

Scientists uncover free radical clue to dementia

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A computer model programmed by scientists at Newcastle University suggests that preventing damage from free radicals could be key to fighting dementia.

Free radicals are molecules known to cause cell damage and this study sheds new light on the processes that cause cells to die in Alzheimer's disease and other brain disorders, which might help treatment development.

Dr. Carole Proctor, at Newcastle University, is investigating how toxic proteins build and cause damage in the brain. In Alzheimer's disease, two culprit proteins called amyloid and tau build and stop nerve cells from working properly, while different proteins are involved in other forms of dementia. The new [computer model](#) is able to mimic how these proteins accumulate.

In a study, funded by Alzheimer’s Research UK, Dr. Proctor and her team investigated the ‘chaperone’ system, which helps to stop the build-up of proteins and prevents cell death. Using the computer simulations, they tested how the system responded to stress caused by [free radicals](#).

Their simulations predicted that although the chaperone system can cope with short periods of stress, over long periods of high stress, the system becomes overloaded and the mechanisms that lead to cell death are activated. The researchers now want to expand and develop the computer model to better understand the processes involved in Alzheimer’s disease. It’s hoped the method will help find new ways to prevent protein build-up and to stop brain cells dying.

The researchers also believe their findings, which are published in the latest edition of *PLoS ONE*, suggest that lifestyle changes to prevent damage from free radicals may help fight dementia and other brain disorders. Such changes could include eating a healthy diet, with plenty of fruit and vegetables, and taking moderate exercise to boost the body’s antioxidant defences.

Dr. Proctor said: “The predictions made by our computer model now need to be tested in the lab, but we hope our findings will give researchers some important new leads to follow. Being able to simulate the processes involved in dementia allows us to better focus our research, giving us a better chance of finding ways of intervening to prevent dementia.

“Our findings suggest that damage from free radicals plays a key role in the build-up of toxic proteins in the brain. We now need to see more research to examine whether reducing this damage – for example, by taking moderate exercise and eating a healthy diet – could help protect against dementia. Research is the only way we will defeat dementia, and I hope these findings will take us further towards that goal.”

Dr. Simon Ridley, Head of Research at Alzheimer’s Research UK, said: “This study provides us with new clues for research, and it’s important that we follow this up to find out more about what causes brain cells to die in dementia. We need to see more research to find out whether reducing damage from free radicals could have a protective effect.

“With 820,000 people in the UK affected by [dementia](#), and nearly 3,000 people in Newcastle alone, research is vital if we are to find preventions and new treatments that are so desperately needed.”

More information: [Modelling the role of the Hsp70/Hsp90 system in the maintenance of protein homeostasis](#), C Proctor & I Lorimer, is published online in *PLoS ONE*.

Provided by Newcastle University

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