



National Human Genome Research Institute (NHGRI)

Patent-pending Technology Available for Licensing

Use of Adenosine Agonists to Prevent Vascular Calcification Disorders

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Patent Status

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Summary

NHGRI scientists have discovered a genetic defect that causes vascular calcification. Specifically, mutations in the ecto-5'-nucleotidase (NT5E) gene encoding Cluster of Differentiation 73 (CD73) lead to a decrease in adenosine, and ultimately, an increase in vascular calcification. CD73 is an enzyme that converts adenosine monophosphate (AMP) to adenosine in the extracellular region of the vascular endothelium. Dr. Gahl and his colleagues are now in the process of testing various adenosine agonists, in an attempt to restore normal intracellular adenosine levels, in preclinical studies with the goal of identifying promising candidate drugs that could be used in the future to treat or prevent various vascular calcification disorders.

Potential Commercial Applications

The discovery of adenosine's role in vascular calcification disorders could be used to identify and clinically test new adenosine agonist-based treatments for such conditions. Vascular or joint capsule calcification is a clinical finding of many diseases and disorders including atherosclerosis, diabetes, Monckberg medial calcification sclerosis, CD74 deficiency, Ehlers-Danlos Syndrome (EDS), Marfan/Loeys-Dietz Syndrome, fibromuscular dysplasia, Kawasaki Syndrome, pseudoxanthoma elasticum, and premature placental calcification.

Related Article

Gahl, W. et al., *NT5E Mutations and Arterial Calcifications*, 364 N ENGL J MED 432 (2011). <http://www.nejm.org/doi/pdf/10.1056/NEJMoa0912923>

Key Words

Vascular Calcification, Atherosclerosis, Diabetes, Adenosine

