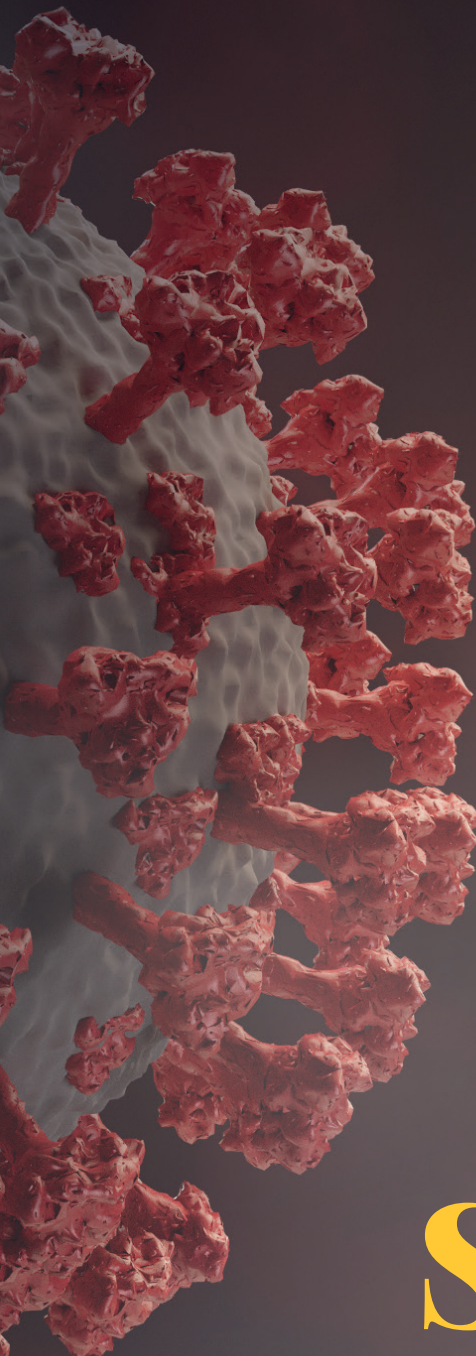


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Silent threat

Hospital Sungai Buloh Infectious Diseases Senior Consultant Datuk Dr Suresh Kumar emphasises human metapneumovirus (hMPV) typically presents more serious health risks for young children, especially those under five. **p12-13**

Solat and joints health. **p10**

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Call for vigilance. **p12-13**



