Increasing Homicide Rates and Linoleic Acid Consumption Among Five Western Countries, 1961–2000

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ABSTRACT: Clinical intervention trials and animal studies indicate that increasing dietary intakes of long chain n-3 FA or reducing linoleic acid intake may reduce aggressive and violent behaviors. Here we examine if economic measures of greater n-6 consumption across time and countries correlate with greater risk of homicide. Linoleic acid available for human consumption was calculated from World Health Organization disappearance data for 12 major seed oils in the food supply for the years 1961 to 2000 in Argentina, Australia, Canada, the United Kingdom, and the United States (US). Homicide mortality rates, adjusted for age, were obtained from the central judicial authority of each country. Apparent linoleic acid intake from seed oil sources ranged from 0.29 en% (percentage of daily food energy) (Australia 1962) to 8.3 en% (US 1990s). Greater apparent consumption of linoleic acid correlated with higher rates of homicide mortality over a 20-fold range (0.51–10.2/100,000) across countries and time in an exponential growth regression model ($r = 0.94$, $F = 567$, $P < 0.00001$). Within each country, correlations between greater linoleic acid disappearance and homicide mortality over time were significant in linear regression models. Randomized controlled trials are needed to determine if reducing high intakes of linoleic acid by seed oils with alternative compositions can reduce the risk of violent behaviors. These dietary interventions merit exploration as relatively cost-effective measures for reducing the pandemic of violence in Western societies, just as dietary interventions are reducing cardiovascular mortality. Low linoleate diets may prevent behavioral maladies that correctional institutions, social service programs, and mental health providers intend to treat.

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For 2–3 million years of human evolution, diets rich in seafood and range-fed animals likely provided appreciable amounts of the n-3 essential FA eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) (1,2). As both n-3 and n-6 FA are essential, diets are dominant determinants of the proportions of essential FA in brain and body tissues. DHA is selectively concentrated in synaptic neuronal membranes and is necessary for optimal neuronal function (3,4). Seed oils, including soy, corn, and canola, are rich in the n-6 FA linoleic acid, and these oils have displaced other dietary fats and calories in the food supply in developed countries during the 20th century. In the United States in 1909, soy oil accounted for approximately 0.02 en% (percentage of daily food energy) of all calories available for food consumption compared to 20 en% in 2000 (5). High dietary linoleate can depress tissue and membrane levels of the n-3 FA EPA and DHA by competing with α-linolenic for conversion to 20- and 22-carbon highly unsaturated FA (HUFA) (6). We previously proposed that the increase in linoleic acid in Western diets, especially relative to EPA and DHA intake, has been a risk factor contributing to the increasing rates of major depression and violence in the 20th century (7). The observation that greater intakes of seafood rich in EPA and DHA correlated with lower rates of homicide mortality across 36 countries ($r = -0.63$, $P < 0.00001$) (8) is consistent with observational studies reporting that lower tissue levels of EPA and/or DHA predict greater hostility measures among 4,000 subjects in the CARDIA epidemiological study (9), among aggressive cocaine addicts (10), and violent prisoners (11). Homicide mortality data are robust indicators of violence, have clear diagnostic criteria with unequivocal importance, and have been prospectively collected. We postulated that greater consumption of seed oils, the major dietary source of linoleic acid, would correlate with greater rates of homicide mortality across time and among Western countries with similar socioeconomic and background seafood intake levels. The positive association reported here is striking.

METHODS

Data on age-adjusted homicide mortality were obtained from the U.S. Department of Justice (12), the United Kingdom Office of National Statistics on Criminal Justice (13), Statistics Canada (14), the Argentina Ministry of Justice (15), and the Australian Bureau of Statistics (16). Seed oils, in particular soybean oil, are the dominant sources of the linoleic acid in the United States. Economic data on the disappearance of 12 primary seed oils as a percentage of available food energy for all commodities from 1961 to 2000 were obtained from the World Health Organization (WHO) Statistical Information Services (17). Disappearance (apparently used for human consumption) was defined as production plus imports plus existing stocks minus exports and remaining stocks (5). The nutrient content of each seed oil was calculated using version 13 of the National Nutrient Database of the United States Department of Agriculture (5). For each year, linoleate availability from each food oil

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Abbreviations: DHA, docosahexaenoic acid; en%, percentage of daily food energy; EPA, eicosapentaenoic acid; WHO, World Health Organization.

was summed and expressed as a percentage of total calories (en%) from all food available for human consumption. No seed oil disappearance or mortality data available for these countries were excluded and no adjustments were made for possible food wastage. Both the food commodity and the homicide mortality data were prospectively collected, and they reflect changes for the whole population of each country. Statistical analyses included both simple linear Pearson's regressions using Statview 5.0 (SAS Institute, Cary, NC) and iterative curve fitting for linear and nonlinear regressions using Sigma Plot 8.0 (SPSS Inc., Chicago, IL).

