Society for Endocrinology media release

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Vitamin D replacement improves muscle efficiency

New research shows for the first time a link between vitamin D levels and muscle efficiency. Vitamin D supplementation may also be effective in improving skeletal muscle function. This study is presented today at the Society for Endocrinology annual conference in Harrogate, UK. The findings may explain the physical fatigue commonly experienced by patients with vitamin D deficiency, with broad implications for a large section of society.

Vitamin D is a hormone produced in the skin using energy from sunlight. Vitamin D deficiency is a significant public health problem as diagnosed cases are on the rise and the hormone is essential for good bone health. Alongside poor bone health, muscle fatigue is a common symptom in vitamin D deficient patients. This fatigue could be due to a problem in the mitochondria: the 'power stations' within each cell of the body. Mitochondria use glucose and oxygen to make energy in a form that can be used to run the cell (an energy-rich molecule called ATP). Muscle cells need large amounts of ATP for movement and they use phosphocreatine as a ready and available energy source to make ATP. The mitochondria also replenish this phosphocreatine store after muscle contraction. Measurement of the time taken to replenish the phosphocreatine store is a measure of mitochondrial efficiency: better mitochondrial function is associated with shorter phosphocreatine recovery times.

Researchers from Newcastle University, led by Dr Akash Sinha who also works within the Newcastle upon Tyne Hospitals NHS Foundation Trust, investigated phosphocreatine recovery times in patients with vitamin D deficiency. They employed a non-invasive magnetic resonance scan to measure phosphocreatine dynamics in response to exercise in the calf muscles of 12 patients with severe vitamin D deficiency before and after treatment with vitamin D. This is the first time a study of this kind has been conducted.

The team found that phosphocreatine recovery significantly improved after the patients took a fixed dose of oral vitamin D for 10-12 weeks (average phosphocreatine recovery half time decreased from 34.4sec to 27.8sec, $p<0.001$). All patients reported an improvement in symptoms of fatigue following supplementation. In a parallel study, the group demonstrated that low Vitamin D levels were associated with reduced mitochondrial function ($r=-0.41$, $p=0.009$).

The research shows for the first time that vitamin D levels are correlated with muscle efficiency, and that muscle aerobic metabolism improves with Vitamin D supplementation. Whilst this is a small study, it establishes clear proof of principle and (for the first time) a link between vitamin D and mitochondria in man. The mechanisms underpinning this effect are an avenue for future research by the group, who also aim to establish whether vitamin D supplementation could alleviate frailty in the elderly or improve the exercise capacity of athletes.

Study leader Dr Akash Sinha, Clinical Research Fellow at Newcastle University and Newcastle upon Tyne Hospitals NHS Foundation Trust said:

"*This is the first time a link has been shown between vitamin D status and muscle aerobic function*. *To do so we used a non-invasive scan to get a unique biochemical perspective on muscle mitochondrial metabolism during exercise: a window into what is really going on in the muscle as it works*

"*Patients with vitamin D deficiency often experience symptoms of muscle fatigue. Our findings in a small group of patients with very low vitamin D levels show that muscle efficiency significantly improves when vitamin D status is improved*.''

"*We'll need further research in more patients to work out how this is happening and whether non-deficient patients can benefit from this too*."

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Notes for Editors:

This research will be presented as a lecture (OC1.6) at the Society for Endocrinology BES meeting, on Monday 18 March 2013. The abstract for this lecture is reproduced at *Endocrine Abstracts*.

The Society for Endocrinology BES 2013 conference is Britain's biggest scientific meeting on hormones, and is taking place at the Harrogate International Centre from 18-21 March 2013. For the full programme, please click here.

Please mention the Society for Endocrinology meeting in any story

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The Society for Endocrinology is Britain's national organisation promoting endocrinology and hormone awareness. For general information, please visit our website: http://www.endocrinology.org

For more information on vitamin D, endocrinology and hormones please visit You & Your Hormones (www.yourhormones.info), the Society for Endocrinology's public information website.

ABSTRACT

Improving the Vitamin D status of Vitamin D deficient adults is associated with improved mitochondrial oxidative function in skeletal muscle

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Objective

Suboptimal mitochondrial function has been implicated in several disorders where fatigue is a prominent feature. Vitamin D deficiency is a well-recognised cause of fatigue and myopathy. The aim of this study was to examine the effects of cholecalciferol therapy on skeletal mitochondrial oxidative function in symptomatic, vitamin D deficient individuals.

Design

This longitudinal study assessed mitochondrial oxidative phosphorylation in the gastro-soleus compartment using Phosphorus-31 magnetic resonance spectroscopy measurements of phosphocreatine recovery kinetics in 12 symptomatic, severely vitamin D deficient subjects before and after treatment with cholecalciferol (10-12 weeks later). All subjects had serum assays before and after cholecalciferol therapy to document serum 25OHD and bone profiles. 15 healthy controls also underwent ^{31}P-MRS and serum 25OHD assessment.

Results

The phosphocreatine recovery half-time ($\tau_{1/2}$PCr, $\tau_{1/2}$ADP) was significantly reduced following cholecalciferol therapy in the subjects indicating an improvement in maximal oxidative phosphorylation ($p<0.001$, $p=0.003$). This was associated with an improvement in mean serum 25OHD levels (8.8±4.2nmol/L to 113.8±51.5nmol/L, $p<0.001$). There was no difference in phosphate metabolites at rest. A linear regression model showed that decreasing serum 25OHD levels are associated with increasing $\tau_{1/2}$PCr ($r=-0.41$, $p=0.009$). All patients reported an improvement in fatigue following cholecalciferol therapy.

Conclusions

Cholecalciferol therapy augments muscle mitochondrial maximal oxidative phosphorylation following exercise in symptomatic, vitamin D deficient individuals. This finding suggests that changes in mitochondrial oxidative phosphorylation in skeletal muscle could at least be partly responsible for the fatigue experienced by these patients. For the first time, we demonstrate a link between vitamin D and the mitochondria in human skeletal muscle.