundle femoral tunnel concept developed. We were told if you put a single bundle in the centre of the ACL attachment, thus having part of the tunnel covering the AM bundle and part the PL bundle we would get better results with more normal kinematics. Firstly this is such bad terminology. There are lots of places that the femoral tunnel could be placed and be "anatomic" all within the footprint. Secondly a number of researchers started reporting higher early re-rupture rates.

This tells us that we really are no closer to solving the ACL enigma.

What we are realizing is that there are other structures (e.g. Antero lateral ligament called the ALL and the ITB) that play a role in the stabilization of the knee against rotatory moments. And maybe it is dawning on us (not surprisingly) that different individuals will have ACLs and peripheral structures that have a greater or lesser effect in this function. That is, there may be ACL dominant knees that are very lax/unstable with a primary "isolated" ACL injury and others that remain stable with the same injury (I call "periphery dominant" knees). The latter being because of the contribution of secondary stabilizers which in that individual play a prominent role.

If we truly want to reconstruct the ACL (Collins Concise Dictionary: "Reconstruct: to build up again-something in its **original** form"), we need to look more closely at how the various fascicles of the bundles interact recruiting fibres as the force and position of the knee requires under load. We need to better appreciate the twisting arrangement of the fibres and bundles which also plays a role in different degrees of flexion. Because only by re-creating this highly complex anatomical arrangement will we be able to truly reconstruct the ACL and by so doing restore the knee to normal kinematics.

We need to learn to differentiate between the different patient types as to who might need just ACL surgery and who might need lateral surgery as well. And even in this lateral surgery and in the anatomy of the structures we are "fixing" there is discord amongst the surgical fraternity.

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So maybe instead of going around in circles and trying to outdo nature we should admit that at this time we cannot "make it like it was". We should heed the studies of people like Andy Williams who has shown that a single bundle A-M graft with a central tibial tunnel and a "high and deep" femoral tunnel (the position that the majority of the thickest AM fibres attach to), still gives the best outcomes[5]; That we recognize the incorrectly termed "anatomic tunnel", that which lies between the centre of attachments of the A-M and P-L bundles, gives rise to higher failures (which is understandable if one looks at the isometry of this bundle- ....tightens significantly more than the A-M bundle going into extension and so will see more load during the healing phase)[6]; That we need to recruit nature, not fight her, by allowing enough time for healing before returning the gladiator to the arena. And it is not just the graft that has to heal, but also the bony injury, thus allowing "joint homeostasis" as described by Scott Dye.[7]

So I would appeal to surgeons to use proven techniques based on careful examination and ellucidation of the injury, especially the secondary structures, and stick to a technique and graft source that gives predictable, good results with low morbidity. That the surgery (technique and graft choice) should be personalized to the patient being treated. Those that want to push the boundaries should more carefully and for a longer time period record their outcomes, and only when after significant time they can prove that new methods, techniques or surgeries will make a difference, then present these to the orthopaedic community so that changes made will positively affect our patients. There have been too many changes in a short period of time with no evidence of improvement, but sadly evidence of increased failure, costs and morbidity in some instances.

So be a Shepherd unto your patients and not a sheep to the pioneers. Let them find the promised land, not promise us they are finding it!

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Review article

# How lipids hurt tendons: Current understanding

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ABSTRACT

Hyperlipidemia has profound effects on tendon substance and mechanical properties. The common misconception is that hyperlipidemia manifests only as tendon xanthomas. Fatty streaks and signal changes are much more common than xanthomas, which may be considered as the end product of organized hyperlipidemic changes. The composition of xanthomas and the cause of lipid-mediated damage to tendons are discussed.



## 1. Introduction

The effect of lipids on tendon structure and healing is one of the most interesting fields of research. Hyperlipidemia has been known for many years to be a risk factor for tendon disorders, and conversely, tendon xanthomas are believed to be a risk factor for cardiovascular disease. Hyperlipidemia has been hypothesized to affect approximately 17% of the US population 20 years and older (National Center for Health Statistics 2006).[1] With an ever-increasing hyperlipidemic population the role of high lipid levels on tendon structure and healing is becoming clearer. There is still a quantum of work that needs to be done to fully understand that this topic, however we present the current state of knowledge in this review.

## 2. What are tendon xanthomas?

Tendon xanthomas are the classic findings of hyperlipidemia and are associated with a 3 times higher risk of cardiovascular disease among hyperlipidemic patients.[2]

Tendon xanthomas are accumulations of collagen and macrophages which contain cholesterol esters (foam cells).[3] Common locations are the Achilles tendon, wrist tendons (especially extensor) and elbow tendons. The main characteristic of tendinous xanthomas is the exceptionally high concentration in free and total cholesterol.[4] Tendon xanthomas are usually accompanied by an increase in lipid size, not only due to the xanthoma itself, but also the surrounding inflammatory reaction and edema.

