Does Environmental Exposure to Manganese Pose a Health Risk to Healthy Adults?

Manganese is an essential nutrient that also may be toxic at high concentrations. Subjects chronically exposed to manganese-laden dust in industrial settings develop neuropsychological changes that resemble Parkinson’s disease. Manganese has been proposed as an additive to gasoline (as a replacement for the catalytic properties of lead), which has generated increased research interest in the possible deleterious effects of environmental exposure to manganese. Low-level exposure to manganese has been implicated in neurologic changes, decreased learning ability in school-aged children, and increased propensity for violence in adults. However, a thorough review of the literature shows very weak cause-and-effect relationships that do not justify concern about environmental exposure to manganese for most of the North American population.

Key words: manganese, environment, toxicity, violence, industrial pollution

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Manganese (Mn) is an essential metal nutrient. It is also toxic and toxicity is well documented in humans. Inhalation of Mn-laden dust can cause a Parkinson’s like neuromuscular condition that has primarily been described in miners who inhale large amounts daily. Toxicity is not considered a risk to individuals outside of Mn-contaminated industrial settings, and thus environmental overexposure to Mn has not been considered a research priority.

However, the journal Science recently featured an article entitled “Manganese: A High-octane Dispute,” which reviewed concerns of the use of methylcyclopentadienyl manganese tricarbonyl (MMT) as a replacement for lead in gasoline. Canada has approved use of MMT because they believe that the amount of Mn in the atmosphere from MMT in gasoline is insignificant compared with total daily environmental exposure, but recent studies suggest that low-level exposure to Mn causes adverse changes in behavior, cognition, and neurologic function. This has led to the hypothesis that, like lead (Pb), there is an incremental risk to increased exposure to Mn that can only be detected at the population level, not at the individual level.

Research that has shown adverse effects from environmental Mn has given rise to much popular concern and activism over the new “lead” in the environment. The World Wide Web lists hundreds of sites devoted to Mn toxicity, including a site that lists Mn-containing foods and provides direct links to attorneys with experience in Mn lawsuits. Other sites, such as the Violence Research Foundation, have the express purpose of demonstrating that environmental toxins, of which Mn is one of the most important, are a major cause of violent crime. The Violence Research Foundation has conducted nutritional supplementation programs in California prisons, and is currently conducting a much larger intervention study with inmates in the Mexico City jail (although these studies have been reported by news organizations, there are no reports in the scientific literature). The general-circulation periodical Popular Mechanics (June, 2003, The Chemistry of Violence) featured an article stating that Mn functions as “reverse Prozac,” decreasing brain serotonin and increasing propensity for violence. Numerous internet sites have published similar articles.

The hypothesis that Mn exposure causes violent behavior also has been advanced by some academicians. President R. Masters of the Foundation for Neuroscience and Society in the School of Government, Dartmouth College, has published 17 books and 150 research articles; he has proposed that Mn and Pb pollution explain many variations in violent crime rates. He hypothesizes that toxic metals cause impulsive and aggressive behavior, that exposure to these metals is high in inner cities, and that criminals are more likely to have been exposed during youth to these chemicals. These hypotheses were developed by comparing crime rates in U.S.
counties that had Environmental Protection Agency (EPA)—documented releases of Mn into the atmosphere (520/100,000; crimes/100,000 population) to counties without documented releases (350/100,000; significant difference, *P* = 0.004). Masters et al. concluded that, after controlling for demographic and socioeconomic variables, Pb and Mn pollution were risk factors for higher rates of violent crime.  

Consequently, environmental exposure to Mn has become a political issue. Even though diet is the greatest source of Mn exposure for most people, the nutrition community has had little to add to the discourse, and a thorough review of the available research shows that a vigorous debate is necessary. Certainly metals can alter behavior; a child exposed to Pb has a decreased IQ, although the impact of low-level exposure is minor compared with socio-demographic factors. Other studies report a link between Pb and Attention Deficit–Hyperactivity Disorder (ADHD). There is no doubt that Mn causes behavioral changes at high levels of exposure, and a Chinese report indicated that high concentrations of Mn in drinking water were associated with learning disabilities in schoolchildren.  

