



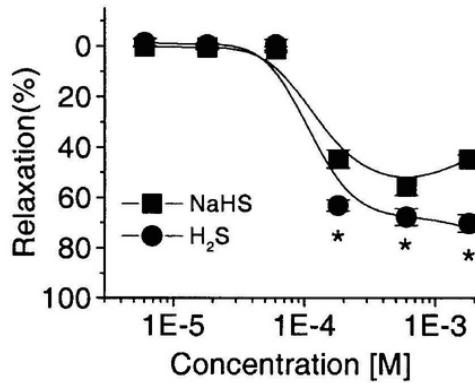
Potassium channels mediate hydrogen sulfide-induced cutaneous vasodilation in healthy young adults.

Jessica L. Kutz, Jody L. Greaney, Lacy M. Alexander, FACSM

Department of Kinesiology, The Pennsylvania State University,
 University Park, PA



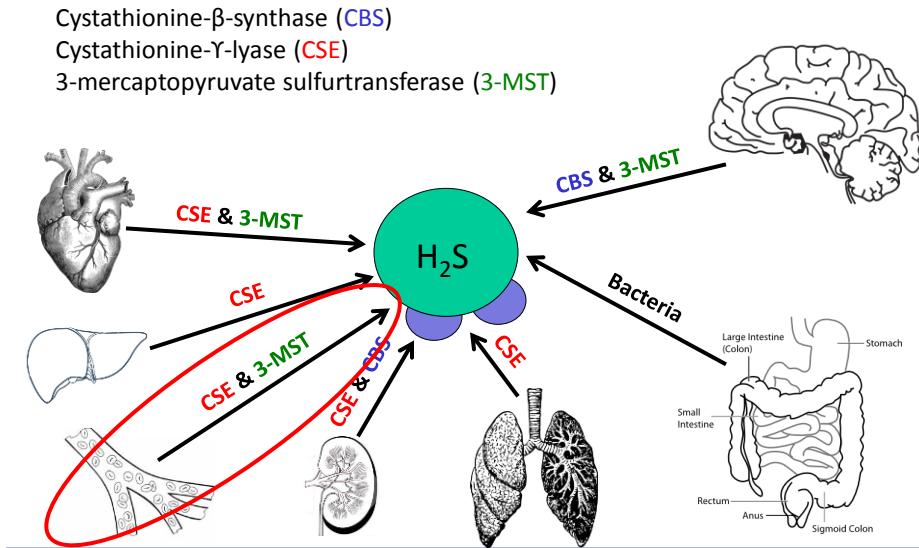
Preclinical models suggest hydrogen sulfide modulates vascular function.



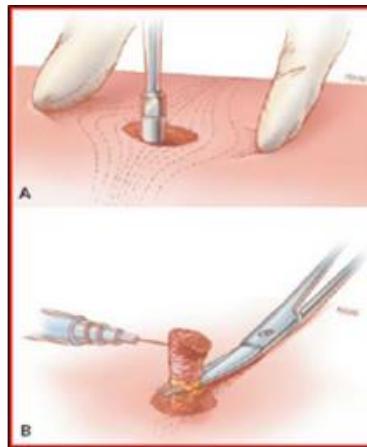
Zhao W, Zhang J, Lu Y, and Wang R. The vasorelaxant effect of H₂S as a novel endogenous gaseous K(ATP) channel opener. *The EMBO Journal* 20: 6008-6016, 2001.



Endogenous hydrogen sulfide production.

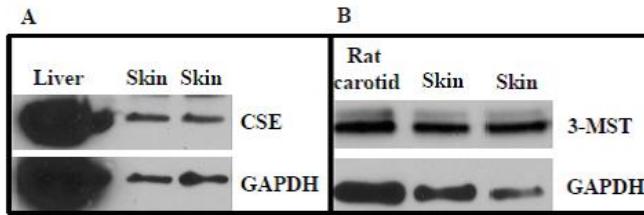


CSE and 3-MST are expressed in the cutaneous circulation of humans.

