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The Effects Of Quercetin On Skeletal Muscle Mitochondrial Biogenesis And OXPHOS Regulation
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A high fat diet causes a reduction in the expression of the genes of the electron transport chain, mitochondrial carrier proteins, and PGC-1α and PGC-1ß mRNA. Mitochondrial dysfunction has been linked to numerous chronic diseases. The antioxidant quercetin has been shown to protect against damage caused by exogenous and endogenous free radicals. The purpose of this study was to determine if quercetin would protect the genes of mitochondrial biogenesis (PGC-1α and PGC-1ß) and OXPHOS regulation (NDUFB5 and SDHB) from the downregulation caused by consumption of a high fat diet. The quadriceps muscle was removed from 21 male, C57BL/6J mice that had been fed a low fat (10% kcal fat), a high fat (45% kcal fat), or a high fat plus quercetin (45% kcal fat + 1.2% quercetin) diet for 3 weeks. RNA was isolated from the samples and quantitative RT-PCR was used to compare gene expression. As expected, there was a significant difference between gene expression in the low fat group compared to the high fat and high fat plus quercetin groups for all measured genes (pgc-1α: $F = 5.76$, $df = 2$, $P = 0.011$; pgc-1ß: $F = 5.28$, $df = 2$, $P = 0.016$; NDUFB5: $F = 5.66$, $df = 2$, $P = 0.012$; SDHB: $F = 5.04$, $df = 2$, $P = 0.018$). We found no significant difference between gene expression of the high fat group compared to the high fat plus quercetin group (pgc-1α: $t = -0.71$, $df = 12$, $P = 0.49$; pgc-1ß: $t = 0.16$, $df = 12$, $P = 0.87$; NDUFB5: $t = 0.27$, $df = 12$, $P = 0.79$; SDHB; $t = -0.20$, $df = 12$, $P = 0.84$). These results indicate that this dose of quercetin is not effective in protecting the expression of genes associated with mitochondrial health from the deleterious effects cause by consumption of a high fat diet.