Acute Mn toxicity in humans, often called Mn-induced Parkinsonism, is characterized by progressive neurologic deterioration with bradykinesia, tremor, impaired postural reflexes and dystonia, and elevated whole blood, urine, and fecal Mn. It is believed that Mn3+ bound to transferrin crosses the blood-brain barrier and enters cells by using receptor-mediated endocytosis; magnetic resonance imaging (MRI) has documented Mn accumulation in the human brain of subjects with signs of toxicity. The specific mechanisms of toxicity are unknown but favored theories include oxidation of dopamine and inhibition of mitochondrial and/or synaptic cleft function. Mn retention is controlled in large measure by biliary excretion, and Mn accumulation in the brain is controlled by the blood-brain barrier; therefore infants, very young children, and adults with liver disease may be more at risk for Mn toxicity. Molecular studies have demonstrated that Fe and Mn may be transported in mammals by a divalent metal transporter (DMT1, DCT1, or Nramp2); this may explain why Fe deficiency increases Mn absorption and, perhaps, susceptibility to toxicity. The following discussion of possible negative effects of Mn does not apply to these groups, therefore, and is only applicable to healthy adults with adequate Fe status (although it is recognized that people with low Fe status or Fe deficiency may represent a sizable portion of the population).

Regulations regarding Mn exposure have been issued independently both by the EPA and the Occupational Safety and Health Administration (OSHA) (Table 1). In general, oral exposure limits are relatively high (limit of 10,000 μg/day; average daily intake of Mn from food ranges from 2000 to 9000 μg/d). Exposure limits for airborne sources of Mn are much lower, with the EPA reference dose set at 0.5 μg Mn/m³. Ambient air concentrations of Mn are 0.01 to 0.03 μg Mn/m³ in non-industrial areas and 0.01 to 0.07 μg/m³ in industrial areas. The daily intake of Mn from the air is 2 μg/day in areas without ferro- or silicomanganese industries, and as high as 10 μg/day in areas with such industries, while some short-duration 24-hour peak emissions have resulted in exposures of 200 μg/day (ambient air Mn concentrations of 0.2 to more than 0.5 μg/Mn/m³).

Oral Mn exposure levels set by the EPA agree with the “no observed adverse effects level” set by the Food and Nutrition board committee for the Institute of Medicine. Both groups have set the upper limit at 10 or 11 mg/day. This upper limit is almost five times the recom-

### Table 1. Manganese Exposure Limits Set by Various Governmental Agencies

<table>
<thead>
<tr>
<th>Measure</th>
<th>Definition</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>EPA RfC Environmental Protection Agency, Reference dose, airborne exposure</td>
<td>EPA, max daily intake over a lifetime</td>
<td>0.5 μg Mn/m³</td>
</tr>
<tr>
<td>TLV-TWA Threshold Limit Value–Time-Weighted Average</td>
<td>Occupational Safety and Health Administration (OSHA), airborne concentration all workers can be exposed for 40 h</td>
<td>0.2 mg/m³</td>
</tr>
<tr>
<td>REL-TWA Recommended Exposure Limit–Time-Weighted Average</td>
<td>OSHA, highest allowable airborne concentration in 10-h shift</td>
<td>1 mg Mn/m³</td>
</tr>
<tr>
<td>PEL-TWA Permissible Exposure Limit–Time-Weighted Average</td>
<td>OSHA, limit that must never be exceeded</td>
<td>5 mg Mn/m³</td>
</tr>
<tr>
<td>EPA Lowest Observed Adverse Effect Level (LOAEL)</td>
<td>EPA, maximum no effect airborne concentration</td>
<td>150 μg/m³</td>
</tr>
<tr>
<td>U.S. EPA Reference dose for oral exposure Adequate Intake (AI)</td>
<td>EPA, maximum oral intake over lifetime</td>
<td>10 mg/d</td>
</tr>
<tr>
<td></td>
<td>Food and Nutrition Board, Institute of Medicine</td>
<td>2.3 mg/d (men)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1.8 mg/d (women)</td>
</tr>
</tbody>
</table>
mended adequate intake for adult men (2.3 mg/d) or women (1.8 mg/d).

