

Poster Abstracts

PO-57

MECHANISM OF HYPOTENSION IN *CLEISTANTHUS COLLINUS* POISONING

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Cleistanthus collinus is a common plant poison used for suicides in Tamil Nadu. Cleistanthin C is the major toxin in the boiled aqueous extract of *C. collinus*. Mortality rate is around 28% and death occurs 3-7 days after poison ingestion. Refractory hypotension due to vasodilatation is the major cause of death in patients.

While investigating vasodilatory mechanisms in a goat arterial strip, we identified a new signaling pathway in which, alpha adrenergic stimulation causes paradoxical vasodilatation in high NO environment (Plos One 2016). The vasodilatation was cGMP independent. It was demonstrated that a combination of L-Arginine/Phenylephrine (PE) induces vasodilatation, which is blocked with L-NNA or phentolamine, but not with propranolol. Here we report that Cleistanthin C acts through the new pathway, leading to vasodilatation, when there is simultaneous alpha adrenergic activation.

Objective:

To study the effects of Cleistanthin C on vascular tension in goat arterial strip

Method:

A section of a small artery was isolated from the goat leg and cut spirally. Spiral strip was suspended in an organ bath (25 ml), filled with physiological salt solution at 37°C, aerated with carbogen (95% O₂ & 5% CO₂). One end of the strip was attached to a force transducer connected to a data acquisition system (Power lab). Optimal preload was applied to keep the thread taut. Drugs were added to the organ bath; changes in tension were recorded and analyzed.

Results:

Cleistanthin C (100µM) *per se* did not produce any change in the vascular tension. But subsequent addition of PE (100µM) resulted in vasodilatation (n = 6, P = 0.028 with Wilcoxon signed rank (WSR) test) (Fig 1).

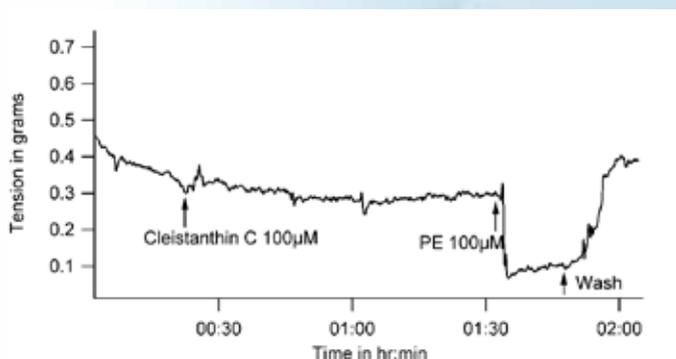


Fig 1: Representative tracing of tension recording in spiral strip of goat artery showing reduction in vessel tension with Cleistanthin C/ PE combination.

Cleistanthin C/ PE induced vasodilatation was abolished in the presence of L- NNA (1mM) (n=4)

Poster Abstracts

($P = 0.011$ with Mann-Whitney U test when tension after Cleistanthin C/ PE combination were compared with and without L-NNA). Cleistanthin C/ PE combination did not produce vasodilatation in the presence of prazosin ($10\mu\text{M}$) ($n = 5$, $P = 0.008$ with Mann-Whitney U test when tension after Cleistanthin C/ PE combination were compared with and without prazosin).

Conclusion:

Cleistanthin C creates an environment in which alpha adrenergic stimulation results in vasodilatation, which is nitric oxide-dependent.