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Red, red wine: How it fights Alzheimer's

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(PhysOrg.com) -- Scientists call it the "French paradox" — a society that, despite consuming food high in cholesterol and saturated fats, has long had low death rates from heart disease. Research has suggested it is the red wine consumed with all that fatty food that may be beneficial — and not only for cardiovascular health but in warding off certain tumors and even Alzheimer's disease.

Now, Alzheimer's researchers at UCLA, in collaboration with Mt. Sinai School of Medicine in New York, have discovered how red wine may reduce the incidence of the disease. Reporting in the Nov. 21 issue of the *Journal of Biological Chemistry*, David Teplow, a UCLA professor of neurology, and colleagues show how naturally occurring compounds in red wine called polyphenols block the formation of proteins that build the toxic plaques thought to destroy brain cells, and further, how they reduce the toxicity of existing plaques, thus reducing cognitive deterioration.

Polyphenols comprise a chemical class with more than 8,000 members, many of which are found in high concentrations in wine, tea, nuts, berries, cocoa and various plants. Past research has suggested that such polyphenols may inhibit or prevent the buildup of toxic fibers composed primarily of two proteins — Aß40 and Aß42 — that deposit in the brain and form the plaques which have long been associated with Alzheimer's. Until now, however, no one understood the mechanics of how polyphenols worked.

Teplow's lab has been studying how amyloid beta (Aß) is involved in

causing Alzheimer's. In this work, researchers monitored how Aß40 and Aß42 proteins folded up and stuck to each other to produce aggregates that killed nerve cells in mice. They then treated the proteins with a polyphenol compound extracted from grape seeds. They discovered that polyphenols carried a one-two punch: They blocked the formation of the toxic aggregates of Aß and also decreased toxicity when they were combined with Aß before it was added to brain cells.

"What we found is pretty straightforward," Teplow said. "If the Aß proteins can't assemble, toxic aggregates can't form, and thus there is no toxicity. Our work in the laboratory, and Mt. Sinai's Dr. Giulio Pasinetti's work in mice, suggest that administration of the compound to Alzheimer's patients might block the development of these toxic aggregates, prevent disease development and also ameliorate existing disease."

Human clinical trials are next.

"No disease-modifying treatments of Alzheimer's now exist, and initial clinical trials of a number of different candidate drugs have been disappointing," Teplow said. "So we believe that this is an important next step."

Source: University of California - Los Angeles