Behavioral and neuropsychologic tests have been used to study physiologic effects of occupational exposure to airborne concentrations of Mn well below those shown to cause acute toxicity; the results of seven studies are summarized in Table 2. These studies were conducted on workers in foundries and alloy production facilities, and residents down-wind of such facilities. Three studies showed clear deleterious effects of Mn exposure,\textsuperscript{35–37} one study found no effects,\textsuperscript{40} and the others had mixed or questionable findings.\textsuperscript{17,38,39,41–43}

Lucchini et al.\textsuperscript{31} found that Mn exposure impaired several neurologic functions, but blood Mn concentrations were not associated with exposure. Wennberg et al.\textsuperscript{32} reported that Mn exposure resulted in impaired finger tapping and digit span, but not electroencephalography (EEG) measures or psychiatric exams. Gibbs et al.\textsuperscript{33} did not find any significant effects of Mn exposure on multiple psychological tests and neurologic measures.

The studies of Mergler et al.\textsuperscript{14,34,35} have received much attention. Subjects from southwestern Quebec, who lived downwind of industrial facilities with a history of Mn release, were divided into two groups with blood Mn concentrations greater than or less than 7.5 \(\mu g/L\). Older men with higher blood Mn had significant disturbances in mood scores.\textsuperscript{14} However, the most serious effects on psychological (distress, learning, and recall) and neurologic (coordinated upper limb movements and tremor) measures were in men with high blood Mn who also were heavy consumers of alcohol (>420 g/wk).\textsuperscript{35}

The authors concluded that Mn neurotoxicity was a continuum of dysfunction with subtle changes at all levels of intake. However, the authors did not justify using 7.5 \(\mu g\) Mn/L blood as a breakpoint for designation of high or low Mn exposure, and most significant effects were two- or three-way interactions, making it difficult to determine the real association. Thus, the mass of this literature still does not give clear indications of the danger of low-level Mn exposure.

There are a few reports of environmental exposure to sources of Mn other than a Mn-emitting industry. An Australian island with extensive geologic deposits of Mn has been mined since the 1960s, and Mn exposure was blamed for neurologic and behavioral impairment in the indigenous population. Residents not involved in mining had elevated concentrations of Mn in the hair and there are numerous reports of neurologic dysfunction.\textsuperscript{36} In reviewing the medical data, however, Kilburn\textsuperscript{36} concluded that “available evidence can only implicate manganese by association. . . until then . . . manganese must remain an ‘element of doubt.’” Loranger and Zayed\textsuperscript{37} used mathematic modeling to predict Mn exposure in the St. Lawerence ecozone (the same general area studied by Mergler et al.) from gasoline-emitted MMT. They showed that more than 99% of total exposure was through food, and the contribution of Mn from MMT was biologically insignificant. Kondakis et al.\textsuperscript{38} studied elderly Greek adults (65–68 y) that consumed well water from three sites with Mn concentrations of 0.004 to 0.15, 0.08 to 0.25, and 1.8 to 2.3 mg Mn/L. Compared with non-exposed controls, individuals with higher Mn intakes showed significantly impaired neurologic scores and increased hair Mn concentrations. However Vierregge et al.\textsuperscript{38} found no neurologic impairment in subjects that had consumed water that contained more than 0.3 to 2.1 mg Mn/L for more than 40 years. Chinese children exposed to fields with sewage irrigation and high Mn content (0.24–0.35 mg/L, high; 0.03–0.04 mg/L, control) had significantly impaired short-term memory, manual dexterity, and visuo-perceptive speed.\textsuperscript{15} The route of Mn exposure was believed to have been through well water, but other sources were not investigated.

Data supporting the hypothesis that Mn exposure can increase violent crime risk also have many weaknesses. Masters\textsuperscript{10} analyzed a subset of the data comparing crime rates in areas with and without Mn release. When a t-test was used to compare counties with violent crime rates over 400/100,000 or less than 100/100,000, higher crime rates were significantly associated with more reports of Mn release. However, the associations became more complex when other variables were con-

