



What Happens to the Fat After Treatment With MEDICELL Device

Free overview of clinical and chemical paperworks treating chemical and kinetic process of body - human fat.

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INTRODUCTION

Fat is nature's way of storing excess energy. Fat is very efficient in storing energy as it takes up very little space and does not require a lot of water when stored in a cell. What appears as empty cells are in fact fat-filled cells. Up to 75% of the volume of fat cells is occupied with what we call fat (triglycerides).

When fat tissue is treated by the MEDICELL device, the generation of cavitation vacuum bubbles in a specific area within the fat layer, the mechanical growing of the bubbles causes mechanical disruption of the membranes of the fat cells, sparing the blood vessels, peripheral sensory nerves and connective tissue. Since the effect is localized in a specific depth, overlaying skin is not damaged.

The most common question that arises is the fate of the fat previously contained in the adipose cells, after the cell membrane is broken. This paper aims to describe the mechanism of fat absorption following the cell disruption by MEDICELL treatment.

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FAT CLEARANCE MECHANISM

Fat, inside fat cells, exists in the form of triglycerides. A triglycerides molecule is made of three fatty acids attached to a glycerol backbone. When the fat cell membrane is destroyed, triglycerides are released into the interstitial fluid between the cells.

The presence of large amounts of triglycerides, in interstitial fluid compartments, has no natural correlate. When outside the fat cell, triglycerides are normally packaged in discrete lipoprotein particles – a combination of apolipoproteins and lipids, cholesterol, triglycerides, and cholesteryl esters. A series of metabolic pathways direct the trafficking of water insoluble molecules of cholesterol and triglycerides through the water-based circulatory system and to the interstitial fluid space.

During the passage through the arteries and interstitial space, lipoprotein-bound triglycerides are catabolized to free fatty acids and glycerol molecules.

There is few or no animal or clinical data describing the distribution and temporal processing of free triglycerides released from traumatized adipocytes. The only clinical correlate may be trauma cases of massive areas of soft tissues (car accidents, burns, etc).

This discussion focuses on the interstitial compartment and the metabolism of triglycerides, free fatty acids and glycerol. It is well established that interstitial fluid compartments contain lipoproteins, biological signals and chemical analysis that all interact and engage cells through cell surface membrane receptors or processes (phagocytosis, etc). The kinetic features of the interstitial compartment are now being understood for normal analysis, as about 42% of the total body water is extracellular.

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Are triglycerides molecules present in the interstitial fluid metabolized to free fatty acids and glycerol?

According to the literature, it seems very reasonable to assume that triglycerides are immediately(4 hours) processed by lipoprotein lipase (LPL), an enzyme bound to adipocytes.

In vitro studies have shown that triglycerides presented as emulsions and not in lipoprotein particles are readily hydrolyzed by LPL to glycerol and free fatty acids.

Glycerol is a water soluble molecule and requires no chaperone or carrier through interstitial fluids or the circulatory system.

A short term increase in glycerol concentration following MEDICELL treatment appears reasonable but has not been directly measured. However interstitial levels of glycerol are similar to plasma levels.

To date, no clinically significant elevation of plasma glycerol levels has been reported in any MEDICELL subject. One may extrapolate that interstitial glycerol levels were not significantly elevated in this compartment or sequestered subsequent to MEDICELL treatments.

Free fatty acids are not readily immiscible in water and the transport of these molecules is accomplished by albumin. Albumin, present in interstitial fluid and circulation, has the capacity to bind 2-3 molecules of free fatty acid per molecule. Recently, a differentially radiolabeled triglycerides was injected into the circulatory system of eight subjects. The glycerol and fatty acids moieties had different radiolabels which allowed the kinetic examination of glycerol and fatty acids fates.

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In the forearm, systemic clearance and forearm fractional extraction of glycerol (59%) was greater than that of oleate (14%). Equal systemic and forearm fractional release levels of LPL-generated were observed; again validating the equilibrium that exists between these two water compartments for glycerol. This study suggested that LPL-mediated fatty acid uptake is an inefficient process, but that muscle is more effective than adipose tissue.

Free fatty acids released during the treatment will eventually be delivered to the liver. In the liver, there is no distinction between fatty acids that originate from disrupted adipocytes, those taken from adipocytes due to physiological needs or those originating from a meal consumed several hours ago. In other words, free fatty acids released from the MEDICELL-treated fat cell are being processed in normal pathways that nature has evolved for the transport of fat.

A very simplified way to describe the normal transport of cholesterol and triglyceride in our circulatory system. Triglyceride (TG) is primarily ingested during the dietary processes [stomach and intestine] and transported by chylomicrons through the capillaries and lymph where a large portion is broken down into free fatty acids and glycerol. Any unprocessed TG in chylomicrons is taken up by the liver. A second source of triglyceride is through production in the liver from excess free fatty acids and glycerol. The important cell type that stores TG as an energy bank or depot is the fat cell, or in more scientific terms – an adipocyte.

MEDICELL disrupts fat cells by breaking down the cell membranes, causing the release of TG from the cells. A great portion of TG is probably broken into the free fatty acids and glycerol because of the enzyme, lipoprotein lipase, on the fat cell membrane walls.

The free fatty acids being relatively insoluble in water bind to albumin and are slowly transported to the liver or other tissues that need these molecules as building blocks or energy.

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Glycerol is soluble in water and is transported to the liver or to other cells that could use this molecule. The free glycerol equilibrates among both the interstitial fluid compartment (tissue fluids) and systemic (blood) fluid compartments.

If the released TG is not broken down, it may bind to very low density lipoprotein particles (VLDL) found in the lymph. VLDL is further processed to other lipoprotein classes (IDL, LDL) and ultimately transported to the liver for recycling back to free glycerol and free fatty acids.

All of these pathways have enormous capacity and fast response times in terms of handling TG, as witnessed in the removal of TG in 3-4 hrs from a 2000 cal milkshake.

SUMMARY

In conclusion, the released TG or its derivatives are processed by known metabolic pathways. No unnatural or new metabolic pathways are required for the body to process the released TG. In addition, TG from adipocytes treated by MEDICELL ultimately travels to the liver, where it is recycled to meet the continuing demands of the body.

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