Letters

Depression–obesity link in children

I read with interest the excellent article, ‘Depression in children’, by Julian Charles and Mandana Fazeli that was published in Australian Family Physician (AFP December 2017).¹ There is one obesogenic aspect worth mentioning. A recent meta-analysis of 51,272 study participants found that a positive association exists between childhood and adolescent obesity and depression. The pooled odds ratio for obese individuals was 1.34 (95% confidence intervals [CI]: 1.10, 1.64, P = 0.005) when compared with normal-weight peers.² As early as 2015, the research group of Sanders et al also showed in a systematic review that overweight and obese children in Australia were significantly more likely to have depression than normal-weight children.³ Another meta-analysis of longitudinal studies from the University of Queensland has concluded that adolescents with depression have a 70% increased risk (relative risk [RR] 1.70, 95% CI: 1.40, 2.07) of being obese, and adolescents who are obese have a 40% greater risk of having depression (RR 1.40, 95% CI: 1.16, 1.70).⁴

General practitioners (GPs) should be aware of this depression–obesity link and regularly monitor waist circumference because this is significantly related to depressive symptoms in children.⁵ Finally, the most recent measured data showed that 27.6% of Australian children were overweight or obese in 2014–15.⁶ Therefore, I absolutely agree with the authors that family and lifestyle support is essential for the affected children in the long term. GPs’ interventions should help children increase their physical fitness (specifically muscular strength) and integrate protective healthy dietary patterns (high in vegetables, fruits, legumes, fish and whole-grain foods) into their daily lives.⁷,⁸ For example, a simple and effective tip from GPs for creating a long-term behavioural change would be: ‘Drink a large glass of water before each meal’.

The following biological mechanisms play an important part in the bi-directional associations between obesity and depression in children and adolescents: hypothalamic–pituitary–adrenal axis dysregulation; altered plasma levels of cortisol, leptin, neuropeptide Y, adiponectin, resistin, orexin-A, orexin-B and insulin; inflammatory cytokines and signals such as tumour necrosis factor-alpha, interleukin-1 beta, interleukin-6, nuclear factor kappa B and C-reactive protein; and gut microbiome diversity.²,⁹

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References

Reply

Our article ‘Depression in children’ was focused on our understanding of the major areas relating to this topic.¹ There was much we were unable to elaborate on. Therefore, I welcome Dr Martin Hofmeister’s letter to the editor, which raised the important issue of the link between obesity and childhood depression. Dr Hofmeister clearly summarises the growing and compelling evidence linking childhood obesity with depression symptomatology and some of the proposed biological mechanisms underlying this link. Dr Hofmeister also offers great advice to the general practitioner (GP) in the consulting room, including, importantly, the type of diet considered to be protective against depression.

I would like to continue the discussion by suggesting that a focus solely on obesity can be problematic. There is extensive and consistent data from around the world showing that diet quality is also an important determinant of mental health in children and adolescents, as well as in adults.² Systematic reviews in adult populations (where much of the evidence exists) have demonstrated consistent inverse relationships between a high-quality diet and depression.³,⁴ A systematic review specific to the child and adolescent population also reported an inverse relationship between a high-quality diet and mental health disturbances, and a positive relationship between unhealthy diets and poorer mental health outcomes.⁵ Of note, data showing that diet quality is related to mental health independently of body weight suggest that dietary patterns...
can affect mental health via pathways that are independent of weight status. Emerging evidence from animal studies suggests that unhealthy diets, via the gut microbiota, induce behavioural changes and neuroinflammation well before any impact on body weight is detected.

An additional risk that comes with a sole focus on weight is that once people are overweight, it is very difficult to reverse. We need to be focusing on diet quality, in addition to addressing obesity, because of its direct relationship with brain plasticity, inflammation, oxidative stress and gut health.

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References

Tinnitus patients: Do you smoke?

I congratulate Aaron A Esmaili and John Renton for their very interesting review of tinnitus in *Australian Journal of General Practice* (AJGP April 2018).

There is one modifiable lifestyle aspect worth mentioning. Tobacco smoking can probably promote the development of tinnitus and increase existing tinnitus. The evidence is sufficient to include smoking among the potential risk factors for tinnitus. In a recent pooled meta-analysis of 20 studies, the odds ratio (OR) of tinnitus among current smokers was 1.21 (95% confidence interval [CI]: 1.09, 1.35) and 1.13 (95% CI: 1.01, 1.26) in former smokers. Furthermore, the research group of Veile et al observed a significantly increased risk for severe tinnitus in the sensitivity analyses of current smoking (OR 1.32, 95% CI: 1.10, 1.58). Another recent small cross-sectional survey of 678 college students aged 18–30 years revealed a higher prevalence of acute tinnitus (OR 3.08, 95% CI: 1.37, 6.93) and subacute tinnitus (OR 2.47, 95% CI: 1.34, 4.53) in smokers, compared with non-smokers. This association is not entirely new, as the renowned otolaryngologist John W House (Los Angeles, California, US) pointed out more than 30 years ago: ’Patients who have tinnitus should stop smoking’.

Tobacco smoke contains numerous known ototoxic chemicals such as nicotine, hydrogen cyanide, carbon monoxide, cadmium, formaldehyde, benzene, arsenic, ammonia and lead, which damage the highly sensitive hair cells lining the cochlea and adversely affect the auditory cortex by impairing neurotransmitter emissions. For example, smokers also have lower amplitudes of evoked otoacoustic emission and reduced strength of the medial olivocochlear reflex than non-smokers. In addition, it has been determined that smoking can promote eustachian tube dysfunction.

General practitioners (GPs) can play a pivotal part in encouraging patients with tinnitus to avoid or quit smoking and improve the cumulative patients’ quality of life. A very good guide for health professionals about supporting smoking cessation can be found on The Royal Australian College of General Practitioners website (www.racgp.org.au/your-practice/guidelines/smoking-cessation). Although further research is urgently needed, a brief indication of the positive correlation between smoking and tinnitus should be included in a clinical review article for GPs.

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References

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