



## Invited Commentary

# Are organically grown foods safer and more healthful than conventionally grown foods?

(Submitted 16 July 2014 – Final revision received 21 July 2014 – Accepted 23 July 2014 – First published online 30 September 2014)

A major literature review and meta-analysis of studies comparing organically grown crops with conventionally grown crops has appeared recently in this journal, and it has concluded that organically grown crops do tend to have a notably lower Cd content than conventionally grown crops – on average, about 48% lower<sup>(1)</sup>. This estimate takes into account eighty-seven different comparisons previously reported in the literature, and hence there is little room for doubt that organically grown crops do tend to have lower Cd content.

The heavy metal Cd is emerging as a major cause of vascular disorders, various common cancers, kidney disease, osteoporosis and other health disorders, even in populations that do not have occupational exposure to this toxin<sup>(2–4)</sup>. Although tobacco smoke is a major source of Cd exposure, Cd is also found in the diet; green vegetables, root vegetables, tubers, grains, organ meats and shellfish often contain reasonable amounts of this toxin. Once Cd gets into the body, there is no physiological mechanism for excreting it; hence, the half-life of Cd in the body has been estimated to be 10–30 years<sup>(5)</sup>. The concentration of Cd in the urine (expressed as µg/mg of creatinine) is thought to represent a reasonably accurate measure of the total body burden of Cd. Although Fe deficiency increases the efficiency of dietary Cd absorption, metabolic determinants of disease risk are not known to influence Cd absorption; moreover, the foods that are the predominant dietary sources of Cd are not inherently harmful to health. Hence, at least in non-smokers, the many studies that have correlated increased urinary Cd concentrations with an increased risk of health disorders very likely imply that Cd does indeed play a causative role in these disorders, consistent with its adverse effects observed in rodent studies. Cd can induce oxidative stress throughout the body, and interference with some of the physiological roles of Zn (as in DNA repair) may also contribute to the pathogenic impact of Cd<sup>(6,7)</sup>.

As an example of the magnitude of the risk involved, the four case–control studies, to date, to assess the correlation of breast cancer risk with urinary Cd concentrations have concluded that Cd exposure may be responsible for 27–68% of the breast cancer cases – higher levels in Japan, where agricultural soils and rice tend to be rich in Cd<sup>(8–11)</sup>. With regard to vascular diseases, recent multivariate-adjusted analyses of the National Health and Nutrition Survey cohort have concluded that Cd exposure may be responsible for 28% of the myocardial infarction cases and 17% of the total CVD and cerebrovascular disease cases<sup>(12,13)</sup>. Analogously,

data from the prospective Strong Heart Study (focusing on Native Americans) suggest that Cd exposure may account for 16, 23 and 28% of the coronary disease, stroke and heart failure cases, respectively<sup>(14)</sup>. Case–control studies attempting to correlate dietary Cd intake with disease risks have usually yielded null results, which probably reflects the fact that the Cd content of a given food can vary widely depending on the soil of origin; hence, estimating dietary Cd intake with FFQ is of dubious merit and has served to create the impression that the epidemiology correlating Cd status with disease risk is equivocal. In fact, if one focuses on urinary Cd concentrations when surveying Cd epidemiology, the hazard of Cd stands out crystal-clear.

High supplemental intakes of Zn, and the antioxidant activity of spirulina, may have potential for attenuating the adverse health impacts of Cd that is already in the body<sup>(4)</sup>. Traditional chelation therapies fail to remove Cd, as most of it is located intracellularly, where chelating drugs cannot reach it. Supplementing meals with Zn and Mg may lessen the absorption of dietary Cd to some degree, and avoidance or correction of Fe deficiency is quite important in this regard<sup>(4,15)</sup>; the fact that women tend to have higher Cd concentrations than men probably indicates that they are more prone to Fe deficiency before menopause. Cd exposure can also be lessened by avoiding tobacco smoke – or by not smoking. However, avoiding dietary Cd poses a special challenge, as a high proportion of dietary Cd derives from plant foods ordinarily considered healthful – vegetables, tubers and grains. Indeed, a Slovak study has found that blood Cd concentrations tend to be higher in vegans than in omnivores<sup>(16)</sup>. Cd occurs naturally in most soils, and plants grown in these soils will incorporate this Cd to a greater or lesser extent; less Cd is taken up from alkaline soils than from acidic soils.

