Carrageenan: A Safe Additive

As the general secretary of Marinalg International, an association representing the worldwide producers of carrageenan, I would like to comment on Tobacman’s review article in EHP(1).

Authorities worldwide have extensively reviewed carrageenan safety. Contrary to Tobacman’s conclusion (J), all of these authorities agree that carrageenan may be used safely in food. As recently as June 2001, the Joint FAO/WHO Expert Committee on Food Additives (JECFA; an independent international panel of expert scientists and government authorities) concluded a multiple year review of all of the safety data on carrageenan (2). This included a specific analysis of the potential for promotion of colon cancer by carrageenan. The JECFA affirmed their earlier conclusion on the safety of carrageenan (e.g., (3,4))—that it may be used safely in the diet at amounts only limited by the amount necessary to achieve its technical function. Overall, the carrageenan sold as a food, drug, and cosmetic additive has been tested extensively, and regulatory authorities worldwide have uniformly found carrageenan to be essentially nontoxic and agreed that it may be used safely in food.

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REFERENCES AND NOTES
1. Tobacman JK. Review of harmful gastrointestinal effects of carrageenan in animal experiments. Environ Health Perspect 110:A176 (2002).
2. Compendium of Food Additives Specifications. Addendum 9. Joint FAO/WHO Expert Committee on Food Additives, 57th Session, Rome, Italy, 5–14 June 2001. FAO Food and Nutrition Paper 52, add. 9. Rome:Food and Agriculture Organization, 2001.
3. JECFA, Evaluation of Certain Food Additives: Fifty-first report of the Joint FAO/WHO Expert Committee on Food Additives. WHO Technical Report series, No. 891. Geneva:World Health Organization, 2000.

Carrageenan: Response

It is difficult to recognize a wolf in sheep’s clothing. This seems to be the situation with regard to carrageenan.

In response to a letter to EHP from Phil Carthew, I commented on some of the data used for the recent Joint FAO/WHO Expert Committee on Food Additives (JECFA) review to which Kirsch refers (J,2). I found the JECFA conclusions disconcerting in view of the available evidence. Previously, the JECFA considered modification of their recommendation about carrageenan to include a minimum average molecular weight (3,4).

Extensive experimental data have demonstrated that a) degraded carrageenan produces neoplasms and ulcerations in animal models; b) acid hydrolysis, such as occurs in the stomach, leads to the production of degraded carrageenan from food-grade carrageenan; and c) food-grade carrageenan contains significant amounts of degraded carrageenan. Human consumption of carrageenan has been increasing steadily in the United States in the 20th century (5–8).

The data with regard to intestinal effects of carrageenan seem sufficient to mandate restriction of carrageenan intake. I remain hopeful that the Food and Drug Administration and the JECFA will revise their recommendations pertaining to the safe use of carrageenan, that industry will substitute other gums for carrageenan, that red seaweed farmers will diversify, and that consumers will select food products without carrageenan.

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REFERENCES AND NOTES
1. Carthew P. Safety of carrageenan in foods (Letter). Environ Health Perspect 110:A176 (2002).
2. Tobacman, JK. Carrageenan in foods: response (Letter). Environ Health Perspect 110:A176 (2002).
3. Greig JB. Carrageenan. WHO Safety Evaluation of Certain Food Additives. Food Additives Series 42. Geneva:World Health Organization, 1999. Available: http://www.inchem.org/documents/jecfa/jecmono/v042je08.htm [cited 9 April 2002].
4. European Commission. Draft Commission Directive. III/543/96 EN. Brussels:European Commission, 1996.
5. Tobacman JK. Review of harmful gastrointestinal effects of carrageenan in animal experiments. Environ Health Perspect 109:983–994 (2001).
6. Yu G, Suan H, Iaconoisi AS, Sikkander SA, Thanawiroon C, Tobacman JK, Toida T, Linhardt RJ. Structural studies on κ-carrageenan derived oligosaccharides. Carbohydr Res 337:433–440 (2002).
7. Manns MJ. The stability of carrageenans to processing. In: Gums and Stabilisers for the Food Industry 9 (Williams PA, Phillips GO, eds). Cambridge, UK:The Royal Society of Chemistry, 1998:345–357.
8. Capron I, Yuen M, Muller G. In-vivo gastric stability of carrageenan. Food Hydrocoll 10:239–244 (1996).