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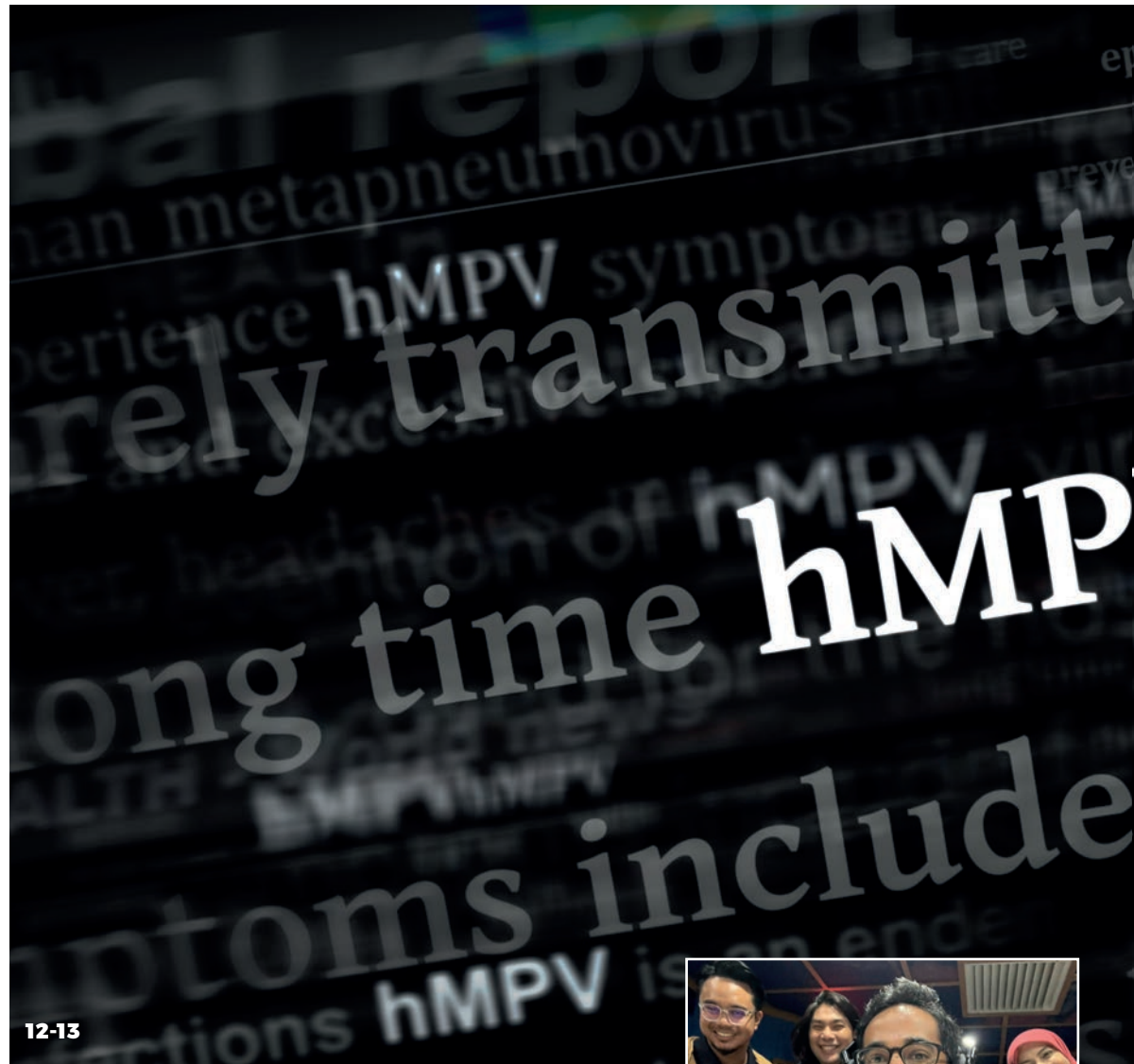
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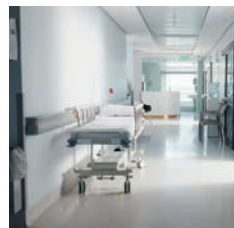
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SMART PARTNERSHIP



Inhale, Exhale, Protect

The surprising science behind smoking and Parkinson's disease



BRAIN BITES

BY DR WAEL MY MOHAMED

Dr Wael MY Mohamed is with the Department of Basic Medical Science, Kulliyah of Medicine, International Islamic University Malaysia (IIUM).

As Mark Twain famously quipped, "Too much of anything is bad, but too much good whiskey is barely enough." Perhaps the same logic applies to nicotine - where just the right amount might have unexpected benefits. Or, as Twain also joked, "Quitting smoking is easy. I've done it thousands of times." - a paradox that mirrors the surprising link between smoking and Parkinson's risk!

INTRODUCTION

THE negative correlation between cigarette smoking and the incidence of Parkinson's disease (PD) has been a persistent conundrum in epidemiology.

Numerous epidemiological studies have consistently shown a reduced frequency of Parkinson's disease among smokers, prompting fascinating enquiries into the possible neuroprotective properties of tobacco smoke constituents.

Nonetheless, the processes that underpin this relationship are little understood, and whether smoking itself provides protection or whether other explanations account for this phenomenon continues to be a subject of contention.

Epidemiological Data. Numerous extensive cohort and case-control studies have shown that smokers have a reduced risk of getting Parkinson's disease compared to non-smokers.

A meta-analysis of epidemiological data indicates that both present and past smokers demonstrate a decreased incidence of Parkinson's disease, with a dose-dependent relationship showing that increased smoking intensity and duration correlate with a lower risk.

This inverse association has prompted researchers to hypothesise that specific components of tobacco smoke may possess neuroprotective properties, possibly postponing the development or mitigating the severity of Parkinson's disease pathology.