RESULTS

Linoleic acid from seed oil sources, in apparent consumption as food, ranged from 0.29 en% (Australia 1962) to 8.3 en% (US 1990s). Rates of homicide mortality nearly tripled in the United States and in the United Kingdom from 1961 to 2000. Time trends were first considered separately by country. Greater linoleate disappearance was correlated with greater homicide mortality over time in linear regression models for the United States ($r = 0.61$, $P < 1.0 \times 10^{-6}$), the United Kingdom ($r = 0.89$, $P < 1.0 \times 10^{-14}$), Australia ($r = 0.74$, $P < 1.0 \times 10^{-10}$), Canada ($r = 0.53$, $P < 0.0004$), and Argentina ($r = 0.75$, $P < 0.0001$). When data from all countries and time points were combined,

greater apparent consumption of linoleic acid correlated with higher rates of homicide mortality over a 20-fold range (0.51–10.2/100,000) in an exponential growth regression model ($r = 0.94$, $F = 567$, $P < 0.00001$) (see Fig. 1). A significant correlational relationship remains ($r = 0.51$, $F = 20.5$, $P < 0.00001$) in a similar three-factor exponential growth model after exclusion of the United States.

DISCUSSION

This comparison indicates a striking correlation between greater apparent consumption of linoleic acid from seed oils and greater risk of homicide mortality across time, from 1961 to 2000, among five Western countries (see Figs. 2–6). These findings suggest, but do not demonstrate, that increased availability of linoleic acid might be a causal factor contributing to the increased rates of homicide during the last half of the 20th century among these five countries. To our knowledge, this is the first examination of a risk factor for homicide across both countries and time. Greater linoleic acid consumption is not proposed here as a sole determinant factor for greater homicide mortality, but as a readily modifiable potential risk factor.

These findings may be due to secondary hidden covariates, which are fairly well established factors that increase the risk of homicide. For example, the availability of firearms is a rea-
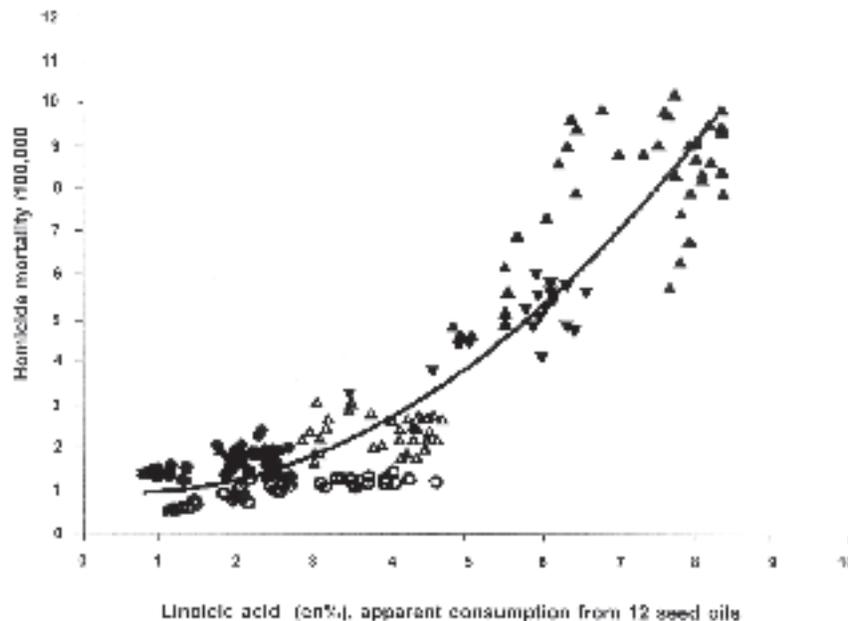