### Table 2. Reports of Neuropsychological Impairment of Subjects Exposed to Low Concentrations of Airborne Mn

<table>
<thead>
<tr>
<th>Study</th>
<th>Subjects</th>
<th>Mn Exposure</th>
<th>Duration</th>
<th>Significant Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Iregren\textsuperscript{28}</td>
<td>Foundry workers</td>
<td>150 (\mu g/m^3), air</td>
<td>10 y</td>
<td>Y</td>
</tr>
<tr>
<td>Roels et al.\textsuperscript{29}</td>
<td>Foundry workers</td>
<td>950 (\mu g/m^3), air</td>
<td>5.2 y</td>
<td>Y</td>
</tr>
<tr>
<td>Mergler et al.\textsuperscript{30}</td>
<td>Alloy production workers</td>
<td>230 (\mu g/m^3), air</td>
<td>14.6 y</td>
<td>Y</td>
</tr>
<tr>
<td>Lucchini et al.\textsuperscript{31}</td>
<td>Alloy production workers</td>
<td>157–1597 (\mu g/m^3), air</td>
<td>NA</td>
<td>?</td>
</tr>
<tr>
<td>Wennberg et al.\textsuperscript{32}</td>
<td>Foundry workers</td>
<td>160–410 (\mu g/m^3), air</td>
<td>NA</td>
<td>?</td>
</tr>
<tr>
<td>Gibbs et al.\textsuperscript{33}</td>
<td>Foundry workers</td>
<td>200 (\mu g/m^3), air</td>
<td>NA</td>
<td>No</td>
</tr>
<tr>
<td>Mergler et al.\textsuperscript{14,34,35}</td>
<td>Residents down-wind of Mn-emitting industries</td>
<td>Low, but unknown</td>
<td>NA</td>
<td>?</td>
</tr>
</tbody>
</table>
would increase Mn exposure to 99% of the population by less than 0.15 μg/m³, resulting in a total of only 3 μg of additional Mn absorbed/day.4

Dietary studies have demonstrated that intakes of >5 mg Mn/day can be safely tolerated by healthy adults. Davis et al.35 supplemented women with 15 mg of Mn/day for 124 days (total dietary intake plus supplemental intake of 17 mg/d) and reported only elevated plasma Mn concentrations and lymphocyte MnSOD activities. Urinary Mn, an excretory route that becomes more important at high Mn intakes, was unaffected by treatment. Finley24 fed women either less than 1 or 9.5 mg Mn/day for 60 days and found that higher Mn intakes were compensated for by decreased absorption and increased excretion. In a follow-up study,46 subjects were fed <1.0 or 20 mg Mn/day for 60 days in a cross-over design. An extensive battery of psychological tests and neurologic exams was administered before and after dietary periods, and Mn whole-body counting was used to estimate Mn absorption retention and turnover. Measures of Mn status (plasma and lymphocyte Mn) were unaffected by dietary Mn; the efficiency of Mn absorption (%) and biologic half-life were almost twice as great in subjects fed low dietary Mn than in subjects fed high dietary Mn, again demonstrating homeostatic control of Mn retention. As a result, neurologic tests were unaffected by Mn status, and the only psychological variable affected was self confidence (decreased self confidence with high dietary Mn).

The above discussion applies to healthy adults with adequate Fe status. There is evidence, however, that low Fe status may alter Mn homeostasis. Low-Fe diets increase Mn absorption,47 and Chua and Morgan reported that Fe-deficient diets had increased uptake of Mn into the brain.48 Iron deficiency in humans also enhances Mn absorption; women in the lowest 10% of serum ferritin concentrations absorbed three- to fivefold more Mn than women in the top 10% of serum ferritin concentrations.24 Presently, however, there is no direct evidence that women with low Fe status are at increased risk of toxicity from inhaled or oral exposure to moderate concentrations of Mn.

Consequently, a review of the available literature reveals only circumstantial evidence that minor environmental exposure of healthy adults to Mn can have deleterious effects. A notable exception may be women with low Fe status, for whom there is evidence that Mn absorption may be greatly increased.24 It is well accepted that inhalation of toxic amounts of Mn (e.g., >1.0 mg/d) can affect neuropsychological function; such knowledge may have caused investigators to see trends and patterns and suggest hypotheses regarding low-level exposure to Mn that simply are not supported by the data.

