

2015

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Smith, Kia, "Nitrosylation of S100A8/ A9 protein complex by inducible Nitric Oxide Synthase" (2015). *Undergraduate Research Posters 2015*. 21.

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Nitrosylation of S100A8/ A9 protein complex by inducible Nitric Oxide Synthase

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Abstract

How does the body fight infection? What causes inflammation? These are only a few questions that have been asked by many doctors across the globe. One thing that is known for certain about infection is that there is a particular sub-group of enzymes called NOS's (eNOS, nNOS and iNOS) that produce Nitric Oxide (NO), a molecule radical capable of oxidizing proteins and alter their function. In our study we focus on inducible Nitric Oxide Synthase (iNOS). NOS's are comprised of oxidase and reductase domains linked by Camodulin (CAM), a polypeptide linker. According to prior studies and SEM photographs, inducible Nitric Oxide's input and output states were analyzed showing the enzyme as highly malleable molecule virtually creating a shape-shifting enzyme capable of various shapes, however, the internal placement of the oxidase and reductase domain within iNOS is not clearly understood. As a result, the method of transport for NO was not revealed. This enzyme is responsible for the releasing of NO throughout the body when activated. NO then targets sites of infection and as a result, causes inflammation in the effected area. The true question is, what method of transport does iNOS use to safely transfer NO (a protein nitrosylating agent) throughout the body as NO is known to be a highly reactive substance. Our computational research study utilized a protein-protein docking program (PatchDock) and a molecular visualizer (PyMol) in order to gain a digital grasp of the transfer path of the NO molecule to NO-carrier proteins like S100A9.

**Supported by the McNair Scholars Program*