

consumption, indirect calorimetry or infrared-based activity levels. No differences were observed in resting tissue glycogen content, epididymal fat pad mass, hindlimb muscle mass or liver and muscle mitochondrial content between KI and respective WT mice. Fat deposition was increased by 61% in W100A liver ($P < 0.05$) and 54% in W98A quadriceps muscle ($P < 0.05$) versus WT, concomitant with reductions in total protein content of AMPK α and β subunits. These data suggest that glycogen-bound AMPK is central to whole-body and tissue metabolism. We reveal new insights into the physiological roles of AMPK and suggest that loss of glycogen binding negatively impacts metabolism via reductions in the cellular AMPK pool.

<https://doi.org/10.1016/j.orcp.2018.11.022>

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Muscle membrane permeability to glucose and blood flow explain increased insulin sensitivity in muscle after exercise



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The relationship between skeletal muscle perfusion, interstitial glucose concentration and sarcolemmal permeability to glucose in exercise-induced increases in muscle insulin sensitivity is not well established. In 10 young men, a euglycemic hyperinsulinemic clamp was initiated 4 h after 1-legged exercise, and leg blood flow (LBF), leg glucose uptake (LGU) and muscle interstitial glucose concentration were measured. During the clamp LGU and LBF were higher ($P < 0.05$) in the previously exercised than the control leg whereas the interstitial glucose concentration decreased to lower ($P < 0.05$) values in the exercised (~ 3.1 mM) than the control (~ 4.8 mM) leg. Apparent sarcolemmal glucose permeability was twice as high ($P < 0.05$) in the exercised compared with the rested leg. The NOS inhibitor L-NMMA decreased LBF in both legs and interstitial glucose concentration dropped to ~ 2.3 mM in the exercised but only to ~ 3.7 mM in non-exercised muscle. This abrogated the augmented effect of insulin on LGU in the exercised leg while apparent sarcolemmal permeability to glucose remained unchanged with L-NMMA in both legs. Doubling LBF by local infusion of ATP increased LGU in both legs without any major change in interstitial glucose concentration or sarcolemmal permeability to glucose. These findings suggest that during flow restriction due to L-NMMA, the interstitial glucose concentration becomes limiting for insulin-stimulated glucose uptake in previously exercised but not in non-exercised muscle. Therefore, the vasodilatory effect of insulin is an important component of the increased insulin sensitivity to stimulate glucose uptake following exercise by limiting the drop in the interstitial glucose concentration that occurs due to the increased sarcolemmal permeability to glucose.

<https://doi.org/10.1016/j.orcp.2018.11.023>

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Time restricted feeding

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Light/dark cycle aligned (circadian) rhythms regulate the processes associated with the acquisition and metabolism of food. In mice, limiting food intake to the non-resting phase induces circadian desynchrony, and results in obesity and impaired glucose tolerance (1). A feature of modern human lifestyles is an extended period of eating, and a curtailed period of fasting, that likely induces circadian desynchrony. Time restricted feeding (TRF) is a novel dietary approach that limits when, rather than what, food is eaten. Time restricted feeding is emerging as a powerful regulator of health and longevity in mouse models, and TRF restores peripheral circadian rhythmicity in liver of mice that are fed a high fat diet. The evidence as to whether TRF will be beneficial for metabolic health in humans is currently limited. Short-term studies are beginning to emerge that suggest that the health effects observed in mice will translate to people who are at risk of type 2 diabetes. Particularly, TRF improves glucose tolerance independently of food intake. Our data similarly shows that TRF improves glycaemic control in men who are at risk of type 2 diabetes. Further, we show that there may be at least some flexibility in the clock time that the fasting time is initiated. We are currently conducting metabolic studies in humans to examine whether TRF can improve 24-hour glucose metabolism, and reset peripheral clocks in adipose tissue. We have also tested the metabolic impacts of TRF during simulated shift work and show that TRF may be a tool to rescue the metabolic consequences of shift work in people.

<https://doi.org/10.1016/j.orcp.2018.11.024>

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Alternating periods of energy restriction and energy balance

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Abstract not available.

<https://doi.org/10.1016/j.orcp.2018.11.025>

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Circadian rhythms, diet and exercise

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Abstract not available.

<https://doi.org/10.1016/j.orcp.2018.11.026>