Table 3. Reports of Associations between Trace Element Concentrations in Hair and Propensity for Violent Crime

<table>
<thead>
<tr>
<th>Study</th>
<th>Metal Concentrations Significantly Elevated in Hair of Violent Criminals</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gottschalk et al.39</td>
<td>Mn</td>
</tr>
<tr>
<td>Schauzer et al.40</td>
<td>Mn, Li decreased</td>
</tr>
<tr>
<td>Pihl and Ervin41</td>
<td>Pb, Cd</td>
</tr>
<tr>
<td>Marlowe et al.42</td>
<td>Pb, Cd, Hg, Si; Li decreased</td>
</tr>
<tr>
<td>Schauss et al.43</td>
<td>Pb, Cd, Cu; Mn non-significantly decreased</td>
</tr>
</tbody>
</table>
37. Kondakis X, Makris N, Leotsinis M, Prinou M,
Beverage Consumption and Risk of Obesity among Native Americans in Arizona

Native Americans face some of the highest rates of obesity and diabetes in the world. Despite numerous education programs to reduce obesity among Native Americans, little attention has been paid to reducing fructose, particularly in the form of high-fructose corn syrup in beverages. Considerable data indicate that energy from beverages does not displace energy from other foods throughout the day, often leading to energy imbalance, and numerous studies have documented that beverages are a leading contributor to energy intakes among Native Americans. Prevention programs that target pregnant women and parents of infants and very young children are necessary to halt the epidemic of obesity among Native Americans; one approach may be by promoting sugar-free beverages.

Key words: Native American, beverage, energy, obesity

Across all age groups and ethnicities, the well-recognized epidemics of overweight and obesity grow worse with each passing year. The majority of adults in the United States (~64.5%) are overweight or obese based on body mass index (BMI, kg/m²), and between 10 and 15% of U.S. children are overweight. Preventing and treating overweight and obesity are high priorities because these conditions are associated with an increased risk for chronic diseases, including cardiovascular disease, diabetes, hypertension, pulmonary stress, and orthopedic problems.

Native Americans particularly suffer from obesity and related health issues, beginning very early in life. Recent data from the Pathways Study (n = 1704) showed that 30.5% of girls and 26.8% of boys were greater than the 95th percentiles for BMI-for-age (i.e., overweight); 21.0% of girls and 19.6% of boys had a BMI-for-age that was ≥85th and <95th percentiles (i.e., at risk for overweight). The proportion of Native American children with a BMI-for-age ≥85th percentile was consistently higher than national averages in all of the communities studied. For example, based on recent data from the National Health and Nutrition Examination Survey (NHANES), 11% of 6- to 11-year-olds across the country have a BMI-for-age that is greater than the 95th percentile, compared with 28.6% of Native American children of the same age in the Pathways study.

Based on research conducted in Arizona, obesity appears to begin very early in life among Native Amer-
icans. Lindsay et al.\textsuperscript{10} measured the birth weights of Pima Indians and found they were comparable to, but slightly heavier than, the general population. After one month of life, however, weight-for-length and BMI-for-age were significantly higher among the Pima than reference standards published by the Centers for Disease Control and Prevention (CDC), National Center for Health Statistics. This trend continued until 6 months, when weight began to change at a rate comparable to reference standards; however, weight increased greatly once again between the ages of 2 and 11 years. Although the increase in weight between 2 and 6 months may not indicate a true change in adiposity, weight changes between 2 and 11 years were the consequence of an increase in fat mass, which reflects changes early in life that affect life-long risk of obesity and chronic disease.

Other studies have reported excess fat accumulation during childhood among Native Americans. The 1990 American Indian School Children Height and Weight Survey, conducted by the Indian Health Service, showed that 40\% of 5- to 18-year-old Native Americans (\(n = 9464\)) were overweight, using the now-outdated definition of BMI \(\geq 85\text{th}\) percentile, NHANES II reference population.\textsuperscript{6} Other data assessing body weight among Native American 5- to 18-year-olds (\(n = 155\)) found that 22\% of children were at risk for overweight (BMI-for-age between the 85th and 95th percentiles), while 41\% of the sample were already overweight (BMI-for-age \(\geq 95\text{th}\) percentile).\textsuperscript{4}

Obesity remains a complex and unresolved issue for Native Americans and other populations, even though the etiology of the disease is fundamentally simple. Weight gain results from an excessive consumption of energy that is not balanced by adequate energy expenditure, such as physical activity and exercise. Although Native American children tend to watch more television and spend less time playing sports than white children, this is likely not the principle contributor to obesity.\textsuperscript{6,11} Salbe et al.\textsuperscript{12} reported that sports activities were not related to percent body fat among 5-year-old Pima children (\(r = -0.10, P = 0.25\)) and only weakly related to percent body fat among the children at 10 years of age (\(r = -0.22, P = 0.01\)). These data emphasize the important role of dietary intake in the epidemics of overweight and obesity, especially among young children.

