G93A transgene mice are the most widely used animal model for human ALS. We studied the effects of high-dose vitamin D3 (50x the adequate intake) vs. adequate intake in a mouse model of amyotrophic lateral sclerosis (a.k.a. Lou Gehrig's disease). We found significant improvements in the physical functional capacity of mice receiving the high dose of vitamin D3, but no changes in the age at disease onset, duration of disease progression or lifespan. HiD females, but not males, may have experienced mild toxicity since they consumed 9% less food corrected for body weight. Although HiD females showed 31% greater gait abnormality score (not significant) prior to disease onset, they exhibited a significant 20% greater paw grip endurance when corrected for gait abnormality score. Pooling the sexes, high-dose mice demonstrated a12% greater paw grip endurance vs. the adequate intake controls, and when analyzing data post-disease onset only, these mice demonstrated a 37% greater paw grip endurance score.

In support of our previous work studying mice under dietary D3 restriction as well as being supplemented with 10x the adequate intake, D3 supplementation at 50x in the current study attenuated the decline in G93A mouse functional capacity, but did not affect age at disease onset, age at full hindlimb paralysis or lifespan. Further, females may have slightly surpassed the threshold for long-term D3 supplementation toxicity.

Reference: Gianforcaro A, Solomon JA, **Hamadeh MJ**.Vitamin D(3) at 50x AI attenuates the decline in paw grip endurance, but not disease outcomes, in the G93A mouse model of ALS, and is toxic in females. PLoS One. 2013;8(2):e30243.

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