FIG. 1. Homicide rates and linoleic acid disappearance for human consumption among five countries, 1961–2000. The following symbols indicate dates within each country for the years 1961–2000: ⊥ Australia; ⊥ United Kingdom; **s**, Canada; t, Argentina; s, United States. The disappearance for human consumption of linoleic acid from 12 seed oils [18:2n-6 (en%)/cap/d] was derived as a percentage of energy available from all commodities available for use as human food, from the Food and Agriculture Organization/WHO database as indicated for each year. Other dietary sources of linoleic acid are not included. Corresponding homicide rates were adjusted for age, expressed per 100,000 population, and obtained from criminal justice statistics for each country. Iterative curve fitting for three-factor exponential growth equations resulted in the following fit: $f = 1.02788 - 0.162742x + 0.145575x^2$, $r = 0.94$, $r^2 = 0.87$, $F = 567$, $P < 0.00001$.
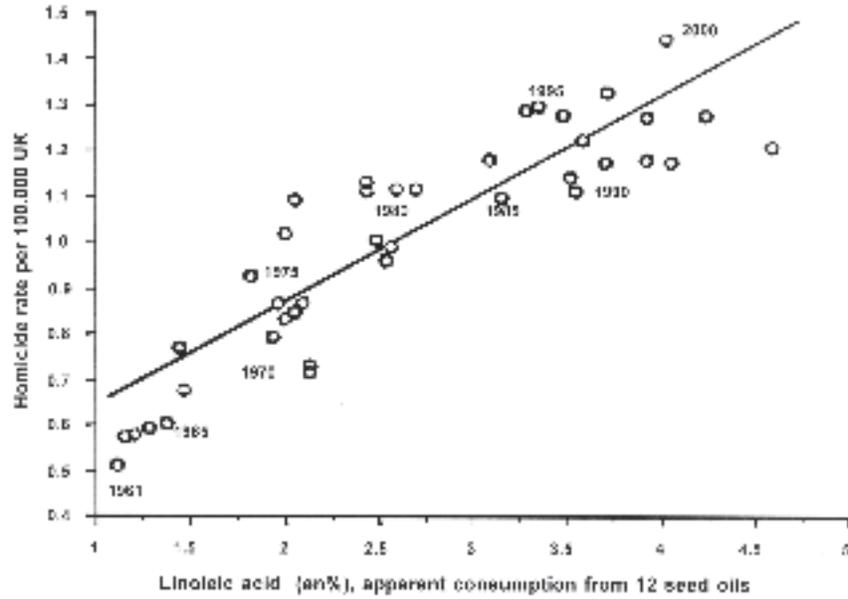
FIG. 2. Homicide mortality and consumption of linoleic acid from seed oils, 1961–2000, in the United Kingdom. The **o** symbol indicates dates 1961–2000 within the United Kingdom. A linear regression model resulted in the following fit: $r = 0.89$, $r^2 = 0.80$, $F = 150.6$, $P < 1 \times 10^{-14}$.

sonable confounding variable that could account for the findings reported here. Unfortunately, data on availability measured by per capita ownership or the percentage of households with firearms is not readily obtainable or reliable across countries in the time periods examined. Alternatively, data are more uniformly available expressing homicides attributable to firearms as a percentage of total homicides. This parameter also

has the advantage of being a more direct assessment of the use of the firearms in the commission of homicides than surveys of availability or percent household ownership (18). In Canada, both firearm availability and the percentage of homicides related to firearms decreased during this time as the result of progressively restrictive gun legislation in 1977, 1991, and 1995 (19). In Australia, the percentage of homicides related to
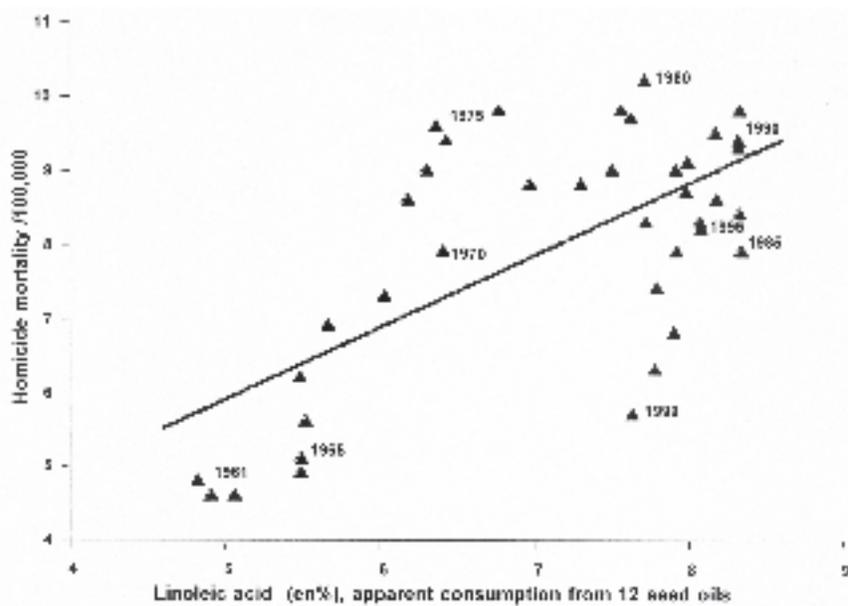

