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New strategy to treat brain bleeding

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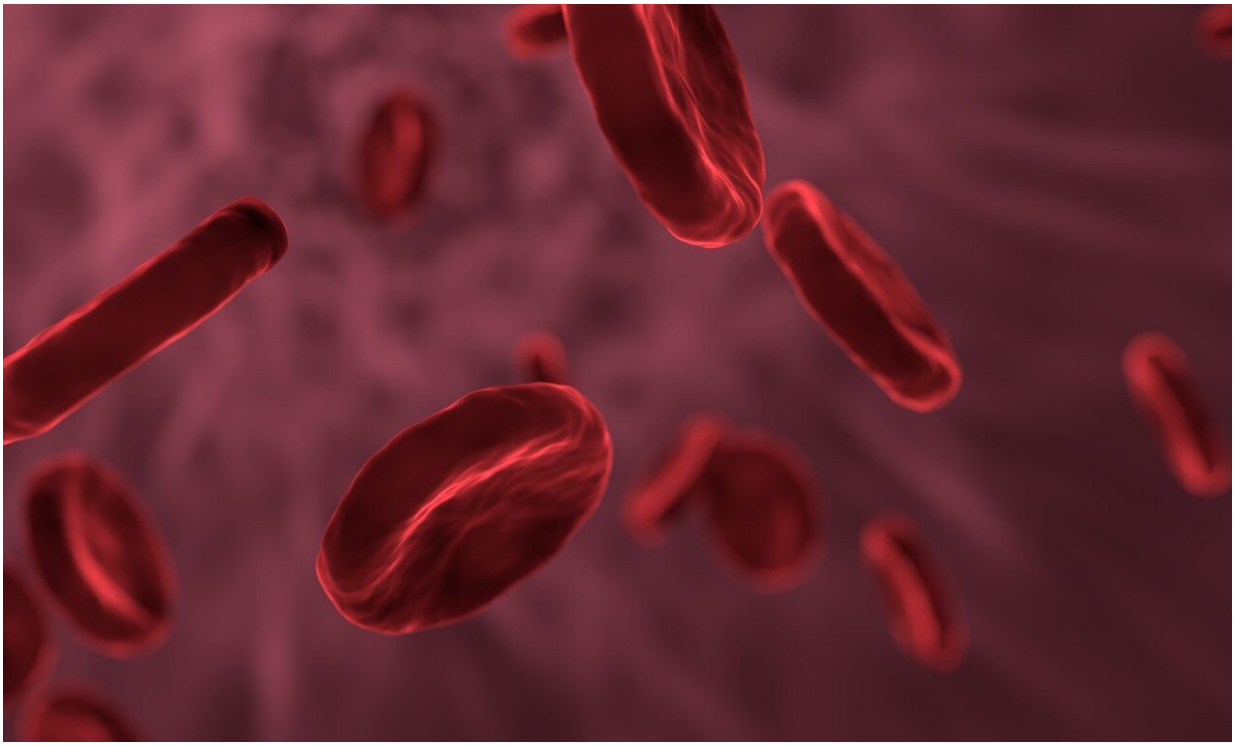

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Subarachnoid hemorrhage (SAH)—bleeding from a ruptured aneurysm—leads to delayed cerebral vasospasm (blood vessel constriction) and stroke. SAH morbidity and mortality are high, and therapeutic options are limited.

Joyce Cheung-Flynn, Ph.D., and colleagues proposed that SAH

downregulates the nitric oxide-protein kinase G (NO-PKG) signaling pathway that normally relaxes cerebral blood vessels.

Using a rat model, they confirmed reduced levels of NO-PKG pathway molecules, including the protein VASP, which modulates contractile machinery to cause vasorelaxation. They designed a family of cell permeant peptide mimics of activated VASP and demonstrated that the peptides caused vasorelaxation of vascular tissues ex vivo.

The findings, reported in the *European Journal of Pharmacology*, suggest that reduced NO-PKG signaling is an underlying mechanism of pathological vasoconstriction after SAH. Treatment with activated VASP peptides could be explored as a therapeutic strategy to reduce neurological deficits caused by SAH-induced vasospasm, the authors suggest.

More information: Peter J. Morone et al. Vasorelaxing cell permeant phosphopeptide mimetics for subarachnoid hemorrhage, *European Journal of Pharmacology* (2021). DOI: 10.1016/j.ejphar.2021.174038

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