The dementia protection hiding in plain sight

Vaccination during adulthood is associated with significantly reduced risks of dementia. The evidence is as powerful as it is surprising.

A large study found that adults aged 65 and over who had received the adult tetanus, diphtheria and pertussis (whooping cough) vaccine had a 42 per cent lower risk of developing dementia during follow-up. Pneumococcal vaccination is associated with nearly a 30 per cent reduction in dementia incidence. Evidence from nearly 300,000 older people who started off dementia-free showed a 30 per cent reduction in the risk of dementia over the next ten years if they'd had the influenza vaccine. A review of over 100,000 people found around a 24 per cent reduction in dementia incidence in those immunised against shingles (Varicella zoster).

It's hard to find another intervention that's associated with such powerful effects, and the evidence suggests that the benefit starts from about the age of 50. It could be earlier. All of the above findings allow for other factors that could explain the effect, such as differences between people who are more or less likely to be vaccinated in the first place.

The question is why?

Varicella zoster virus is a herpes virus, and researchers have long wondered whether herpes infections are a trigger for cognitive decline leading to dementia, given their affinity for the nervous system. At least one of the ways the zoster vaccine works to prevent dementia appears to be by keeping the virus at bay.

But there also seems to be a separate effect on the immune system, and this may help explain why influenza, pneumococcal, and tetanus/whooping cough vaccines appear to have a preventive effect as well. The leading theory is that chronic low-grade inflammation, driven in part by persistent viral activity, is a key pathway in dementia development. Vaccination may reduce that inflammatory burden over time, offering protection that goes well beyond the infections they were designed to prevent.

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COVID-19 is relevant here too. A meta-analysis of over 800,000 people found that 65 per cent of those aged 65 and over who were hospitalised with COVID-19 developed new symptoms of cognitive impairment. A UK Biobank study comparing people aged over 50 who had COVID with those who had not found a 58 per cent increased risk of all-cause new onset dementia, particularly vascular dementia. COVID-19 vaccination reduces the risk of Long COVID significantly, and while direct evidence on cognitive outcomes is still emerging, the overall picture strongly favours vaccination.

With flu season underway, it is worth having these conversations proactively, particularly with adults over 50 who may be behind on pneumococcal, shingles, or Tdap vaccines. The dementia prevention angle gives the conversation a new dimension that many people have not yet considered.

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What exercise does to your biological age

When you hear about billionaire biohackers claiming they are years younger than their chronological age, they are sometimes referring to their epigenetic clocks. These are measures of chemical reactions around our genes called DNA methylation, which can turn genes off or change their level of activity. The most reliable clocks have been calibrated against large populations of people whose health and wellbeing are tracked in detail over time.

The result is a tool that can measure your DNA methylation and indicate, on average, how typical that pattern is for a person of a certain age. You might be 50 but your DNA methylation might resemble that of a 48-year-old or a 53-year-old. That gap between chronological and biological age turns out to matter quite a lot for health outcomes.

These clocks have made it possible to study whether specific interventions are associated with being biologically younger or older, in a way that goes beyond the usual self-reported health measures. A recent meta-analysis published in The Lancet brought together 44 studies of exercise involving over 145,000 people to ask exactly that question.

What they found was that the more exercise people did, the younger their biological age. The association held across different types of exercise, different age groups, and different populations. People who exercised regularly showed DNA methylation patterns that were, on average, several years younger than their non-exercising counterparts of the same chronological age.

The important caveat is that the study could not prove cause and effect, and many of the individual associations across the 44 studies did not reach statistical significance. People who choose to exercise may be biologically younger to begin with, for reasons unrelated to the exercise itself. Genetics, socioeconomic factors, diet, sleep, and a range of other variables all influence both exercise behaviour and biological ageing. Further research, ideally using randomised designs, is needed to establish how much of the effect is directly caused by exercise and how much is explained by the kinds of people who exercise.

That said, the consistency of the finding across a very large, combined sample is not easily dismissed. Exercise is already associated with reduced risks of cardiovascular disease, type 2 diabetes, depression, and a range of cancers. The possibility that it is also slowing biological ageing at a cellular level adds another dimension to a conversation that GPs are already well placed to have.

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AI chatbots and youth mental health

A US survey of young people aged 12 to 21 found that nearly one in five have used AI chatbots for assistance with mental health issues. Most of them kept it to themselves. Many found it useful. Around 43 per cent used chatbots for mental health advice at least monthly, and many had already seen a doctor. The older the young person, the more likely they were to have used AI for this purpose. Females were more likely to use it than males.

This could be good news, if the advice being given is reliable and the systems have adequate guardrails in place for young people who may be at risk of self-harm. The trouble is that AI chatbots as mental health interventions have not been well studied, and there have been high-profile cases of young people finding ways around inbuilt safety systems, with the chatbot subsequently facilitating rather than preventing harm.

The problems with AI chatbots in this context are more fundamental than safety settings. The way these systems are trained to conduct therapeutic-style conversations is not transparent. More importantly, chatbots are designed to be friendly, engaging and empathic, whereas clinical psychologists are trained to be objective and focussed on the real problems of the person, steering them towards a clear therapeutic strategy. These are not the same thing, and in some presentations, they are directly at odds. A system optimised for engagement may keep a distressed young person talking without ever moving them towards help.

There is also the question of what happens when a young person develops a reliance on a chatbot as their primary source of emotional support. Research on parasocial relationships with AI is still in its early stages, but the concern is that chatbot interaction may substitute for rather than supplement professional help-seeking and real-world connection, particularly in young people who are already socially withdrawn or anxious.

For general practice, the practical implication is straightforward but easy to overlook. Young people presenting with mental health concerns may already be using AI tools regularly, and they are unlikely to volunteer this information. Asking directly and non-judgementally about AI use opens the conversation. The goal is not to discourage it categorically, but to understand what they are using, how they are using it, and whether it is complementing or replacing professional support.

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