FIG. 3. Homicide mortality and consumption of linoleic acid from seed oils, 1961–1999, in the United States. The s symbol indicates dates 1961–1999 within the United States. A linear regression model resulted in the following fit: $r = 0.65$, $r^2 = 0.43$, $F = 27.7$, $P < 0.000001$.
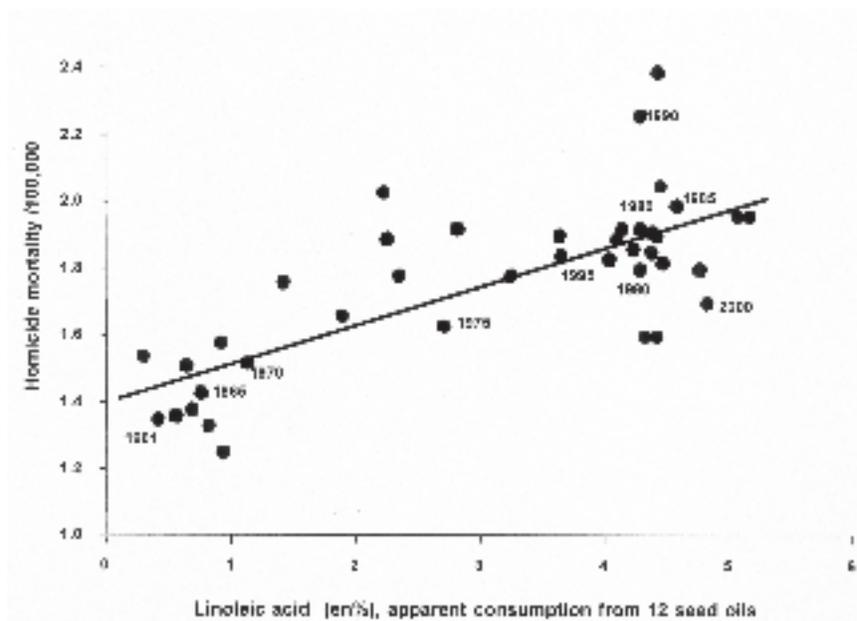
FIG. 4. Homicide mortality and consumption of linoleic acid from seed oils, 1961–2000, in Australia. The ⊥symbol indicates dates 1961–1999 within Australia. A linear regression model resulted in the following fit: $r = 0.74$, $r^2 = 0.54$, $F = 44$, $P < 1 \times 10^{-10}$.

firearms significantly decreased from 1960 to 2000 (20). Homicide rates by all methods rose in these countries, but, in general, for the period between 1960 and 2000 the percentage of homicides attributable to firearms decreased over time in Canada or Australia and changed little in the United Kingdom (21) or the United States (22). Since the availability of firearms as measured by their use in homicides has a negative or neutral

trend in this analysis, availability is unlikely to be a hidden causal factor underlying the positive relationships between greater linoleate intake and greater homicide mortality in these countries over time. Since the United States has a higher rate of firearm availability and a higher percentage of firearm-related homicides, it may be a driving force confounding the cross-national relationship. However, a significant correlational
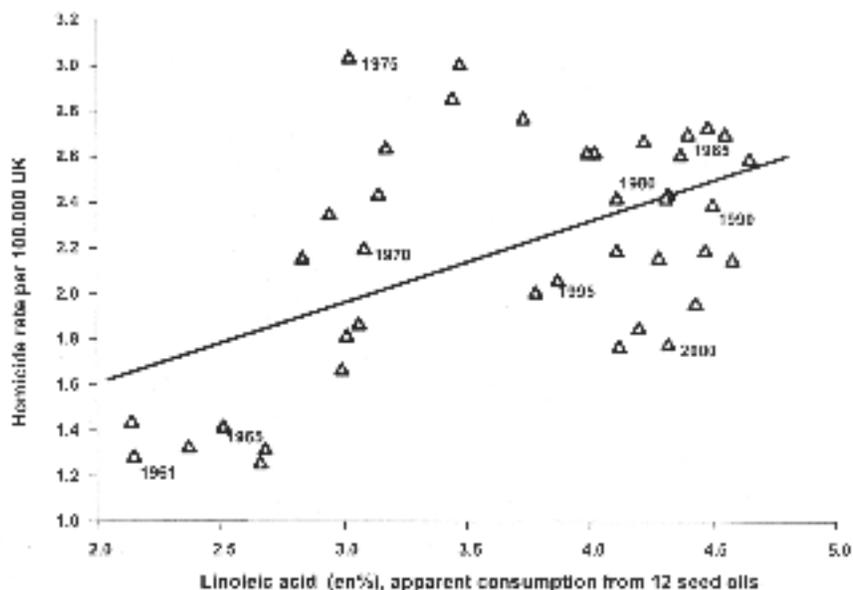