The new review by Baranski *et al.*<sup>(1)</sup> provides the first compelling evidence that organic farming techniques have a major favourable impact on crop Cd content; on average, the Cd content of organically grown foods is approximately half as high as that of the same foods grown conventionally. This probably reflects the fact that many phosphate fertilisers used in conventional agriculture are significantly contaminated with Cd. Studies have concluded that the more frequently such fertilisers are applied to soil, the higher the Cd content of the foods grown in that soil is<sup>(17–20)</sup>. It should be borne in mind, however, that organically grown crops do not inherently have a low Cd content; foods grown in soil



that is natively high in Cd will have a Cd content that reflects the soil's content. Also, the new study has found that organically grown crops do not have a lower content of Pb or As, two other mineral contaminants linked to health risks, when compared with conventionally grown crops.

This new review has also found that organically grown crops tend to have a higher content of many antioxidant phytochemicals, some of which are likely to be health protective<sup>(21–26)</sup>. Many of these phytochemicals function to protect plants from the pests that prey on them; hence, plants tend to synthesise less of these if artificial insecticides are used. Not surprisingly, organic foods tend to have lower levels of pesticide residues. The jury on whether or not ingesting trace levels of insecticides from foods is harmful to human health is still out, albeit there is growing evidence that environmental exposure to organophosphate pesticides can have adverse neurodevelopmental effects on children<sup>(27)</sup>. In any case, the lesser amounts of Cd and the higher levels of natural phytochemical antioxidants constitute rational grounds for choosing organically grown foods when these are available.

What would be the health consequences of switching to organically grown foods? If non-smokers were to consume only such foods throughout life, the review by Baranski *et al.*<sup>(1)</sup> enables us to predict that their body burdens of Cd would be approximately half as high as those of non-smokers who ate foods raised with conventional agriculture. To appreciate the impact of this, note that an analysis of the National Health and Nutrition Examination Survey cohort (1988–94) found that, after correction for numerous covariates, including those linked to smoking (smoking status, cumulative smoking dose and serum cotinine), subjects in the 80th percentile of urinary Cd, as opposed to those in the 20th percentile, had a hazard ratio of 1.52 (95% CI 1.00, 2.29) for total mortality<sup>(28)</sup>. With respect to cardiovascular mortality specifically, the corresponding hazard ratio was 1.74 (95% CI 1.07, 2.83). This study is not an outlier – a recently published study focusing on people in Cd-non-polluted areas in Japan has found that, once again after adjustment for pertinent covariates, men in the 4th quartile of urinary Cd had a hazard ratio of 1.50 (95% CI 1.11, 2.02) for total mortality; women had the corresponding hazard ratio of 1.50 (95% CI 1.08, 2.09)<sup>(29)</sup>. The large apparent effect of Cd on total mortality in these studies presumably reflects the fact that Cd has a pathogenic impact on a high proportion of potentially fatal disorders. Although these data do not enable us to calculate the degree to which a halving of Cd exposure would decrease total mortality, it would not be unreasonable to estimate a 20% reduction of total mortality.

Indirect evidence that this estimate may be in the right ballpark comes from the Age-Related Eye Disease Study 1 supplementation trial, a large prospective study designed to assess the impact of certain supplemental nutrients on the progression of age-related macular degeneration. Some of the participants in this study were randomised to receive 80 mg of Zn daily, with or without ancillary antioxidants. Zn antagonises the toxicity of Cd by inducing the Cd-binding protein metallothionein and also probably by competing with Cd for binding to intracellular proteins<sup>(4)</sup>. During a

follow-up period averaging 6.5 years, total mortality in the Zn-supplemented group, in comparison with participants not receiving Zn, was 27% lower (95% CI 0.61, 0.89)<sup>(30)</sup>. No efforts to replicate this remarkable finding, reported 10 years ago, have been made. Arguably, it could be a real effect reflecting, at least in part, the ability of Zn to offset the pathogenicity of the body Cd pool.