Public Fear of Dioxins from Modern Municipal Waste Incinerators Is Not Justified

In spite of the important increase of the three “r’s” (reduction, reuse, and recycling) in the management of municipal solid waste (MSW), there are still many places in which incineration continues to be an important option for the disposal and treatment of MSW. Although incineration recovers energy and reduces the volume of waste that requires landfilling, because it also involves the emission of a number of pollutants where incinerators are used or proposed, people are often afraid that resulting pollutants will adversely affect their health. Concern has been especially notable with respect to dioxins and furans. These organic pollutants are toxic in extremely tiny amounts and bioaccumulate in humans (1–5). Moreover, in February 1997 using recent epidemiologic data on exposed human populations and experimental carcinogenicity bioassays in laboratory animals, the International Agency for Research on Cancer (IARC) classified 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) as carcinogenic to humans (IARC group 1) (6). Recent episodes such as the Belgian PCB and dioxin incident of 1999 (7,8) have contributed to increase the concern with respect to dioxins.

As a direct consequence of a notable growth in the public opinion against the binomial MSW incineration and dioxins, numerous municipal, regional, and national governments have placed a moratorium on construction of new MSW incinerators (MSWIs) and more stringent controls on existing units. The argument against incineration of MSW is mainly based on the following premises: MSWIs emit dioxins, and dioxins are carcinogenic; therefore, MSWIs are carcinogenic facilities. However, in recent years, dioxin emissions from MSWIs have been reduced to levels < 0.1 ng I-TEQ/Nm³. As a consequence of this, the current national dioxin inventories show that in those countries with a stringent regulation of dioxin emissions from MSWIs (e.g., the member states of the European Union) that started legislation in the early 1990s, MSW incineration is currently a minor contributor to any national inventory (9,10).

Taking the above information into account, the main purpose of this letter is to try to abate the fear of dioxins in relation to MSWIs, especially for the population living near these facilities. To reduce total dioxin exposure, a simple change in the dietary habits can be as relevant as the decrease in direct exposure to environmental dioxins in the vicinity of a MSWI. The data shown in Tables 1 and 2 are based on studies from two MSWIs in Catalonia, Spain, in which technical improvements were recently carried out and dioxin emissions were reduced to concentrations < 0.1 ng I-TEQ/Nm³ (11,12). To assess human health risks before and after these improvements, the following routes of dioxin exposure were evaluated: direct contact from inhalation of
Table 1. Comparison of the reduction in direct exposure to dioxins with a single change of dietary habits: consumption of 300 g/day semi-skimmed milk instead of whole milk.

|                | Whole milk | Semi-skimmed milk | Reduction |
|----------------|------------|-------------------|-----------|
| Dioxin concentrations in milk (pg I-TEQ/g wet weight) | 0.011 | 0.006 | 0.005 |
| Dioxin intake through milk consumption (pg I-TEQ/kg bw/day) | 0.047 | 0.026 | 0.021 |

Table 2. Dioxin exposure for adult populations living at 500 and 1,000 m from two municipal waste incinerators (MSWI-1 and MSWI-2) before and after pronounced reductions in the emissions of dioxins from the facilities.

|                | MSWI-1 Before | MSWI-1 After | MSWI-2 Before | MSWI-2 After |
|----------------|--------------|--------------|--------------|--------------|
| Total direct exposure (pg I-TEQ/kg/day) x 10^{-2} | 5.102 | 1.271 | 4.087 | 0.995 |
| Total exposure to dioxins (pg I-TEQ/kg/day) | 2.82 | 0.92 | 2.81 | 0.91 |

Air and particles; ingestion and dermal contact with soil and dust; and indirect exposure (dietary intake). For risk estimations, I assumed the worst scenario (e.g., all dioxins in the neighborhood of the respective MSWI would be emitted by the facility).

For adults living at 500 m from MSWI-1 (Montcada), the direct exposure to dioxins before technical improvements was 5.102 × 10^{-2} pg I-TEQ/kg/day, and the total dioxin exposure was 2.82 pg I-TEQ/kg/day. This resulted in a contribution to dioxins from the MSWI of 1.81%. Two years after technical improvements were carried out, direct exposure to dioxins decreased to 1.271 × 10^{-2} pg I-TEQ/kg/day, while the total dioxin exposure diminished to 0.92 pg I-TEQ/kg/day, which results in a contribution to dioxins from the MSWI of 1.36%. This indicates that the important percentage reductions (75.1%) in the direct exposure to dioxins is practically imperceptible when compared with the contribution of indirect exposure (dietary intake) to total dioxin exposure, which decreased from 2.82 (13) to 0.92 pg I-TEQ/kg/day (12) during the same period.

