

Physical activity delays onset of Huntington's in mouse model

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The simple act of running in an exercise wheel delays the onset of some symptoms of Huntington's disease in a mouse model of the fatal human disorder according to research published in the open-access journal *BMC Neuroscience*. These findings add insights into the pathogenesis of the disease and suggest possible preventive therapeutic targets.

Huntington's disease affects up to one person in every 10 000, but clusters in families and certain populations. Affected people develop clusters of a defective protein in their neurons and shrinkage of brain areas associated with movement. The disorder causes disability and eventually death, but does not normally manifest until after people have had children, allowing the disease gene to be passed on.

“Although Huntington's disease is considered the epitome of genetic determinism, environmental factors are increasingly recognised to influence the disease progress”, the researchers write.

The research team from the University of Oxford and the Howard Florey Institute, University of Melbourne, report findings of a study in mice with the genetic mutation that causes Huntington's in humans. Just as mentally stimulating these mice by enriching their environment had previously been shown to delay onset and progression of motor symptoms, so does the simple physical activity of running in a wheel.

“Of particular interest was the fact that the wheel exercise was started in juvenile mice, much earlier than in a previous study that showed more

limited protective effects of physical activity”, explains Anthony Hannan of the Howard Florey Institute. This finding suggests that the protective effect has a specific time window.

Hannan notes “Physical activity did not postpone all the motor symptoms delayed by environmental enrichment, which suggests that sensory stimulation, mental exercise, and physical activity could all be used for the benefit of human sufferers”. Early intervention is also possible in people who will develop Huntington’s, because genetic diagnosis is possible.

Density of protein aggregates in neurons and shrinkage in brain regions in mice that had benefited from physical activity were as advanced as in those raised without wheels, the authors suggest therefore that benefits stem from stimulation of neuronal receptors and other molecules that prolongs normal function and delays motor deficits.

Source: BioMed Central

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