With respect to flavonoid intake, a recent meta-analysis (also in *British Journal of Nutrition*) has reported that a 10 mg/d increase in flavonoid intake is associated with a 5% reduction in cardiovascular risk (95% CI 0.91, 0.99)<sup>(21)</sup>. Baranski *et al.*<sup>(1)</sup> found that organic produce tend to have about 30% higher total flavonoid content than non-organic produce. In the Baltimore Longitudinal Study of Aging, daily intake of flavonoids was estimated to be 280 mg<sup>(31)</sup>. If we presume that most of these flavonoids came from conventionally grown produce, switching to organic produce while holding total produce intake constant might yield a daily intake of about 350 mg; in the previously cited study, it was found that a 70 mg increase in daily flavonoid intake would predict a 35% decrease in cardiovascular risk. This is doubtless a considerable overestimate, as the association between flavonoid intake and reduced cardiovascular risk is almost certainly attributable in part to other factors in flavonoid-rich foods (or their displacement of other less healthful foods). Nonetheless, in light of the undoubted biological activity of dietary flavonoids, it seems likely that a significant increase in flavonoid intake *per se* would have a worthwhile impact on cardiovascular health.

It should also be noted that there are ecological and ethical grounds for supporting organic farming, which may be beneficial for soil health, water quality and the health of farmworkers and their families.

## Acknowledgements

This work did not receive any funding support.

The authors have no conflicts of interest to report.

Mark F. McCarty<sup>1</sup>  
James J. DiNicolantonio<sup>2</sup>

<sup>1</sup>*Catalytic Longevity*  
7831 Rusb Rose Drive  
Apartment 316  
Carlsbad  
CA 92009  
USA

<sup>2</sup>*Mid America Heart Institute at St Luke's Hospital*  
Kansas City  
MO  
USA