Although people are concerned about exposure through MSWI emissions, diet is the main route of dioxin exposure in humans. With respect to this, the absolute reduction of 0.038 pg I-TEQ/kg/day in the dioxin levels at 500 m from the MSWI-1 2 years after introducing the technical improvements is of the same order of magnitude as a simple change in dietary habits: daily consumption of 300 g of semi-skimmed milk instead of the same quantity of whole milk would result in a reduction in dioxin exposure of 0.021 pg I-TEQ/kg/day (dioxin concentrations in semi-skimmed and whole milk of 0.006 and 0.011 pg I-TEQ/g wet weight, respectively; Table 1). Similar results would be also obtained for those living at 1,000 m from the facility (total reduction from direct exposure of 0.031 pg I-TEQ/kg/day). For adult subjects living at 500 and 1,000 m from MSWI-2 (Tarragona), the decreases in direct exposure to dioxins were 0.016 and 0.013 pg I-TEQ/kg/day, respectively (Table 2), values that are also similar in magnitude to the reduction of 0.021 pg I-TEQ/kg/day derived from a hypothetical consumption of semi-skimmed milk instead of whole milk (Table 1).

For a number of different reasons, strong arguments in favor or against incineration as a way of disposal and treatment of MSW can be justified. However, it seems quite evident that the public concern over the health risks due to exposure to dioxins emitted by modern MSWIs or MSWIs equipped with modern technologies is not scientifically justified. Therefore, decisions regarding the construction or closing of modern MSWIs should not be primarily based on public opinion and fears of dioxin emissions from these plants. Because the diet is the main route of human exposure to dioxins, only efforts to reduce emissions from all sources can significantly contribute to decrease environmental dioxin concentrations, and consequently, their levels in food.

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REFERENCES AND NOTES

1. Bertazzi PA, Consonni D, Bachetti S, Ruhagatti M, Baccarelli A, Zacchetti C, Pesatori AC. Health effects of dioxin exposure: a 20-year mortality study. Am J Epidemiol 152:1031–1044 (2001).
2. Birnbaum LS, Cummings AM. Dioxins and endometriosis: a plausible hypothesis. Environ Health Perspect 110:15–21 (2002).
3. Geusau A, Abraham K, Geissler K, Sator MO, Stingl G, Tscherchler L, Severe, 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) intoxication: clinical and laboratory effects. Environ Health Perspect 109:885–896 (2001).
4. Kimbrough RD, Krouse SK. Polychlorinated biphenyls, dibenzo-p-dioxins, and dibenzofurans and birth weight and immune and thyroid function in children. Regul Toxicol Pharmacol 34:42–52 (2001).
5. Kopiaginas M. Human health effects of dioxin: cancer, reproductive and endocrine system effects. Hum Reprod Update 7:331–339 (2001).
6. McGregor DB, Partensky C, Wilburn R, Rice JM. An IARC evaluation of polychlorinated dibenzo-p-dioxins and polychlorinated dibenzofurans as risk factors in human carcinogenesis. Environ Health Perspect 106(suppl 2):755–760 (1998).
7. Schepens PJ, Covaci A, Jorens PG, Hens L, Scherpe S, van Larebeke N. Surprising findings following a Belgian food contamination with polychlorophenyls and dioxins. Environ Health Perspect 109:101–103 (2001).
8. van Larebeke N, Hens L, Schepens P, Covaci A, Baeyens J, Everaert K, Bernheim JL, Vlietinck R, De Poorter G. The Belgian PCB and dioxin incident of January–June 1999: exposure data and potential impact on health. Environ Health Perspect 109:285–273 (2001).
9. Anderson DR, Fisher R. Sources of dioxins in the United Kingdom: the steel industry and other sources. Chemosphere 46:371–381 (2002).
10. UNEP Chemicals. Dioxin and Furan Inventories: National and Regional Emissions of PCDD/PCDF. Geneva:United Nations Environment Programme, 1999. Available: http://irtcp.unep.ch/paps/pdf/dioxinfuran/difurpt.pdf (cited 10 April 2003).
11. Domingo JL, Schuhmacher M, Llobet JM, Mühler L, Rivera J. PCDD/F concentrations in soil and vegetation in the vicinity of a municipal waste incinerator after a pronounced decrease in the emissions of PCDD/Fs from the facility. Chemosphere 43:217–226 (2001).
12. Domingo JL. Unpublished data.
13. Domingo JL, Schuhmacher M, Granero S, Llobet JM. PCDDs and PCDFs in food samples from Catalonia, Spain: an assessment of dietary intake. Chemosphere 38:3517–3528 (1999).

Corrections and Clarifications
In the article “High-Resolution Revolution” [EHP 110: A238–239], the units of measurement in the article listed as millimeters should be micrometers. Also, in the photo caption on p. A239, the scan image on the computer, which is identified as rat tissue, is from a mouse. EHP regrets the errors.