

Emergence of a R-Type Ca^{2+} Channel Contributes to Cerebral Artery Constriction Following Subarachnoid Hemorrhage

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Overview

Cerebral aneurysm rupture and subarachnoid hemorrhage (SAH) inflict disability and death among thousands of individuals each year. The consequences of SAH following cerebral aneurysm rupture are devastating with mortality rates as high as 50% and the majority of survivors left with moderate to severe disability. Cerebral vasospasm, characterized as a delayed and sustained arterial constriction, is a major contributor to these high morbidity and mortality rates. Large diameter arteries have been implicated in contributing to decreased blood flow resulting in SAH. However, small diameter arteries, below the resolution limits of standard angiography, may also be affected by subarachnoid blood.

Invention

It has been discovered that SAH leads to enhanced Ca^{2+} entry in myocytes of small diameter cerebral arteries through the emergence of R-type voltage-dependent Ca^{2+} channels (VDCCs) encoded by the gene CaV 2.3.

The inventors believe that the administration of an R-type voltage-dependent calcium channel inhibitor will allow the management of the cerebral blood flow during a time when it would normally decrease blood flow in small diameter arteries. The emergence of CaV 2.3 in cerebral arteries following SAH is unique and the first to be described in the expression of this ion channel in vascular smooth muscle. A unique peptide has been identified that may selectively antagonize CaV 2.3 and therefore reverse subarachnoid hemorrhage-induced vasospasm.

Advantages

- Allows management of cerebral blood flow
- Potential to reverse SAH-induced vasospasm
- Can be administered by a variety of methods, at various times

Applications

- Sold as kit to hospitals, clinics
- Administered by I.V., orally, as a suppository, infused into cerebral spinal fluid, and more

I.P. Status

- US Patent #s [7,829,527](#) & [8,268,795](#)

Learn more about Dr. Wellman's research at:
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