Who is the bad apple?

Re: Canadian medical students want more nutrition instruction, News Aug. 4.[1] This survey couldn't come at a better time. Despite what many developed countries are calling the 'obesity epidemic,' more and more physicians seem stumped when it comes to giving nutritional advice. Dr. Geneviève Moineau, associate dean of undergraduate medical education at the University of Ottawa, points out that many health issues can either be prevented or treated through proper nutrition. Why, then, is nutrition not integrated into each and every module of medical students' training?

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For the full letter, go to: www.cmaj.ca/cgi/eletters/181/3-4/133#169264

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Acetaminophen and asthma

I must congratulate Padmaja Subbarao and colleagues for their elegant review, Asthma: epidemiology, etiology and risk factors[1]. But they missed covering acetaminophen as an etiological cause of asthma in both adults and children. Several studies have shown a relationship between acetaminophen consumption and presence of asthma. For example, Cohet and colleagues[2] studied the association between infections in children aged 0 to 4 years and medications used (antibiotics and acetaminophen) and the subsequent presentation of asthma at age 6 to 7; the authors observed that the use of acetaminophen in the first year of life was weakly associated with wheezing, asthma, rhinitis, and eczema. In their prospective study, Barr and colleagues[3] found that acetaminophen use was associated with the presence of newly diagnosed asthma among women. Several mechanisms have been postulated that may explain the possible risk of asthma with acetaminophen use. The main mechanism involves glutathione, which is a potent antioxidant found in lung tissue. Acetaminophen has shown to lower serum glutathione levels in healthy volunteers.[4] A second possible mechanism is thought to be acetaminophen's lack of inhibition of the enzyme cyclooxygeanse, the key enzyme involved in the production of prostaglandins playing a major role in the inflammatory cascade in asthma.[5] Finally, an emerging hypothesis involves the possible antigenic effect of acetaminophen and subsequent rise in IgE and histamine levels with exposure to acetaminophen.[6]

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For the full letter, go to: www.cmaj.ca/cgi/eletters/cmaj.080612v1#201684

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The authors' response:

We would like to thank Dr. Singh for his comments and his in-depth review of the acetaminophen and asthma literature.[1] As in any review, it is difficult to address all possible risk factors and we attempted to link data that was causally linked in multiple studies with incident asthma. The meta-analysis by Etminan et al,[2] published earlier this year provides a comprehensive overview of the studies associating acetaminophen and asthma risk. As per the overview, the evidence is mounting that acetaminophen use is linked to increased rates of asthma but to date little evidence is available to suggest that acetaminophen is actually causing asthma. Of particular note is the absence of a consistent data between prenatal acetaminophen use and asthma development. As mentioned in the Etminam article, at this point there are many potential confounders such as viral infection which may in part explain this finding. Careful further study may help to elucidate the role of acetaminophen in the development of incident asthma.

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For the full letter, go to: www.cmaj.ca/cgi/eletters/cmaj.080612v1#207256

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H1N1-related SIRS?

Much of the journalism concerning H1N1 influenza virus continues the simplistic infectious disease model that it is a virulent virus which can be managed with handwashing and vaccination.[1] The infectious disease model suggests that lower standards of living and medical care explain these disparities. A more plausible explanation is available. The response to H1N1 may be one of two types: severe inflammatory response syndrome (SIRS) versus mild influenza. It has been known for years that distinct populations behave differently after organ transplantation. One of the mechanisms proposed for this difference is the