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College: COS

Co-presenter(s): Not applicable

Faculty Mentor(s): Dr. Nancy Buckley

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Project Title: Garlic and Allicin Modulate Candida Albicans-Induced Tumor Necrosis Factor Alpha (TNF-a) Secretion From J774A.1 Murine Macrophages

Synopsis: To characterize the effect of garlic on C. albicans-induced cytokine production from J774A.1 macrophages in vitro and identify the component within garlic responsible.

Abstract: Garlic is known to have antifungal properties. It is not well understood how garlic fights off yeast and which component in garlic modulates this property. Allicin is one of the main components in crushed garlic. We investigated the effect of garlic and allicin on macrophage response to heat killed Candida albicans (yeast). Macrophages, key cells in the innate immune response, produce cytokines such as Tumor Necrosis Factor alpha (TNF-a), a cytokine produced in response to pathogens like yeast. In this study, we characterized garlic’s effect on yeast-induced TNF-a secretion from macrophages, and if allicin was responsible for this effect. Macrophages were challenged with yeast and treated with garlic and allicin. The cells were then incubated at 37oC for various hours after which cell supernatants were collected. TNF-a levels were determined via enzyme-linked immunosorbent assay. Yeast-induced TNF-a secretion increased overtime, peaking at 2 days and garlic suppressed the yeast-induced TNF-a secretion. To determine whether allicin within the garlic caused the effects, garlic was incubated at 37oC for up to 5 days. HPLC revealed that the allicin content in garlic decreased under these conditions. Incubated garlic did not have an effect on yeast cytokine production, suggesting other components in addition to allicin might be responsible for the observed garlic effect. Allicin seems to increase yeast-induced TNF-a production compared to yeast alone, further suggesting that allicin does not work alone in modulating the immune system in this model. These results show that garlic modulates macrophage TNF-a secretion for which allicin is partially responsible.