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# Research Highlights Protein's Role in Cell Health

June 9 2010, by Jennifer Nachbur

(PhysOrg.com) -- Simple, but necessary human actions like breathing oxygen can damage our cells' DNA. If left unrepaired, this damage can cause multiple mutations and the type of unregulated cell division common in cancerous tumor formation. Fortunately, our body is equipped with special proteins designed to deal with these injuries.

Gaining an understanding of how these enzymes recognize DNA damage and how to increase this repair function could help decrease the DNA damage that results in the development of cancer.

The DNA repair system includes glycosylases — a family of maintenance enzymes that are responsible for "patrolling" DNA strands in search of damage and have the ability to remove these lesions. Each of these glycosylases has a very different and specific task.

Recent research published by Susan Wallace, Ph.D., University of Vermont professor and chair of microbiology and molecular genetics, and colleagues at UVM, University of Utah, Vanderbilt University and National Institute of Standards and Technology, reported new findings regarding a key protein involved in DNA repair.

The Wallace lab, for the first time, was able to purify fully active NEIL3 and her team discovered never-before-identified activities specific to this protein. Wallace and colleagues confirmed that NEIL3 is one of these DNA-maintenance enzymes and determined that it was larger and more complicated than its related enzymes NEIL1 and NEIL2, both of

which were previously discovered through UVM research.

The researchers discovered that NEIL3 is highly successful at removing a lesion called FapyGua, which was also found to have a potential role in causing dangerous DNA mutations. According to Wallace, earlier research by a separate group demonstrated that the NEIL3 protein is predominantly found in stem cells, where scientists presumed it was responsible for repairing DNA damages in dividing cells.

As an internationally-recognized DNA repair expert, the more Wallace learns about these repair functions, the better able she and others are to figure out how to increase these repairs, thus helping to decrease the potential for the end product of DNA damage — cancer.

Provided by University of Vermont

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