email markfmccarty@gmail.com

doi:10.1017/S0007114514002748

## References

1. Baranski M, Srednicka-Tober D, Volakakis N, *et al.* (2014) Higher antioxidant and lower cadmium concentrations and lower incidence of pesticide residues in organically grown crops: a systematic literature review and meta-analyses. *Br J Nutr* **112**, 794–811.
2. Jarup L & Akesson A (2009) Current status of cadmium as an environmental health problem. *Toxicol Appl Pharmacol* **238**, 201–208.
3. Nawrot TS, Staessen JA, Roels HA, *et al.* (2010) Cadmium exposure in the population: from health risks to strategies of prevention. *Biomarkers* **23**, 769–782.
4. McCarty MF (2012) Zinc and multi-mineral supplementation should mitigate the pathogenic impact of cadmium exposure. *Med Hypotheses* **79**, 642–648.
5. Suwazono Y, Kido T, Nakagawa H, *et al.* (2009) Biological half-life of cadmium in the urine of inhabitants after cessation of cadmium exposure. *Biomarkers* **14**, 77–81.
6. Cuypers A, Plusquin M, Remans T, *et al.* (2010) Cadmium stress: an oxidative challenge. *Biomarkers* **23**, 927–940.
7. Hartwig A (2013) Cadmium and cancer. *Met Ions Life Sci* **11**, 491–507.
8. McElroy JA, Shafer MM, Trentham-Dietz A, *et al.* (2006) Cadmium exposure and breast cancer risk. *J Natl Cancer Inst* **98**, 869–873.
9. Gallagher CM, Chen JJ & Kovach JS (2010) Environmental cadmium and breast cancer risk. *Aging (Albany NY)* **2**, 804–814.
10. Nagata C, Nagao Y, Nakamura K, *et al.* (2013) Cadmium exposure and the risk of breast cancer in Japanese women. *Breast Cancer Res Treat* **138**, 235–239.
11. Strumylaite L, Kregzdyte R, Bogusevicius A, *et al.* (2014) Association between cadmium and breast cancer risk according to estrogen receptor and human epidermal growth factor receptor 2: epidemiological evidence. *Breast Cancer Res Treat* **145**, 225–232.
12. Everett CJ & Frithsen IL (2008) Association of urinary cadmium and myocardial infarction. *Environ Res* **106**, 284–286.
13. Agarwal S, Zaman T, Tuzcu EM, *et al.* (2011) Heavy metals and cardiovascular disease: results from the National Health and Nutrition Examination Survey (NHANES) 1999–2006. *Angiology* **62**, 422–429.
14. Tellez-Plaza M, Guallar E, Howard BV, *et al.* (2013) Cadmium exposure and incident cardiovascular disease. *Epidemiology* **24**, 421–429.
15. Gallagher CM, Chen JJ & Kovach JS (2011) The relationship between body iron stores and blood and urine cadmium concentrations in US never-smoking, non-pregnant women aged 20–49 years. *Environ Res* **111**, 702–707.
16. Krajcovicova-Kudladkova M, Ursinyova M, Masanova V, *et al.* (2006) Cadmium blood concentrations in relation to nutrition. *Cent Eur J Public Health* **14**, 126–129.
17. Taylor MD (1997) Accumulation of cadmium derived from fertilisers in New Zealand soils. *Sci Total Environ* **208**, 123–126.
18. Chen W, Chang AC & Wu L (2007) Assessing long-term environmental risks of trace elements in phosphate fertilizers. *Ecotoxicol Environ Saf* **67**, 48–58.
19. Al-Faiyz YS, El-Garawany MM, Assubaie FN, *et al.* (2007) Impact of phosphate fertilizer on cadmium accumulation in soil and vegetable crops. *Bull Environ Contam Toxicol* **78**, 358–362.
20. Cheraghi M, Lorestani B & Merrikhpour H (2012) Investigation of the effects of phosphate fertilizer application on the heavy metal content in agricultural soils with different cultivation patterns. *Biol Trace Elem Res* **145**, 87–92.
21. Wang X, Ouyang YY, Liu J, *et al.* (2014) Flavonoid intake and risk of CVD: a systematic review and meta-analysis of prospective cohort studies. *Br J Nutr* **111**, 1–11.
22. Woo HD & Kim J (2013) Dietary flavonoid intake and smoking-related cancer risk: a meta-analysis. *PLOS ONE* **8**, e75604.
23. Zamora-Ros R, Forouhi NG, Sharp SJ, *et al.* (2014) Dietary intakes of individual flavanols and flavonols are inversely associated with incident type 2 diabetes in European populations. *J Nutr* **144**, 335–343.
24. Macready AL, George TW, Chong MF, *et al.* (2014) Flavonoid-rich fruit and vegetables improve microvascular reactivity and inflammatory status in men at risk of cardiovascular disease – FLAVURS: a randomized controlled trial. *Am J Clin Nutr* **99**, 479–489.
25. Tresserra-Rimbau A, Rimm EB, Medina-Remon A, *et al.* (2014) Polyphenol intake and mortality risk: a re-analysis of the PREDIMED trial. *BMC Med* **12**, 77.
26. McCarty MF (2004) Proposal for a dietary “phytochemical index”. *Med Hypotheses* **63**, 813–817.
27. Munoz-Quezada MT, Lucero BA, Barr DB, *et al.* (2013) Neurodevelopmental effects in children associated with exposure to organophosphate pesticides: a systematic review. *Neurotoxicology* **39**, 158–168.
28. Tellez-Plaza M, Navas-Acien A, Menke A, *et al.* (2012) Cadmium exposure and all-cause and cardiovascular mortality in the U.S. general population. *Environ Health Perspect* **120**, 1017–1022.
29. Suwazono Y, Nogawa K, Morikawa Y, *et al.* (2014) Impact of urinary cadmium on mortality in the Japanese general population in cadmium non-polluted areas. *Int J Hyg Environ Health* (Epublication ahead of print version 12 May 2014).
30. Clemons TE, Kurinij N & Sperduto RD (2004) Associations of mortality with ocular disorders and an intervention of high-dose antioxidants and zinc in the Age-Related Eye Disease Study: AREDS report no. 13. *Arch Ophthalmol* **122**, 716–726.
31. Maras JE, Talegawkar SA, Qiao N, *et al.* (2011) Flavonoid intakes in the Baltimore Longitudinal Study of Aging. *J Food Compos Anal* **24**, 1103–1109.