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Vasodilatory effect of asafoetida essential oil: the role of nitric oxide, prostacyclin, and calcium and potassium channels

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Previous studies have shown that asafoetida from Ferula assa-foetida L. species in the Apiaceae family has antispasmodic effects on rat's and pig's ileum. Thus we evaluated the effect of asafoetida essential oil (AEO) on the tension of isolated rat's aorta rings and the underlying cellular mechanisms, as well as the determination of its constituent by GC-MS. Male Wistar rat's aortic rings were placed in an organ bath and after 1 hour stability with 1 gram force, their tension assessed to obtain the IC50 of AEO in the presence and absence of endothelium. To determine the role of nitric oxide synthase (NOS) and cyclic oxygenase (COX), their blockers were used. To determine the role of potassium channels, they were blocked by barium chloride, 4-aminopyridine, and glibenclamide. To determine the role of intracellular and extracellular calcium influx, free calcium medium in the presence and absence of phenylephrine, respectively, was used. More than 50% of AEO constituents were disulfides. The data showed that IC50 of AEO was 1.6 µL/L and 19.2 µL/L in intact and denuded endothelium, respectively. The maximal force of contraction of rings in the presence of NOS and COX inhibitors and potassium channel blockers were significantly lower than that in groups exposed only to AEO. Adding AEO to the free calcium solution before adding calcium lead to lowering of the maximal force of contraction induced by KCL or phenylephrine. It is concluded that the vasodilatory effect of AEO is endothelium-dependent and non-dependent. Also, it is mediated through the inhibition of calcium channels and activation of potassium channels. It appears that disulfides might be responsible for the vasodilatory effect of AEO.

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