

CD47 Blocking Antibody and Peptides to Decrease Ischemia and Pulmonary Hypertension

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Technology description

Investigators at the University of Pittsburgh and NIH have developed novel blocking antibodies and peptides for preventing, ameliorating, and/or reducing tissue ischemia and/or tissue damage due to ischemia and ischemia/reperfusion and preventing thrombotic complications and clot formation. These therapeutics work by maximizing physiologic NO thus increasing blood vessel diameter, blood flow and tissue perfusion. The therapeutics have been found effective in models of tissue ischemia, ischemia reperfusion injury and in peripheral vascular disease. By suppressing CD47 and/or blocking TSP1 and/or CD47 activity or interaction the therapeutics increase the pro-survival and pro-flow effects of NO. Modulating the interaction of CD47-TSP1 in blood vessels allows for control of blood vessel diameter and blood flow, and additionally has profound effects on blood pressure and cardiac function. Under conditions of decreased blood flow, for instance through injury or atherosclerosis, blocking TSP1-CD47 interaction allows blood vessels to dilate and increases blood flow, tissue perfusion and tissue survival. This in turn reduces or prevents tissue necrosis and death. The therapeutics identified herein allow for precise regulation of blood flow to tissues and organs which need it, while substantially avoiding systemic complications.

Application area

1. Prevent or reverse ischemic disease induced by heart attack (myocardial infarction), stroke and peripheral vascular disease (PVD).
2. Prevent or reverse cerebral ischemia, and prevent tissue loss secondary to stroke
3. Regulate blood pressure and treat hypertension
4. Control blood flow during reconstructive surgery
5. Prevent or reverse ischemia/reperfusion injury during skin and organ transplants
6. Prevent thrombosis and clot formation.

Advantages

1. Precise regulation of blood flow to tissues and organs.

2. Efficiently increase tissue survival under conditions of trauma and surgery.
3. Efficiently modulate thrombospondin-1 and CD47 to affect tissue perfusion.
4. Efficiently modulate Redox, Calcium, cGMP, and cAMP signaling to enhance blood flow
5. Suppress inflammation by maximizing physiologic NO
6. Minimize thrombosis by maximizing NO

Institution

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