A New Mechanism For Hypertriglyceridemia In Humans

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SUMMARY

UCLA researchers in the Department of Medicine have identified autoantibodies against GPIHBP1, a GPI anchored protein of capillary endothelial cells, which may provide a novel therapeutic strategy for patients with hypertriglyceridemia.

BACKGROUND

Hypertriglyceridemia patients suffer from elevated levels of triglycerides in their blood. It has been associated with atherosclerosis and predisposition for cardiovascular disease. In certain circumstances, the underlying cause of hypertriglyceridemia is unknown and traditional treatments, such as lifestyle modulation or pharmacological therapies are ineffective. There is a need for more detailed understanding of the etiology of the disease and more effective therapies in cases for which traditional therapies are ineffective.

INNOVATION

Researchers at UCLA have identified a novel thereapeutic strategy for patients with hypertriglyceridemia who also have autoantibodies to GPIHBP1. It has been found that autoantibodies to GPIHBP1 were found present in patients with hypertriglyceridemia and that these autoantibodies blocked the binding of lipoprotein lipase (LPL) to GPIHBP1. Researchers has shown that patients may be treated for hypertriglyceridemia by administration of an immunosuppressive treatment and/or HPIhBP1 activator.

APPLICATIONS

- Diagnosis of the etiology of hypertriglyceridemia

ADVANTAGES

- Can address patients for which traditional therapies are ineffective
- Diagnostic kit can be developed to detect autoantibodies in patients

STATE OF DEVELOPMENT

Autoantibodies have been documented in four human subjects suffering from hypertriglyceridemia. Development is ongoing.

PATENT STATUS

Country	Type	Number	Dated	Case
United States Of America	Issued Patent	11,090,310	08/17/2021	2016-986

RELATED MATERIALS

- Beigneux, Anne P., et al. "GPIHBP1 missense mutations often cause multimerization of GPIHBP1 and thereby prevent lipoprotein lipase binding." Circulation research 116.4 (2015): 624-632.
- Mysling, Simon, et al. "The acidic domain of the endothelial membrane protein GPIHBP1 stabilizes lipoprotein lipase activity by preventing unfolding of its catalytic domain." Elife 5 (2016): e12095.
- Fong, Loren G., et al. "GPIHBP1 and Plasma Triglyceride Metabolism." Trends in Endocrinology & Metabolism (2016).
- Hu, Xuchen, et al. "Monoclonal Antibodies That Bind to the Ly6 Domain of GPIHBP1 Abolish the Binding of LPL." Journal of Lipid Research (2016): jlr-M072462.

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INVENTORS

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OTHER INFORMATION

KEYWORDS

hypertriglyceridemia, diagnostic, autoantibody, GPIHBP1, LPL, triglyceride, lipoprotein lipase, therapeutic, atherosclerosis, cardiovascular disease

CATEGORIZED AS

- **Medical**
  - Disease: Cardiovascular and Circulatory System
  - Other
  - Screening

RELATED CASES

2016-986-0

▶ Allan, Christopher M., et al. "An LPL–specific monoclonal antibody, 88B8, that abolishes the binding of LPL to GPIHBP1." Journal of Lipid Research57.10 (2016): 1889-1898.

▶ Allan, Christopher M., et al. "Mobility of HSPG-bound LPL explains how LPL is able to reach GPIHBP1 on capillaries." Journal of Lipid Research (2016): jlr-M072520.

ADDITIONAL TECHNOLOGIES BY THESE INVENTORS

- Monoclonal Antibodies Against GPIHBP1
- Mouse Model for Premature Aging: Zmpste24 Knockout Mice
- Monoclonal Antibodies Against Prelamin A

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