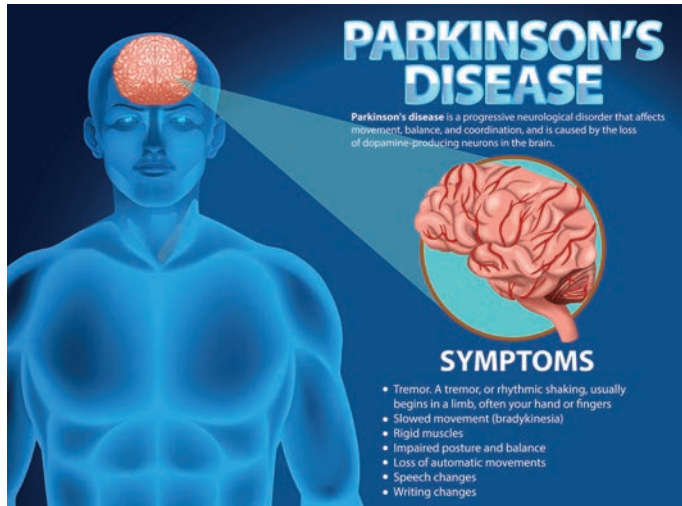
SMOKING AND PD

Epidemiological studies have strangely associated cigarette smoking with a decreased incidence of Parkinson's disease (PD), but the underlying processes are not well understood.

Considering that carbon monoxide (CO) levels are consistently but slightly higher in smokers, we hypothesised that CO may have a role in neuroprotection in Parkinson's disease (PD).

Employing mouse models of Parkinson's disease distinguished by α -synuclein buildup and oxidative stress, researchers show that low-dose carbon monoxide alleviates neurodegeneration and diminishes α -synuclein pathology.

The oral injection of carbon monoxide activates signalling cascades controlled by heme oxygenase-1 (HO-1), an enzyme generated by stress that regulates oxidative stress and facilitates



ish neuroinflammation, and provide protection against oxidative stress - mechanisms pertinent to the aetiology of Parkinson's disease.

Nonetheless, despite these encouraging pre-clinical results, recent clinical studies examining nicotine replacement treatment in Parkinson's disease patients have not shown substantial disease-modifying benefits, raising questions about nicotine's significance as a principal protective component.

A further probable factor in the observed adverse relationship is carbon monoxide (CO), a by-product of tobacco combustion. At low concentrations, carbon monoxide (CO) has been shown to have cytoprotective benefits by activating heme oxygenase-1 (HO-1), an enzyme that mitigates oxidative stress and promotes protein breakdown.

Recent research using mouse models of Parkinson's disease indicates that low-dose carbon monoxide injection reduces α -synuclein aggregation and neuronal degeneration, offering a credible molecular rationale for the epidemiological observations.

Although biological processes provide plausible explanations, competing ideas must also be evaluated. One explanation is the "healthy smoker effect", which posits that persons susceptible to Parkinson's disease may be less inclined to smoke long-term owing to early, subclinical neurological symptoms that influence habit building.

Moreover, genetic and lifestyle variables obscure the association between smoking and Parkinson's disease, confounding causal interpretations.

CONCLUSIONS AND DIRECTIONS

Despite epidemiological statistics indicating a reduced incidence of Parkinson's disease among smokers, the precise processes are unclear, and smoking is not a feasible preventative approach owing to its established health hazards.

Future study should concentrate on finding and isolating certain protective chemicals in tobacco smoke and exploring their therapeutic potential in Parkinson's disease models. A more comprehensive knowledge of these pathways may enable the creation of tailored neuroprotective therapies devoid of the detrimental consequences linked to smoking. - **The HEALTH**

α -Syn breakdown, therefore providing neuroprotection.

The results demonstrate that low concentrations of CO - similar to those seen in smokers—provide neuroprotection in PD models by decreasing α -Syn buildup and stimulating pathways that alleviate oxidative stress.

The findings indicate that molecular processes activated by low-dose CO may decelerate PD development, necessitating more research into its therapeutic potential. A clinical study assessing the effectiveness of low-dose carbon monoxide treatment in Parkinson's disease patients is now under development.

Additionally corroborating this idea, this research revealed increased concentrations of HO-1 in the cerebral fluid of smokers relative to non-smokers. Furthermore, in post-mortem brain tissue from Parkinson's disease patients, HO-1 expression was elevated in neurones without α -Syn pathology.

The results indicate that molecular pathways stimulated by low-dose CO may postpone disease onset and mitigate PD pathogenesis. Clinical research evaluating orally delivered low-dose carbon monoxide in Parkinson's disease patients is proposed, given the demonstrated safety of low-dose carbon monoxide in several Phase 1 and Phase 2 clinical trials across diverse diseases.

MECHANISMS OF NEUROPROTECTION

Nicotine, a major constituent of cigarette smoke, has been thoroughly investigated for its possible neuroprotective effects. It interacts with nicotinic acetylcholine receptors (nAChRs), which have a role in regulating dopaminergic neurotransmission.

Research involving animals and in vitro experiments indicates that nicotine may augment dopamine release, dimin-