FIG. 5. Homicide mortality and consumption of linoleic acid from seed oils, 1961–2000, in Canada. The Δ symbol indicates dates 1961–2000 within Canada. A linear regression model resulted in the following fit: $r = 0.53$, $r^2 = 0.28$, $F = 15.1$, $P < 0.0004$.
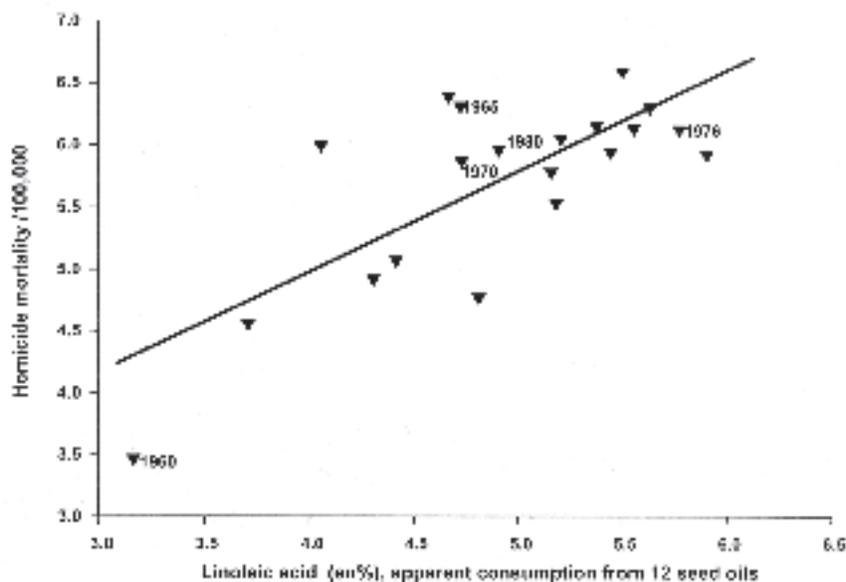
FIG. 6. Homicide mortality and consumption of linoleic acid from seed oils, 1960–1981, in Argentina. The t symbol indicates dates 1961–1985 within Argentina. A linear regression model resulted in the following fit: $r = 0.75$, $r^2 = 0.56$, $F = 23.2$, $P < 0.0001$.

relationship remained after excluding the United States. The magnitude of the relationships between homicide rates and linoleate disappearance ($r = 0.93$ including and $r = 0.51$ excluding the United States) reported here is similar to, or greater than, the magnitude of the relationship between firearm availability and homicide mortality in a cross-national analysis of 26 countries ($r = 0.69$ including and $r = 0.25$ excluding the United States) (23). In summary, it is unlikely that the correlational relationships between linoleate availability and homicide mortality are caused by differences in the availability or use of firearms when examined across countries and across time.

We do not propose that increased linoleate consumption during 1961 to 2000 can be isolated from many other societal, cultural, economic, or nutritional factors that might contribute to increased risk of homicide. However, the epidemiological associations in this report can help support a rationale for designing future controlled clinical intervention studies that directly alter linoleate intake and test for changes in aggressive behavior. The large differences in homicide mortality rates among the five countries suggest that many societal and economic factors contribute to greater risk of homicide (24). The American Academy of Pediatrics and five other prominent medical societies (25) cited exposure of youth to violent media *via* videogames, cable television, and the Internet as a contributing factor to societal violence. In the 40 years spanning the data examined here this media exposure has increased. These years have included the perception that the integrity of social systems declined in the United States and other countries, accompanying progressively higher divorce rates, a possible marker of disintegration of the family unit. In addition, drug and alcohol use have become widespread, along with gang behaviors, teenage pregnancy, sexually transmitted dis-

eases, and an increasing population of generally dysphoric, discontented youth, each of which is a potential risk to greater social disintegration. Many of these social and cultural factors are difficult to quantify, and comparable data sets are not available across time and countries. As direct data are not available on a yearly basis for all potential risk factors, we do not attempt here to create a statistical model comparing the relative contributions of each potential risk factor, and thus we do not conclude that these epidemiological associations constitute causal proof that increasing linoleate availability increases homicide risk.

There was wide diversity in the apparent consumption of linoleic acid from seed oils as a percentage of energy, ranging from 0.29 en% in Australia, in 1962, to 8.3 en% in the United States, 1985–1992. The disappearance data for comparable seed oil and other commodities were used to examine the relative differences in apparent linoleate intakes across time and countries. Clearly, seed oils are the major but not the sole source of linoleic acid in the diet, and no attempt was made here to determine the absolute per capita ingestion of total linoleic acid and total calories. Estimates of linoleic acid that included other food sources during comparable time periods were higher in the United Kingdom (26) and lower in the Australian diet (27) than those reported here. Differential degrees of hydrogenation, waste, spoilage, reports of total calorie disappearance, and oil composition not reflected by the nutritional composition tables were likely to have increased variability in estimates of linoleic acid intake as a percentage of energy and reduced the strength of the robust correlations reported here.

