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Negative Feedback System for Glycogen Synthesis in Skeletal Muscle

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The major site for blood glucose uptake in the human body is skeletal muscle, because more than 50% of body weight is skeletal muscle. Elevated blood glucose level from carbohydrate ingestion stimulates insulin secretion in pancreas, leading to skeletal muscle glucose uptake. Although blood insulin level does not increase after exercise, skeletal muscle takes in blood glucose vigorously. That means skeletal muscle insulin sensitivity to blood glucose uptake is enhanced after exercise. Muscle glycogen, which is major energy source in skeletal muscle, is depleted during prolonged exercise. Enhanced muscle insulin sensitivity to blood glucose uptake makes it possible to replenish muscle glycogen rapidly after glycogen depleting exercise.

Glycogen supercompensation is the increase in muscle glycogen concentration, to levels far above those found in well-fed individuals, that occurs in response to carbohydrate feeding 24 to 48 hours after a bout of glycogen-depleting exercise. Insulin sensitivity is decreased rather than increased in glycogen supercompensated muscle, because muscle does not need to take in blood glucose any more. This is the negative feedback system for glycogen replenishment in skeletal muscle.

Our research group (Kentaro Kawanaka and Akiko Sano) is trying to figure out the mechanism responsible for this negative feedback system. The goal of our research is to elucidate the cellular and molecular events that decrease insulin sensitivity in glycogen supercompensated muscle. We believe that as our basic understanding of the mechanism improves, it will become possible to develop nutritional treatment that prolongs increased insulin sensitivity after exercise. Such an approach enhances positive effects of exercise, leading to the maintenance of health and treatment of disease in diabetics.

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