The glucocorticoid cortisol (known as corticosterone in rodents) is important in stress responses. However, some diseases such as diabetes, are linked to chronically high circulating levels of cortisol and the levels of cortisol may contribute to disease progression. In this study, we showed that continual exposure to higher than normal levels of corticosterone was sufficient to reduce the number of blood vessels within skeletal muscle of rats. We next examined the direct effects of corticosterone on cultured endothelial cells (those cells comprising the blood vessels). Corticosterone prevented endothelial cell sprouting, and induced cell rounding, indicative of a failure to form new blood vessels or to maintain pre-existing ones. This was linked to a reduced amount of cell proliferation and reduced capacity for the cells to produce matrix metalloproteinases, enzymes that are critical for allowing new blood vessel formation. Altogether, this study provides novel evidence that a continual elevation in the glucocorticoid corticosterone can exert inhibitory effects on blood vessels within skeletal muscle, which would in turn have negative consequences on muscle function and contribute to metabolic imbalances within the muscle.

Reference: Shikatani EA, Trifonova A, Mandel ER, Liu ST, Roudier E, Krylova A, Szigiato A, Beaudry J, Riddell MC, Haas TL. Inhibition of proliferation, migration and proteolysis contribute to corticosterone-mediated inhibition of angiogenesis. PLoS One. 2012;7(10)

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