Anti-SEMA7A for the treatment of post-ischemic tissue damage

Myocardial infarction remains one of the leading causes of death worldwide. Treatment involves early reperfusion of the myocardium, which however can induce reperfusion injury, causing additional damage to the originally ischemic tissue. Semaphorin 7A (SEMA7A) inhibition offers a specific molecular target to protect the myocardial tissue from reperfusion injury.

IP RIGHTS
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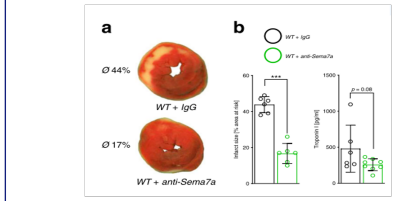
SEMA7A inhibitors effectively prevent heart tissue from reperfusion injury

Inventors from Julius- Maximilians university Würzburg and Eberhard Karls university Tübingen have shown that SEMA7A inhibitors can effectively counteract reperfusion injury and thus tissue damage after reperfusion.

01 SEMA7A inhibitors counteract post-ischemic tissue damage
02 Specific molecular target
03 Reduces infarct size in mouse studies

REFERENCES:
Köhler et al, Nature Communications (2020)



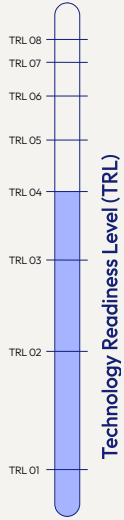
Mice were injected with either anti-Sema7a or IgG control before starting 120 min reperfusion after ischemia, with samples taken after 1 or 120 min of reperfusion. a.) Representative TTC-stained heart slices of myocardial infarcts (blue/dark = retrograde Evans blue staining; red and white = AAR, white = infarcted tissue) with b systematic evaluation of infarct sizes and correlating troponin I plasma levels.

CHALLENGE

Current treatment options for reperfusion injury are limited and mainly include ischemic preconditioning, antioxidant therapies and anti-inflammatory medications. However, these approaches show only moderate efficacy and cannot specifically target the complex thromboinflammatory processes triggered by platelet-neutrophil complexes. New therapeutic approaches are urgently needed because despite successful reperfusion, up to 50% of patients still suffer significant myocardial damage.

INNOVATION

The invention is based on the observation that platelet – neutrophil complexes (PNCs) are increased in patients with acute myocardial infarction and that this is associated with increased levels of neuronal guidance protein semaphorin 7A (SEMA7A). By inhibiting SEMA7A, the myocardial tissue can be protected from reperfusion injury. SEMA7A inhibition offers for the first time a specific molecular target to interrupt the pathological interaction between red blood cells, platelets, and immune cells, thereby significantly reducing the extent of reperfusion injury.



01 Basic principles observed · **02** Technology concept formulated · **03** Experimental proof of concept · **04** Technology validated in lab · **05** Technology validated in relevant environment · **06** Technology demonstrated in relevant environment · **07** System prototype demonstrated in operational environment · **08** System complete and qualified

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Bayerische Patentallianz GmbH
Dr. Katrin Bercht
kbercht@baypat.de
+49 (0)89 5480177-16
Prinzregentenstr. 52
80538 München