

Role Of Integrins In Insulin Signaling

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ABSTRACT

Researchers at the University of California, Davis have developed single-chain insulin (SCI) technology, an insulin-based therapeutic approach that may improve treatment for diabetes and cancer by combining insulin, IGF, and integrin signaling properties.

FULL DESCRIPTION

This technology involves isolated proteins and vectors encoding single-chain insulin analogs where insulin's A and B chains are linked via an integrin-binding domain from IGF1 or IGF2, either wild-type or mutated. The SCI acts either as an antagonist or agonist of the insulin receptor, enabling dual therapeutic functionality: suppressing cancer cell proliferation or improving insulin sensitivity and glucose uptake. The SCI's design addresses current insulin therapy limitations by enhancing stability, reducing cold-chain dependencies, and targeting integrin-mediated signaling pathways relevant to metabolic disorders and malignancies.

APPLICATIONS

- ▶ Diabetes treatment, including type 1, type 2, and associated metabolic syndromes.
- ▶ Cancer therapeutics targeting breast cancer, prostate cancer, melanoma, and other malignancies.
- ▶ Development of novel insulin analog therapies with enhanced stability and efficacy.
- ▶ Biopharmaceutical production of integrin-targeting single-chain insulin proteins and gene therapy vectors.
- ▶ Combination therapies co-administered with existing glucose-lowering drugs.
- ▶ Research tools for studying insulin, IGF, and integrin signaling in metabolic and cancer biology.

FEATURES/BENEFITS

- ▶ Improves stability and relaxes storage requirements versus traditional insulin, reducing reliance on strict cold-chain handling.
- ▶ Delivers dual therapeutic activity by suppressing cancer-related signaling while improving insulin sensitivity.
- ▶ Modulates receptor signaling through an integrin-binding linker domain, enabling targeted control of pathway cross-talk.
- ▶ Enables tunable insulin receptor behavior by switching between agonist or antagonist activity via specific linker mutations.
- ▶ Reduces treatment cost and operational complexity by simplifying storage, distribution, and overall therapy logistics.

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OTHER INFORMATION

KEYWORDS

agonist, antagonist,
cancer cell proliferation,
diabetes, integrin
binding, insulin receptor,
metabolic disorders,
single-chain insulin,
therapeutic compositions,
vectors

CATEGORIZED AS

- ▶ **Medical**
 - ▶ Disease: Cancer
 - ▶ Disease: [Metabolic/Endocrinology](#)

RELATED CASES

2025-418-0

- ▶ Supports combination therapy by remaining compatible with established glucose-lowering agents (e.g., metformin, GLP-1 receptor agonists).
- ▶ Expands administration options by supporting both gene-delivery (vector-based expression) and protein formulation delivery approaches.
- ▶ Eliminates cold-chain-driven storage and distribution burdens that increase insulin cost and logistical complexity.
- ▶ Addresses the lack of more stable, higher-performing insulin analog options by improving durability and usability.
- ▶ Fills the gap for therapies that jointly target diabetes and cancer by leveraging insulin and integrin pathway mechanisms.
- ▶ Provides a needed insulin receptor antagonist approach for cancer therapy, where such options are currently limited.
- ▶ Exploits previously underused insulin/IGF/integrin signaling interplay by translating complex pathway cross-talk into a therapeutic strategy.

PATENT STATUS

Patent Pending

ADDITIONAL TECHNOLOGIES BY THESE INVENTORS

- ▶ [Suppression of sPLA2-Integrin Binding for Treating an Inflammatory Condition or Suppressing Cell Proliferation](#)
- ▶ [Development of Dominant Negative CD40L Antagonists DACD40L](#)
- ▶ [Novel Insight into Inhibiting IGF1 Signaling](#)
- ▶ [The Isolated Heparin-binding Domain \(HBD\) of VEGF165 and the Isolated D1 Domain of VEGFR2 \(KDR\)](#)
- ▶ [Novel Fibroblast Growth Factor 1-Derived Peptides for Therapy and Drug Discovery](#)
- ▶ [Modulating MD-2-Integrin Interaction for Sepsis Treatment](#)
- ▶ [Integrin Binding to P-Selectin as a Treatment for Cancer and Inflammation](#)

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