

### Muscle pain and delayed onset muscle soreness

This is the characteristic pain or discomfort felt 12-48 hours following exercise. The less fit a person is the more quickly and more painfully they tend to be affected.

During the fatigue stages of exercise, muscles produce lactic acid as they respire anaerobically (converting glucose into energy without the presence of oxygen). Lactic acid build-up, combined with the increase in carbon dioxide, causes the pH of the tissue to change from (approx) 7.42 to 7.24. This brings on a condition in the tissue called acidosis. The change in pH stimulates fine unmyelinated C-fibres which lie between the muscle fibres, causing a pain response. The pain encourages us to stop exercising. If lactic acid build-up is too great energy production will slow or stop anyway, but the greater the concentration of lactic acid in the muscle, the greater the pain will be. Therefore the build-up of lactic acid is responsible for the pain at the time of exercise, but it has little or no role in the delayed soreness felt later.

Most muscles in the body, with only a few exceptions, are approximately 50/50 fast and slow twitch fibres. Fast and slow twitch fibres use different systems to produce energy for movement and they contract at different speeds. Fast twitch fibres primarily produce energy for movement anaerobically – up to 200 times faster than aerobic energy production – and are capable of fast explosive movement – up to 5 times faster than slow twitch fibres – but fatigue much more quickly. As the lactic acid builds up in the fast twitch fibre, glucose stores are depleted and ATP (energy) production slows or stops.

ATP is needed for the breaking of the myosin crossbridge actin bonds to allow free and repetitive myosin cross bridge oscillation allowing the sliding filament mechanism to increase and decrease in length and thus the muscle to move. When ATP (adenosine triphosphate) is absent the fibre will become rigid. This is the basis of rigor mortis as ATP cannot be produced after death, the bonds cannot be broken and the muscle becomes rigid.

The slow twitch fibres will continue to work but will also have to overcome the resistance of the locked fast twitch fibres. This effect causes a feeling of stiffness in the muscles and is usually experienced as fatigue sets in. The internal protein structure of fast twitch muscle is more fragile than slow twitch, and the slow twitch fibres will tear the now rigid fast twitch fibres as they continue to move fuelled by their aerobically produced energy. This causes considerable damage to the micro-protein structure inside the fast twitch fibres. As the slow twitch fibres themselves fatigue they too suffer damage to their micro-protein structure.

As soon as exercise ceases 85% of lactic acid dissipates into the blood stream and the rest is metabolised aerobically. Lactic acid cannot exist in the presence of oxygen, and once the 'oxygen debt' is repaid, levels fall rapidly and two hours after exercise there will be no trace in the muscle tissue. Lactic acid therefore plays no further part in delayed onset muscle soreness or DOMS.

The sliding filament mechanism is enabled primarily by the action of actin and myosin fibres. However the whole structure is maintained in symmetry by a protein skeleton made of a fibre called desmin. The protein structure formed by desmin fibres suffers the greatest damage of all the fibres and once damaged produces a chemical called bradykinin. The damage in general also causes production of other inflammatory products such as serotonins, histamines, and prostaglandins. Such is the damage to the fibre that there will be tears in the cell wall through which these inflammatory products escape into the surrounding tissue.

Once outside the cell (muscle fibre) these chemicals, primarily bradykinins, sensitise the C-fibres that normally register pH changes causing them to register pressure instead. In their sensitised state they register normal intra-muscular pressure as pain. In any muscle there is a brief rise in intra-muscular pressure during contraction. When experiencing acute DOMS, even normal movement and the accompanying rise in intra-muscular pressure will cause a pain response; and if the muscle is pressed or prodded it is usually tender. This is compounded by the inflammatory effect of the chemicals in the surrounding tissue causing further pressure.

Observation suggests a light workout on the day following the heavy one will ease discomfort. The reasons for this are unclear, but it is possible the light muscle movement pumps the sensitising chemicals away from the C-fibres aiding dispersal and re-absorption.

During DOMS there is a steep rise in the levels of desmin as the fibres repair. As repair is completed and the inflammatory and sensitising chemicals are re-absorbed and dispersed, pain reduces. Ironically, this damage and repair mechanism is responsible for strengthening and toning muscles, and we are trying to initiate it to a greater or lesser extent when we exercise.

Top athletes now train hard to deliberately initiate these changes in their muscles and then spend time in an ice bath immediately following the exercise to reduce the inflammatory response. This avoids much of the pain and reduces the recovery time allowing a more intensive training program.