The ecological associations between greater risks of homicide mortality and both greater linoleate intake and lower seafood intake (28) are consistent with animal studies and controlled intervention trials in humans that reported decreased

measures of aggression or violence by increasing intakes of long chain n-3 FA relative to n-6 intake. This proposition that dietary differences in essential FA status can alter aggressive behavior has been supported by six placebo-controlled, randomized clinical trials where supplementation with EPA, DHA, or both reduced violent behaviors or measures of aggression (29–33). A trial using essential FA, multivitamins, and minerals also reported a substantial reduction in aggression among prisoners (34). Aggressive behavior was also markedly higher among the adult pups of mothers who were fed diets containing 43 en% from soy oil throughout gestation (35). Muricidal behavior was induced in adult rats by diets rich in linoleic acid (36), and DHA was lower in the brains of rats with isolation-induced muricidal behavior (37).

One mechanism that may link excessive linoleic acid intake or deficient EPA and DHA status is a deficit in serotonergic neurotransmission in the frontal cortex, which has been repeatedly implicated in the pathophysiology of lifelong impulsive and violent behaviors (38,39). Higley (39) reported that at 2 weeks of age, low concentrations of a marker of frontal serotonergic function, cerebrospinal fluid 5-hydroxyindolacetic acid, predicted a lifetime predisposition toward aggressive behavior among rhesus monkeys. Dietary deficiencies of n-3 intake during fetal life and early in development resulted in residual deficits in serotonergic neurotransmission (40). This four fold lower serotonin release stimulated by fenfluramine, measured at 60 days old, could be prevented by restoring dietary α-linolenic acid, but only if done before 14 days of life (40). When piglets' infant formulas were supplemented with arachidonic acid and DHA, frontal cortex concentrations of serotonin, dopamine, and their respective metabolites increased by nearly 50% (41). Reduction in serotonergic neurotransmission in the medial prefrontal cortex is one plausible mechanism of action that can be tested in randomized placebo controlled trials designed to specifically decrease intakes of linoleic acid and/or increase the intakes of EPA and DHA. Dietary interventions that reduce linoleate intake and improve the tissue status of n-3 FA and other basic nutrients (34) can potentially become relatively cost-effective measures for reducing the pandemic of violence in Western societies, just as dietary interventions are reducing cardiovascular mortality. Low linoleate diets may prevent behavioral maladies that correctional institutions, social service programs, and mental health providers intend to treat.

