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many thanks On the trend of superhydrophobic surface in textile materials. The growing demand for personal protective clothing for civilian and military activities has raised great concerns on the long-term protection and durability of these desired water-resistant materials. Typical smart fabrics (smart superhydrophobic nano/micro structures for lightweight, high-performance and durable fabrics, e.g., superhydrophobic PTFE/polyester, polyester/polyurethane, natural fiber/polyester, etc.) have been intensively studied for durability in the last few decades. However, these smart fabrics have drawbacks in terms of long-term protection: if these fabrics are used for liquid water, the water will penetrate into the fabric and not off. Water penetration makes these fabrics useless for long-term wearable applications, as the fabric moisture will continue to affect the product integrity. In this paper, we first describe the typical superhydrophobic layers/fabrics and the phenomenon that leads to the buildup of water inside the fabric. We also propose a sustainable alternative that changes the strategy of fabric design to be hydrophobic, which can be realized in a

short time and has satisfactory degradation durability. By replacing fibers with hydrophobic nanofibers, we can create a water-absorbing and self-cleaning coating on the surface of the fabric, thus, making the fabric self-protection durable for a very long time.Effect of chronic alcohol abuse on hepatic lipid metabolism. Livers obtained from alcoholics showed a decrease in the activity of fatty acid synthase (FA synthase) and glucokinase and no change in the activity of glucose-6-phosphatase. The other enzymes participating in the synthesis of fatty acids, including acyl CoA thioesterase, acetyl CoA carboxylase and ATP-citrate lyase, were unaffected. Stimulation of the activity of microsomal triglyceride transfer protein (MTP) was observed. Hepatic triacylglycerol and cholesteryl ester decreased, which corresponded to a rise in free fatty acid concentrations. Hepatic apolipoprotein A-I (apoA-I) decreased together with apolipoprotein B (apoB). The decrease in apoA-I is due to a decrease in the secretion of the protein from the liver, and the rise in hepatic free fatty acids could be the cause of the increase in apoB. A good correlation was found between MTP


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