The major constituent of tendon xanthoma are lipids (33% of dry weight) and collagen (24% of dry weight).[5] The lipid composition is typically 55% free cholesterol, 28% cholesterol esters and 13% phospholipids. This is similar to adult atherosclerotic lesions or fatty streaks.[4] Staining sections have revealed that unesterified cholesterol is predominantly extracellular whereas esterified cholesterol is both intra and extracellular.[6,7]

There appears to be rapid and total exchange of cholesterol between blood plasma and the xanthoma, as demonstrated by radionuclide experiment, suggesting that the xanthoma originates secondary to blood lipid levels rather than local lipid levels.[8] Within the lesion itself there is active uptake of LDL by the macrophages within the xanthoma.[9]

LDL is believed to be trapped within the collagen and glycosaminoglycan portion of the tendon ECM. Here it can become oxidized to oxLDL on contact with macrophages which then take it up intracellularly and form foam cells.[10,11]

## 3. Why do xanthomas form?

The short answer here is hyperlipidemia. However, not all hyperlipidemics have tendon xanthomas and vice versa. Recent research has found that only a subset of hyperlipidemic patients

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manifest with xanthomas. Xanthoma formation appears to be linked to genetic variation in specific genes for hypercholesterolemia. Oosterveer et al. (2010) found that among their 1208 hyperlipidemia patients, there was significant association between tendon xanthoma and genetic variation in the reverse cholesterol transport (RCT) and LDL oxidation genes which are the pathophysiological basis for atherosclerosis.[12]

## 4. Does hyperlipidemia manifest only as xanthoma?

No. In a MR based study on Achilles tendon properties among hyperlipidemic patients, Dussault et al. (1995) found that 92% of patients had abnormal signal changes within the tendon, whereas 73% had increased width of tendon itself. The abnormal signal finding was a diffuse stippled pattern with many low-signal round structures of equal size surrounded by high signal material on all pulse sequences.[13] Interestingly, in only 30% of patients did the signal changes appear like xanthoma, suggesting that there is more happening at the molecular level within the tendon substance, than simple xanthoma formation.

Thus fatty streaks and signal changes are probably a precursor to xanthoma formation. Xanthoma can be viewed as the end result of organized local hyperlipidemia. Clinical studies have noted a link among Achilles tendon thickness, hyperlipidemia and intima-media thickness of the carotid artery, suggesting thickening of the Achilles tendon as a potential indicator of atherosclerosis.[14] Thus, rather than xanthoma itself, any fatty change within the tendon proper may be an indicator of hyperlipidemia.

## 5. Is the type of lipid important?

Yes. It appears that the type of lipid is also important. In their study of 47 patients with Achilles tendon ruptures, Ozgurtas et al. (2003) found that Total cholesterol, TG, VLDL and LDL concentrations were significantly higher in the tear group than control group, whereas the HDL concentration was significantly lower in the tear group.[15] These findings have been confirmed by Mathiak et al.[16] Abboud and Kim (2010) further studied the effect of lipids on rotator cuffs and reached similar conclusions.[17]

## 6. So how does hyperlipidemia, in itself, damage the tendon?

Hyperlipidemia leads to tendon injury in several ways:

1. The deposition of cholesterol byproducts is implicated in the formation of tendon xanthomas, which may change tendon properties and result in increased propensity for tendon rupture.
2. Hyperlipidemia may alter the tendon's ECM in such a way that there is increased injury or impaired healing. Rönnemaa et al. found that embryonic fibroblasts react differently to hyperlipidemic rat serum than to normal rat serum. Hyperlipidemic serum was less likely to stimulate the formation of noncollagenous proteins or incorporate glucosamine and cytidine (components of ECM) compared with normal lipid serum.[18]
3. There is reduced baseline elastic modulus and strength of patellar tendons of hyperlipidemic mice compared with controls.[19]
4. Hyperlipidemia impairs both macro and microcirculation, but how this contributes is yet unclear.[20]

Thus hyperlipidemia has profound effects on the tendon substrate, tendon mechanical properties and tendon vascularity.

## 7. What is the end result of these changes?

Tendon tears and impaired tendon healing post-tear. The disruption of tendon architecture, whether in the form of fatty streaks or xanthomas, along with the reduced mechanical properties increases the propensity of tears in hyperlipidemic patients. Beason et al. showed that hyperlipidemic mice have significantly decreased elastic modulus of their patellar tendons as compared to controls, with increased liability for tendon tears.[19]

Furthermore, once a tear, whether macro or micro, has propagated, the resulting healing of the tendon is also impaired as the tendon is now working against the natural inflammatory reaction along with the hyperlipidemic changes, rather than just the inflammatory reaction, as would be the case in normal lipid patients.

## 8. Summary

Hyperlipidemia has profound effects on tendon substance and mechanical properties. Not all hyperlipidemia results in tendon xanthomas, which in fact can be considered as an end stage to fatty streaks. Most patients will present to us with fatty streaks than with tendon xanthomas. It is important to understand the underlying process of xanthoma formation and of hyperlipidemia on the tendon to better tailor future therapies. The one thing that is clear, is that hyperlipidemia, in any form, has negative effects on tendon.

## Conflicts of interest

The authors have none to declare.

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