REFERENCES

1. Broadhurst, C., Cunnane, S., and Crawford, M. (1998) Rift Valley Lake Fish and Shellfish Provided Brain-Specific Nutrition for Early Homo, *Br. J. Nutr. 79,* 3–21.
2. Walter, R.C., Buffler, R.T., Bruggemann, J.H., Guillaume, M.M., Berhe, S.M., Negassi, B., Libsekal, Y., Cheng, H., Edwards, R.L., von Cosel, R., Neraudeau, D., and Gagnon, M. (2000) Early Human Occupation of the Red Sea Coast of Eritrea During the Last Interglacial, *Nature 405,* 65–69.
3. Niu, S.L., Mitchell, D.C., Lim, S.Y., Wen, Z.M., Kim, H.Y., Salem, N., Jr., and Litman, B.J. (2004) Reduced G Protein-Coupled Signaling Efficiency in Retinal Rod Outer Segments in Response to n-3 Fatty Acid Deficiency, *J. Biol. Chem. 279,* 31098–31104.
4. Salem, N., Jr., and Niebylski, C.D. (1995) The Nervous System Has an Absolute Molecular Species Requirement for Proper Function, *Mol. Membr. Biol. 12,* 131–134.
5. Gerrior, S., and Bente, L. (2002) Nutrient Content of the U.S. Food Supply, 1909–1999: A Summary Report, Home Economics Research Report. No 55, U.S. Department of Agriculture, Center for Nutrition Policy and Promotion, Washington, DC., Netlink: http://www.usda.gov/cnpp/nutrient_content.html (accessed December 2004).
6. Rahm, J.J., and Holman, R.T. (1964) Effect of Linoleic Acid upon the Metabolism of Linolenic Acid, *J. Nutr. 84,* 15–19.
7. Hibbeln, J.R., and Salem, N., Jr. (1995) Dietary Polyunsaturated Fatty Acids and Depression: When Cholesterol Does Not Satisfy, *Am. J. Clin. Nutr. 62,* 1–9.
8. Hibbeln, J.R. (2001) Seafood Consumption and Homicide Mortality, *World Rev. Nutr. Diet 85,* 41–46.
9. Iribarren, C., Markovitz, J.H., Jacobs, D.R., Schreiner, P.J., Daviglus, M., and Hibbeln, J.R. (2004) Dietary Intake of n-3, n-6 Fatty Acids and Fish: Relationship with Hostility in Young Adults—The CARDIA Study, *Eur. J. Clin. Nutr. 58,* 24–31.
10. Buydens-Branchey, L., Branchey, M., McMakin, D.L., and Hibbeln, J.R. (2003) Polyunsaturated Fatty Acid Status and Aggression in Cocaine Addicts, *Drug Alcohol Depend. 71,* 319–323.
11. Virkkunen, M.E., Horrobin, D.F., Jenkins, D.K., and Manku, M.S. (1987) Plasma Phospholipids, Essential Fatty Acids and Prostaglandins in Alcoholic, Habitually Violent and Impulsive Offenders, *Biol. Psychiatry 22,* 1087–1096.
12. Federal Bureau of Investigation. (2000) Homicide Trends in the United States. Uniform Crime Reports, 1950–99, Department of Justice, US Federal Government, Netlink: www.ojp.usdoj.gov/bjs/homicide (accessed December 2004).
13. Home Office. (2002) Criminal Statistics, National Statistics of the United Kingdom. http://www.homeoffice.gov.uk/rds/crimstats02.html (accessed September 2004)
14. Statistics Canada. (2001) Homicide in Canada, 85-002-XPE Government of Canada, Netlink: www.statcan.ca (accessed December 2004).
15. Ministerio de Justicia de la Nación. (2003) *Registro Nacional de Reincidencia y Estadística Criminal,* Government of Argentina, Buenos Aires.
16. Australian Institute of Criminology. (2002) Adapted from *Causes of Death Australia,* Catalogue No. 3303.0, Australian Bureau of Statistics, Netlink: http://www.abs.gov.au/ausstats/abs@nsf/Lookup/C86E6B498E247877CA256889000CCDE0 (accessed December 2004).
17. FAO/WHO. (2004) FAOSTAT Database, Food and Agricultural Organization of the United Nations, Sept 9, 2004, Netlink: http://faostat.fao.org/faostat/collections (accessed September 2004).
18. Krug, E.G., Powell, K.E., and Dahlberg, L.L. (1998) Firearm-Related Deaths in the United States and 35 Other High- and Upper-Middle-Income Countries, *Int. J. Epidemiol. 27,* 214–221.
19. Dandurand, Y. (1998) Firearms, Accidental Deaths, Suicide and Violent Crime: An Updated Review of the Literature with Special Reference to the Canadian Situation, WD1998-4e International Centre for Criminal Law Reform and Criminal Justice Policy, Netlink:http://canada.justice.gc.ca/en/ps/rs/rep/wd98-4a-e.html (accessed December 2004).
20. Australian Institute on Criminality. (2000) Australian Crime: Facts and Figures 2000. Homicide Involving Firearms as a Percentage of Total Homicide, 1915 to 1998, Australian Government, Netlink: http://www.aic.gov.au/publications/facts/2000/sec3.html (accessed December 2004).
21. Smith, C., and Allen, J. (2004) Violent Crime in England and Wales, Home Office Online Report 18/04, Home Office Re-

search, Development and Statistics Directorate Communication Development Unit, Netlink: http://www.homeoffice.gov.uk/rds/pdfs04/rdsolr1804.pdf (accessed December 2004).

22. Department of Justice. (2004) FBI Uniform Crime Reports Online, U.S. Federal Government, Netlink: http://bjsdata.ojp.usdoj.gov/dataonline/ (accessed December 2004).

23. Hemenway, D., and Miller, M. (2000) Firearm Availability and Homicide Rates Across 26 High-Income Countries, *J. Trauma 49,* 985–988.

24. Krug, E.G., Dahlberg, L., Mercy, J.C., Zwi, A.B., and Lozano, R. (2002) *World Report on Violence and Health,* World Health Organization, Netlink: http://www.who.int/violence_injury_prevention/violence/world_report/en (accessed December 2004).

25. American Academy of Pediatrics. (2000) Joint Statement on the Impact of Entertainment Violence on Children: Congressional Public Health Summit, Dec. 6, Netlink: http://www.aap.org/advocacy/releases/jstmtevc.htm (accessed December 2004).