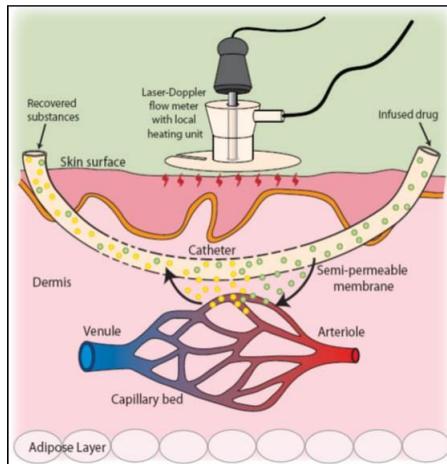




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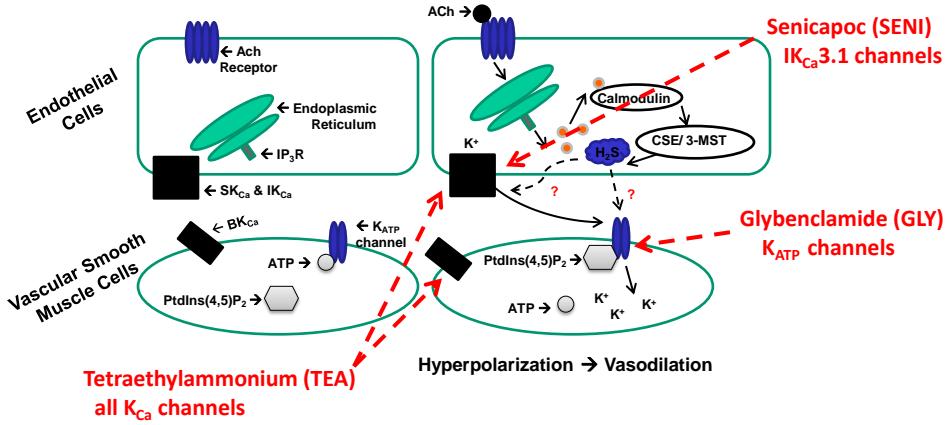
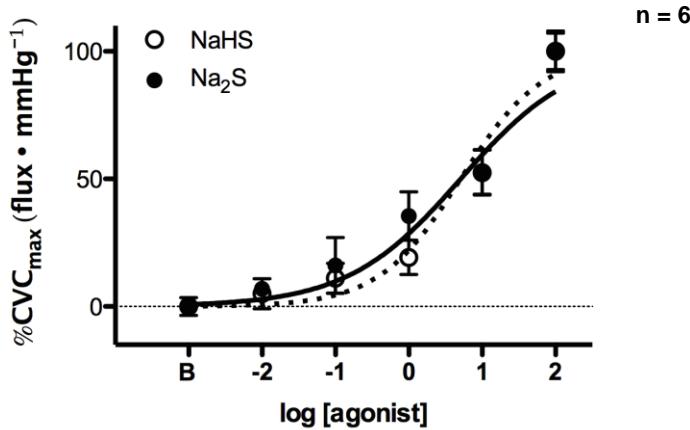


Hydrogen sulfide elicits vasodilation in the cutaneous circulation in a dose-dependent manner.





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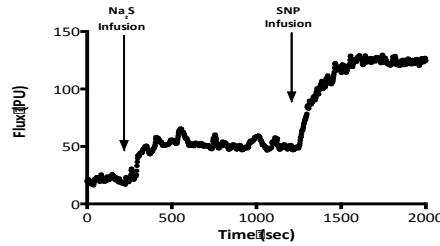


Hypothesis: Exogenous H₂S-induced cutaneous vasodilation is mediated by K_{ATP} channels.