The health effects of numerous food components, including fat and saturated fat, are well known by the public, but the role of fructose is often overlooked, even though it may contribute to excessive intakes of energy and compound the obesity trend. The practice of using high-fructose corn syrup in food and beverage production began relatively recently. Initial use of high-fructose corn sweetener occurred in the 1970s, but by 1985, high-fructose corn syrup accounted for approximately 35\% of the total amount of sweeteners in the U.S. food supply.\textsuperscript{3} Based on data from the 1977–1978 Nationwide Food Consumption Survey, Park and Yetley\textsuperscript{13} reported that the mean fructose intake in the United States was 40 g/day, most of which came from sources other than fresh fruits and other natural products. These data, however, likely underestimate current average intakes.

More recent food disappearance data show that per capita usage of high-fructose corn syrup increased from 0.23 kg in 1970 to a staggering 28.4 kg in 1997, greater than a 100-fold increase in less than 30 years.\textsuperscript{14} Much of the increase in dietary fructose can be attributed to increased intakes of high-fructose beverages, such as soft drinks, juices, and juice drinks. The U.S. Department of Agriculture reported an increase in per capita soft drink consumption by almost 500\% during the past 50 years.\textsuperscript{14} In 1997, per capita consumption of soda in the United States was 204 L, with only 24\% sweetened with sugar substitutes, and per capita consumption of juice was 33 L.\textsuperscript{15}

Fructose utilization in the body lends itself to hepatic triglyceride production. Due to its entry into the glycolysis pathway at a much later step than glucose itself, fructose preferentially provides carbon atoms for both the glycerol and fatty acid portions of triglycerides.\textsuperscript{3} Indeed, fructose ingestion has been shown to markedly increase rates of de novo lipogenesis compared with glucose.\textsuperscript{16,17} In addition, fructose does not stimulate the production of insulin or leptin, which are important hormones involved in the long-term regulation of energy homeostasis.\textsuperscript{3}

Since the late 1970s, total daily energy intakes have increased by nearly 200 kcal/day due mainly to increases in carbohydrate consumption.\textsuperscript{15} Much of the carbohydrate increase in the diet can be attributed to high-fructose beverage intake, and sugar given as a liquid seems to promote obesity more consistently than sugar given as a solid.\textsuperscript{18} Tordoff and Alleva\textsuperscript{19} asked subjects to drink 4 bottles/day (1135 g) of soda sweetened with either high-fructose corn syrup or aspartame. After 3 weeks of consuming soda sweetened with high-fructose corn syrup, female subjects gained significantly more weight (0.97 kg, \(P < 0.01\)). Men also gained weight (0.52 kg), but the difference was not significant. By contrast, men lost significantly more weight (0.47 kg, \(P < 0.05\)) after 3 weeks of drinking soda sweetened with aspartame.

A review of recent literature suggests that caloric compensation might occur only if a test meal is provided very quickly after a liquid load ingestion (0–30 min later).\textsuperscript{20} Such compensation might depend on gastric emptying or the rapidity of metabolism of the load.\textsuperscript{20}
Timing, therefore, might be as important as the liquid caloric load itself.

Data from the 1994–1996 Continuing Survey of Food Intakes by Individuals (CSFII) show that Americans frequently choose beverages, especially colas, as snacks.21 Chamnugam et al.22 compared dietary intakes between 1989–1991 and 1994–1996 and showed that soft drink intake increased significantly \( (P < 0.05) \) and that this change was responsible for most of the increase in energy intake from beverages. Of all the food groups assessed, whole milk and soft drink intakes changed the most during the brief time interval: whole milk intake decreased by \( \sim 100 \, \text{g/day} \) and soft drink intake increased by \( \sim 90 \, \text{g/day} \). An increase of approximately 85 g/day in reduced-fat milk consumption was noted in these data as well; this was nearly enough to offset the large decrease in whole milk consumption. Other findings by Mrdjenovic and Levitsky,23 however, showed that consumption of sugary beverages displaced milk in children’s diets, leading to significantly lower intakes of protein, calcium, magnesium, and vitamin A.

