

IGF-1 LR3 — Research Summary

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1. What It Is

IGF-1 LR3 (Long Arg3 Insulin-like Growth Factor-1) is an 83-amino-acid recombinant analog of human insulin-like growth factor 1 (IGF-1), engineered with a 13-amino-acid N-terminal extension and an arginine substitution at position 3. These modifications reduce the analog's affinity for IGF-binding proteins (IGFBPs), extending its active window to a reported 20–30 hours in circulation, versus about 20 minutes for native IGF-1.

Molecular weight: ~9,197 Da. Mechanism: Activates the IGF-1 receptor (IGF-1R), triggering downstream PI3K and MAPK signaling.

Important regulatory distinction: FDA-approved IGF-1 therapy exists — mecasermin (brand name Increlex) — for severe primary IGF-1 deficiency. Mecasermin is a different molecule from IGF-1 LR3; the approved drug's clinical data does not transfer to IGF-1 LR3.

2. Reported Effects in Research Literature

This is a peptide with essentially no direct human clinical trial data — worth stating plainly in documentation.

- A dedicated evidence review identified 10 total published studies on IGF-1 LR3, all preclinical: successful recombinant expression in yeast, mixed results in animal models (amyloid plaque remodeling without cognitive benefit in transgenic Alzheimer's mice, nerve regeneration enhancement in rats, minimal growth effects in fetal sheep), and acute metabolic effects on insulin secretion in isolated tissue that did not persist.
- A 2025 fetal sheep study (Children's Hospital Colorado / University of Colorado research group) tested low-dose IGF-1 LR3 infusion in a model of fetal growth restriction from placental insufficiency; the treatment did not improve beta-cell growth or function and significantly reduced circulating branched-chain amino acids, extending earlier findings from the same group that high-dose IGF-1 LR3 infusion in normal fetal sheep increased body weight while reducing insulin levels.
- No human clinical trials or randomized controlled trials have been conducted on IGF-1 LR3 itself.
- Broader IGF-1 pathway literature (from native IGF-1 and mecasermin research) documents IGF-1's role in muscle protein synthesis, tissue repair, and prevention of age-related muscle atrophy in animal models — but this evidence pertains to different molecules, not IGF-1 LR3 specifically.
- Performance-enhancement/bodybuilding research interest in IGF-1 LR3 is extensive in community/anecdotal contexts but is poorly represented in peer-reviewed literature.
- Bottom line: IGF-1 LR3 is one of the least clinically validated compounds in this set — its evidence base is entirely preclinical (animal), and public human pharmacokinetic and safety data are essentially absent.

3. Clinical & Preclinical Study References

Engel MG, Narayan S, Cui MH, et al. IGF-1 LR3 preclinical evidence summary (Alzheimer's model). J Alzheimers Dis. 2025.

Review: "Optimizing IGF-I for skeletal muscle therapeutics." PMC — covers the broader IGF-1 pathway literature, not IGF-1 LR3 specifically.

Fetal sheep growth-restriction study, Children's Hospital Colorado neonatology research group. 2025.

Regulatory/Safety Note: IGF-1 LR3 is not approved by the FDA for the diagnosis, treatment, cure, or prevention of any disease; it is distinct from the FDA-approved drug mecasermin (Increlex).

Verification Update: One additional preclinical (animal) study was identified and added: a 2025 fetal sheep study from the Children's Hospital Colorado neonatology group testing low-dose IGF-1 LR3 in a fetal growth restriction model. This adds detail to, but does not change, the existing conclusion that IGF-1 LR3 has no human trial data.

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