
Departmental Seminar

Seminar Title	: Bacosine inhibits arecoline-induced inflammasome by activating autophagy through the NRF2-TFEB axis in oral cancer
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Venue	: LS Seminar Hall
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Abstract	: Numerous phytochemicals exhibit potent antioxidant activity, which is crucial in shielding cells from oxidative damage caused by reactive oxygen species (ROS). Amongst phytochemicals from various plants, recent studies have emphasized the antioxidant properties of <i>Bacopa monnieri</i> , a medicinal herb eminent for its cognitive-enhancing properties. The antioxidant properties of <i>Bacopa</i> are primarily accredited to its abundant content of bioactive compounds such as bacosides, flavonoids, and phenolic acids. This study demonstrated that Bacosine, a flavonoid from <i>Bacopa monnieri</i> , abates cellular ROS production and increases ROS-sensitive factor NRF2 and its downstream target. Additionally, bacosine treatment led to decreased arecoline-induced ROS production, which plays a critical role in the formation of an inflammasome. Herein, we demonstrate that bacosine treatment increases the number of LC3 puncta and autophagosome and induces autophagosome-lysosome fusion. Importantly, we find that bacosine enhanced the lysosomal activity by regulating the lysosomal biogenesis protein TFEB and induced autophagic flux. Interestingly, pretreatment with NRF2 inhibitor (ML-385) and genetic inhibition of NRF2 (siNRF2) suppressed the bacosine-induced autophagy in oral cancer cells. Further, bacosine suppressed arecoline-induced inflammasome activation through autophagy in oral cancer cells.