DiMeglio and Matte15 fed subjects isoenergetic carbohydrate loads as either jelly beans (a solid load) or soda (a liquid load) during 4-week periods. Based on dietary records, subjects eating the solid load consumed significantly \( (P < 0.001) \) less energy while free-feeding than prior to the intervention; when combined with the energy contribution from the jelly beans, however, there was no change in energy intake. By contrast, the liquid load did not cause a reduction in free-feeding energy intake, causing total energy intakes to increase significantly by \( \sim 17\% \).15 While measurements of caloric intake were not made under precise laboratory conditions for comparison, significant increases were noted in both body weight and BMI only during the liquid load period.15

Raben et al.24 reported that subjects whose diets were supplemented with sucrose (\( \sim 152 \, \text{g sucrose/day}, \, 70\% \) from soft drinks) for 10 weeks had significantly higher energy intakes, body weight, fat mass, and blood pressure; decreases or no changes in these variables resulted when subjects consumed artificially sweetened supplements (0 g sucrose/day). Raben et al.24 specifically noted that energy obtained from sweetened beverages is less satisfying than energy from solid foods, making it easier to over-consume energy when drinking liquids than when eating solids.

De Castro25 investigated the effects of various beverages and foods on energy displacement and reported that subjects \((n = 323)\) who drank sugar-free soda consumed significantly less carbohydrate throughout the day than individuals who did not drink diet soda. By contrast, individuals who drank sugary sodas consumed more carbohydrate and energy on days when they drank soda than on days when they did not. After subtracting the energy content of beverages from total energy intakes, energy intakes became equal for both the days the drinks were consumed and the days the drinks were not.25

These data indicate that energy derived from beverages leads to elevated energy intakes and does not displace energy ingested from other foods (i.e., individuals do not compensate by reducing food intake). With a sample of 135 preschool children, Wilson26 studied whether lunch-time beverages affected children’s energy intake at lunch and during a mid-afternoon snack. Although chocolate milk sweetened with sucrose caused the children’s lunch-time energy intakes to increase significantly (\( \sim 65 \) extra kcal compared with plain milk and milk sweetened with aspartame, \( P < 0.05) \), there was no compensation for energy when the children selected their afternoon snack 3 hours later.

The risk of nutrition-related health problems is greatest among low-income populations, and Native Americans as a group are economically disadvantaged.27,28 More than 40% of Native Americans live below 100% of the federal poverty level, and this percentage is even higher for Native Americans living on reservations or trust lands.29 Unemployment is high, and in some cases, households lack running water and electricity, general necessities for cooking.30–32 Along with the difficulties of poverty come inevitable hunger and food insecurity issues. Even so, underweight is not a major health problem for this group.27 Instead, Native Americans suffer from the highest prevalence of overweight and obesity among all racial/ethnic groups.6

Sugary beverages may play an important role in obesity development among Native Americans. In 1959, Hesse33 was the first to note the high consumption of sweetened beverages among Native Americans in Arizona: “...a large amount of soft drinks of all types is consumed between meals.” Unfortunately, though, Hesse did not quantify sugary beverage consumption or publish these results, and nearly 45 years later, fast food restaurants, grocery stores, and convenience stores have proliferated on and near reservations, increasing the availability of high-sugar beverages.6,27

Teufel and Dufour31 reported that their sample of 18- to 35-year-old Hualapai women (Figure 1) ate at restaurants—including fast food restaurants—an average of 5 times per week. The leading source of energy in the diets of these women was non-alcoholic beverages, particularly drinks made from powdered mixes and sodas. On average, these beverages contributed 359 kcal/day to women’s diets, which comprised nearly 14% of energy intake. Vaughan et al.34 evaluated the dietary intake of 92 Havasupai adults (\( \sim 30\% \) of the population of the Supai village) and found that sugars were responsible for
14% of energy intake. Specifically, Kool-Aid® (Kraft Foods, Northfield, IL) and soda accounted for 11% of subjects’ energy intakes; on average, adults consumed 29 fluid ounces of sugary beverages each day.

