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CONCISE COMMUNICATION

Voluntary exercise improves murine dermal connective tissue status in high-fat diet-induced obesity

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Abstract Obesity is a risk factor for several cardiovascular and metabolic diseases. Its influence on the skin is less obvious, yet certain negative effects of adipose tissue inflammation on the dermis have been suggested. Excess weight is closely associated with sedentary behavior, so any increase in physical activity is considered beneficial against obesity. To investigate the effects of obesity and physical exercise on the skin, we established a mouse model in which mice were kept either on a high-fat diet or received standard chow. After the two groups achieved a significant weight difference, physical exercise was introduced to both. Animals were given the opportunity to perform voluntary exercise for 40 min daily in a hamster wheel for a period of 8 weeks. We evaluated the status of the dermis at the beginning and at the end of the exercise period by in vivo nonlinear microscopy. Obese mice kept on high-fat diet lost weight steadily after they started to exercise. In the high-fat diet group, we could detect significantly larger adipocytes and a thicker layer of subcutaneous tissue; both changes started to normalize after exercise. Nonlinear microscopy revealed an impaired collagen structure in obese mice that improved considerably after physical activity was introduced. With the ability to detect damage on collagen structure, we set out to address the question whether this process is reversible. With the use of a novel imaging method, we were able to show the reversibility of connective tissue deterioration as a benefit of physical exercise.

Keywords Obesity · Adipose tissue inflammation · Matrix degradation · Physical exercise · In vivo nonlinear microscopy

Introduction

Sedentary lifestyle accompanied by the consumption of food rich in saturated fat and carbohydrates is increasingly more common that leads to the "obesity epidemic". The fat tissue is now considered a complex network of cells; a much more complicated tissue than a great amount of adipocytes that simply store the excess fat [23]. The matrix of the adipose layer contains additional types of cells such as preadipocytes, fibroblasts, endothelial, and immune cells. It is known that an excess in fat tissue leads to a systemic inflammation. The subcutaneous fat layer is infiltrated with neutrophils, regulatory T-cells, and macrophages, where a shift from M2 to M1 subset is seen [14]. Through release of inflammatory cytokines and reactive oxygen species along with proteases and matrix degrading enzymes, adipose tissue inflammation leads to a remodeling of the surrounding connective tissue, a process needed for the local expansion of fat tissue [14, 23].

A clinical sign of the systemic inflammation in obesity is that it is strongly associated with an increased rate of metabolic diseases, such as insulin resistance, type 2 diabetes, dyslipidemia, and hypertension [9]. Obesity and diabetes-related dermatological complications are now commonly seen with the increased incidence of metabolic syndrome. The most severe consequences on the skin include impaired

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