New study tries to link Alzheimer's disease and DDT; media thinks it succeeded

By ACSH Staff — January 28, 2014

A small biomonitoring study of Alzheimer's disease (AD) patients exposure to DDT, as compared to those of non-AD patients, [1] came up with some statistically significant associations of otherwise no clinical significance. But that didn't stop the news media from blaring the findings hither and yon, without giving a moment's thought to the underlying mechanisms or significance. As usual.

A group of researchers, led by Jason Richardson, Ph.D., of the Rutgers-Robert Wood Johnson Medical School, Dept. of Environmental and Occupational Medicine, matched levels of DDE, a breakdown product of the long-banned insecticide DDT, among 86 AD and 79 control patients. These levels were measured in serum.

DDT was banned in the USA and most of the western world thereafter in 1972, based on fears of bird toxicity and because of the nascent environmentalist movement's bible, Rachel Carson's Silent Spring, which targeted DDT. The scientific basis of this ban was essentially nothing, but the ban, fomented by the legions of Carson acolytes (including the EPA's administrator, William Ruckelshaus), led to the loss of the most effective insecticide countering the scourge of malaria in Africa and Asia.

The chemical (the discovery of which won the Nobel Prize for Medicine for its main researcher, Dr. Paul Muller, in 1948) is persistent (meaning it does not break down rapidly in the environment), as is DDE. But the levels measured in the study subjects were in the nanogram per milligram of cholesterol range: where a nanogram is one-millionth of a milligram! Simply put, the levels of DDE were somewhat akin to a drop of water in an Olympic-sized swimming pool or less.
The results, such as they are, indicated that the measured levels of DDE were 3.8 fold higher in the AD patients than the controls. Does this mean that the DDT/DDE caused AD in those higher-exposed? Not at all. In fact, the 2 study groups were assembled in 2 different locations, and each group's numbers failed to show any effect. The authors took care of that inconvenient problem by pooling both groups, and voila! the statistics came back to them as they hoped.

But while that teeny-tiny amount may make this whole endeavor ridiculous, even more so is this simple fact: while the amount of DDT/DDE in the environment has clearly declined since it was banned and its manufacture nearly disappeared forty-plus years ago, the incidence of AD has climbed, indeed accelerated over that same period. That's tough to explain using the DDT linked to Alzheimer's scare story. Isn't it? Also, can you postulate the likely biological hypothesis for how these chemicals infiltrate one's brain and interfere with memory on a progressive basis? No? Neither can I.

Heather Snyder, the Alzheimer's Association's director of medical and scientific operations, told USA Today's Liz Szabo that [T]here are good data to suggest that people can reduce their risk of Alzheimer's disease by making certain lifestyle changes -- such as exercising, avoiding tobacco and eating a healthy, balanced diet.

Studying environmental exposures could help scientists better understand Alzheimer's, Richardson says. "That's important, because environmental exposures are things you can do something about." ACSH's Dr. Gil Ross noted the above comments by the author and Ms. Snyder, and had this epiphany: That is exactly why this study was done: to try to discover some--any--remediable factor to try to prevent AD. Otherwise, we just feel helpless and at the mercy of fate. But that's a poor excuse for twisting yourself into a pretzel to come up with some bizarre linkage such as this study. And then there's this insinuation that all pesticides are alike, which is utter nonsense.