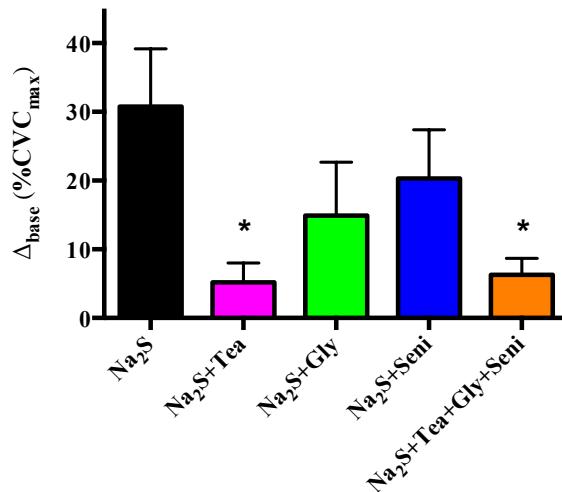


Protocol

	Insert Fibers 20 min	Hyperemia 60-90 min	Baseline 30 min	5 mM Na ₂ S 15 min	Flush Ringers 5 min	SNP + 43° C
Site 1	Ringers	Control: Ringers		+ Na ₂ S	Ringers	SNP
Site 2	Ringers	K _{Ca} Inhibited		+ Na ₂ S	Ringers	SNP
Site 3	Ringers	IK _{Ca} Inhibited		+ Na ₂ S	Ringers	SNP
Site 4	Ringers	K _{ATP} Inhibited		+ Na ₂ S	Ringers	SNP
Site 5	Ringers	Triple Blockade		+ Na ₂ S	Ringers	SNP

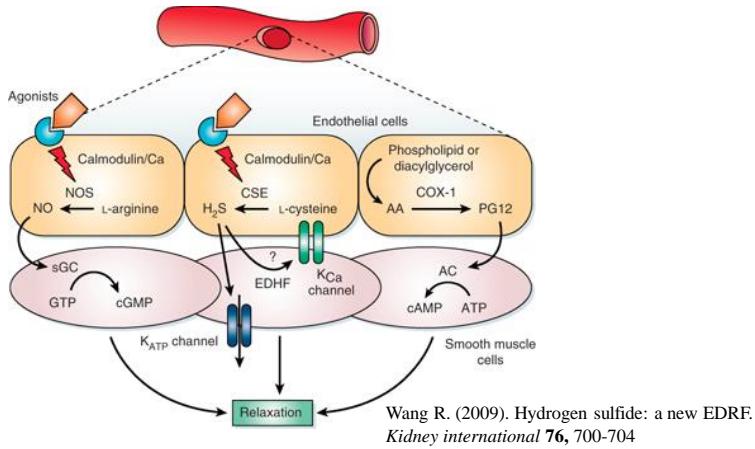


Exogenous H₂S-induced cutaneous vasodilation is mediated, in part, by TEA-sensitive K_{Ca} channels.

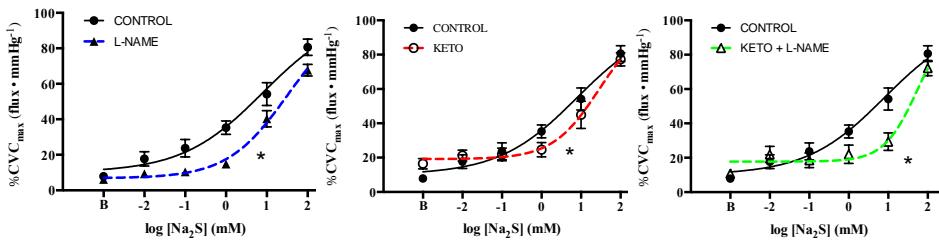




Both NO and by-products of COX contribute, in part, to exogenous hydrogen sulfide-mediated cutaneous vasodilation.



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In the cutaneous circulation of humans...

- CSE and 3-MST are expressed
- Exogenous H₂S donors elicit vasodilation in a dose-dependent manner
- K_{Ca} channels, at least in part, mediate H₂S induced vasodilation
- NO and downstream product of COX contribute to exogenous H₂S-mediated vasodilation