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Taiwan discovers link between muscle aging and mortality

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NHRI has discovered a direct link between skeletal muscle aging and mortality through thermogenic and metabolic abnormalities



Using genetic modified mouse models, researchers at the National Health Research Institutes (NHRI) in Taiwan have shown promise for advances in understanding the causal relationship between muscle aging, thermogenesis, and mortality.

Lamin A is a component of the nucleoskeleton. De novo mutation of human lamin A gene (i.e.*LMNA*) generated progerin protein, which is causal to the Hutchinson-Gilford progeria syndrome (HGPS).

The aging process in HGPS patients is about 8 time faster than normal people. Dr. Chi and her team developed a mouse model which was conditionally overexpressed with human progerin in muscle, and discovered that muscleÃ¢â?¬Â•specific overexpression of progerin was sufficient to induce muscular dystrophy and alter wholeÃ¢â?¬Â•body energy expenditure, leading to premature death.

The team found sarcolipin (Sln), an ERÃ¢â?¬Â•associated protein involved in heat production, is upregulated in the skeletal muscle expressing progerin. The depletion of *Sln* accelerated the early death of *Lmna* mutant mice.

A further examination at the molecular level revealed that progerin recruits Sln and Calnexin to the nuclear periphery. Furthermore, progerinÃ¢â?¬Â•expressing myoblasts presented enhanced storeÃ¢â?¬Â•operated Ca^{2+} entry.

These findings suggested that progerin dysregulated calcium homeostasis through an interaction with a subset of ERÃ¢â?¬Â• associated proteins, resulting in thermogenic and metabolic abnormalities.