Skeletal muscle transcriptional coactivator PGC-1alpha mediates mitochondrial, but not metabolic, changes during calorie restriction

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Résumé en anglais	Calorie restriction (CR) is a dietary intervention that extends lifespan and healthspan in a variety of organisms. CR improves mitochondrial energy production, fuel oxidation, and reactive oxygen species (ROS) scavenging in skeletal muscle and other tissues, and these processes are thought to be critical to the benefits of CR. PGC-1alpha is a transcriptional coactivator that regulates mitochondrial function and is induced by CR. Consequently, many of the mitochondrial and metabolic benefits of CR are attributed to increased PGC-1alpha activity. To test this model, we examined the metabolic and mitochondrial response to CR in mice lacking skeletal muscle PGC-1alpha (MKO). Surprisingly, MKO mice demonstrated a normal improvement in glucose homeostasis in response to CR, indicating that skeletal muscle PGC-1alpha is dispensable for the whole-body benefits of CR. In contrast, gene expression profiling and electron microscopy (EM) demonstrated that PGC-1alpha is required for the full CR-induced increases in mitochondrial gene expression and mitochondrial density in skeletal muscle. These results demonstrate that PGC-1alpha is a major regulator of the mitochondrial response to CR in skeletal muscle, but surprisingly show that neither PGC-1alpha nor mitochondrial biogenesis in skeletal muscle are required for the whole-body metabolic benefits of CR.

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