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 therapeutic 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 GPIHBP1 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
Patent Pending

RELATED MATERIALS

ADDITIONAL TECHNOLOGIES BY THESE INVENTORS
- Mouse Model for Premature Aging: Zmpste24 Knockout Mice
- Monoclonal Antibodies Against GPIHBP1
- Monoclonal Antibodies Against Prelamin A

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