26. Sanders, T.A. (2000) Polyunsaturated Fatty Acids in the Food Chain in Europe, *Am. J. Clin. Nutr. 71,* 176S–178S.

27. Meyer, B.J., Mann, N.J., Lewis, J.L., Milligan, G.C., Sinclair, A.J., and Howe, P.R. (2003) Dietary Intakes and Food Sources of Omega-6 and Omega-3 Polyunsaturated Fatty Acids, *Lipids 38,* 391–398.

28. Hibbeln, J.R. (2001) Seafood Consumption and Homicide Mortality: A Cross-National Ecological Analysis, *World Rev. Nutr. Diet. 88,* 41–46.

29. Zanarini, M.C., and Frankenburg, F.R. (2003) Omega-3 Fatty Acid Treatment of Women with Borderline Personality Disorder: A Double-Blind, Placebo-Controlled Pilot Study, *Am. J. Psychiatry 160,* 167–169.

30. Stevens, L., Zhang, W., Peck, L., Kuczek, T., Grevstad, N., Mahon, A., Zentall, S.S., Arnold, L.E., and Burgess, J.R. (2003) EFA Supplementation in Children with Inattention, Hyperactivity, and Other Disruptive Behaviors, *Lipids 38,* 1007–1021.

31. Hirayama, S., Hamazaki, T., and Terasawa, K. (2004) Effect of Docosahexaenoic Acid-Containing Food Administration on Symptoms of Attention-Deficit/Hyperactivity Disorder—A Placebo-Controlled Double-Blind Study, *Eur. J. Clin. Nutr. 58,* 467–473.

32. Hamazaki, T., Thienprasert, A., Kheovichai, K., Samuhaseneetoo, S., Nagasawa, T., and Watanabe, S. (2002) The Effect of Docosahexaenoic Acid on Aggression in Elderly Thai Subjects—A Placebo-Controlled Double-Blind Study, *Nutr. Neurosci. 5,* 37–41.

33. Hamazaki, T., Sawazaki, S., and Kobayashi, M. (1996) The Effect of Docosahexaenoic Acid on Aggression in Young Adults. A Double-Blind Study, *J. Clin. Invest. 97,* 1129–1134.

34. Gesch, C.B., Hammond, S.M., Hampson, S.E., Eves, A., and Crowder, M.J. (2002) Influence of Supplementary Vitamins, Minerals and Essential Fatty Acids on the Antisocial Behaviour of Young Adult Prisoners: Randomised, Placebo-Controlled Trial, *Br. J. Psychiatry 181,* 22–28.

35. Raygada, M., Cho, E., and Hilakivi-Clarke, L. (1998) High Maternal Intake of Polyunsaturated Fatty Acids During Pregnancy in Mice Alters Offsprings' Aggressive Behavior, Immobility in the Swim Test, Locomotor Activity and Brain Protein Kinase C Activity, *J. Nutr. 128,* 2505–2511.

36. Miachon, S., Augier, S., Jouvenet, M., Boucher, P., and Vallon, J.J. (2001) Nutritional Parameters Modify Muricidal Behavior of Male Wistar Rats: Preventive Effects Of Amino Acids and 4′ Cl Diazepam, *Life Sci. 69,* 2745–2757.

37. Augier, S., Penes, M.C., Debilly, G., and Miachon, A.S. (2003) Polyunsaturated Fatty Acids in the Blood of Spontaneously or Induced Muricidal Male Wistar Rats, *Brain Res. Bull. 60,* 161–165.

38. Stanley, B., Molcho, A., Stanley, M., Winchel, R., Gameroff, M.J., Parsons, B., and Mann, J.J. (2000) Association of Aggressive Behavior with Altered Serotonergic Function in Patients Who Are Not Suicidal, *Am. J. Psychiatry 157,* 609–614.

39. Highley, J.D. (2001) Individual Differences in Alcohol-Induced Aggression: A Nonhuman-Primate Model, *Alcohol Res. Health 25,* 12–19.

40. Kodas, E., Galineau, L., Bodard, S., Vancassel, S., Guilloteau, D., Besnard, J.C., and Chalon, S. (2004) Serotoninergic Neurotransmission Is Affected by n-3 Polyunsaturated Fatty Acids in the Rat, *J. Neurochem. 89,* 695–702.

41. de la Presa Owens, S., and Innis, S.M. (1999) Docosahexaenoic and Arachidonic Acid Prevent a Decrease in Dopaminergic and Serotoninergic Neurotransmitters in Frontal Cortex Caused by a Linoleic and α-Linolenic Acid Deficient Diet in Formula-Fed Piglets, *J. Nutr. 129,* 2088–2093.