Ballew et al.35 found that soft drinks contributed 7% of the energy intake of subjects in the Navajo Health and Nutrition Survey. Although this percentage is low compared with other studies, Ballew et al.35 did not stratify their subjects into age groups; rather, subjects’ ages ranged from 12 to over 60. Had children and young adults been considered specifically, soft drinks likely would have contributed more energy to overall intakes, as seen by the significantly higher intake of vitamin C, a marker of consumption of beverages like Kool-Aid and Tang® (Kraft Foods, Northfield, IL).34 among the 12- to 19-year-olds and 20- to 39-year-olds.35

Koehler et al.32 assessed the dietary intake of Navajo 9- to 16-year-olds (n = 68) by determining “core foods,” defined as foods or beverages that were consumed at least 3 to 5 times a week. The leading core food among these children was soda, with 71% of the children reporting frequent consumption. The next leading core foods were fruit juices and potatoes/French fries, which were frequently consumed by 67% of the children. Gilbert et al.36 substantiated these results with their finding that sugary sodas were the most commonly reported food or drink for school-aged Navajo boys and girls (n = 352). In fact, 86% of girls and 93% of boys reported drinking sugary sodas in the two 24-hour recalls obtained in the study.

Lytle et al.37 studied the dietary intake of 80 Native American children from various tribes involved in the Pathways Study (including the White Mountain Apache, San Carlos Apache, Navajo, Tohono O’odham, and Gila River Indian Community of Arizona). They demonstrated that sugary beverages, especially Kool-Aid® and soda, were the leading contributors to children’s energy intakes, providing 9.5% of energy from meals and snacks eaten at home and other non-school sites. Brown and Brenton30 reported that 38% of total carbohydrate intake of Hopi children (n = 96) came from simple sugars, the majority of which were provided in beverages. This amounts to nearly 400 kcal, or 19% of mean energy intake.

Further evidence of excess consumption of sugary beverages comes from dental research, which shows that Native American children suffer rates of dental decay that are five times higher than the average U.S. child.30,40 Although the literature related to bottle-feeding practices among Native Americans is extraordinarily small, Douglass et al.41 reported that up to 85% of Native American preschool children have early childhood caries, which are associated with inappropriate or prolonged bottle-feeding with sugary beverages. Weinstein42 noted that Native American infants develop caries by the age of 1 year due to prolonged exposure to refined carbohydrates. The American Academy of Pediatrics recommends exclusive breastfeeding for the first 6 months of life.43 Native American mothers generally have a lower breastfeeding incidence and duration than white women, which reflects cultural changes that have reduced breastfeeding as a traditional practice.29,44 Data from the 2002 Pediatric Nutrition Surveillance System show that more than 70% of Native American women in Arizona initiate feeding at birth.
breastfeeding, but only ~20% of infants are still breastfed at 6 months of age.\textsuperscript{33}

Obesity is a multi-factorial condition, with no ideal strategy to reverse its devastating outcomes. Because the likelihood of undoing the damage of chronic, excessive energy intake is minimal, prevention techniques offer the best solutions. A focus on education to change unhealthy behaviors before they become a way of life is integral in preventing overweight and obesity.

Data show that Native Americans frequently choose sugary beverages; this behavior can be linked to specific reasons, including quenching of thirst, appeal, and availability. For example, much of Arizona is in the Sonoran Desert, where the average high temperature exceeds \(38^\circ\text{C} (100^\circ\text{F})\) for several months.\textsuperscript{45} The arid climate continuously prompts thirst signals, and dehydration quickly follows unless fluids are consumed. Numerous authors have noted that whereas intake of sugary beverages is high throughout the year, intake increases in the summer months.\textsuperscript{33,34}

Often, sugary beverages are marketed toward younger age groups; collectively, the Native American population in Arizona is very young and might find sugary beverages especially appealing. About 40% of the Native American population is under 20 years of age with a median age of 26 years.\textsuperscript{27,28} Furthermore, healthy food supplies and transportation are limited for many Native Americans due to the impoverished conditions in which many live.\textsuperscript{6,46} Inexpensive, less healthy food items (such as powdered drink mixes and sodas) are suitable for long-term storage but provide greater amounts of sugar and energy. These items are purchased at the expense of healthier choices, like fruits and vegetables,\textsuperscript{35} and are more readily available.

Although ample evidence exists demonstrating the high-sugar intakes among Native Americans, the focus of health and diet education remains on the reduction of fat intake to combat obesity.\textsuperscript{8,13,32,37,47} More work is needed to remind consumers that sugary beverages are a leading contributor to the “empty calories” of excess simple sugars and energy. Native Americans, in particular, need exposure to this message; unfortunately, Native Americans are disparately under-exposed to health media overall.\textsuperscript{48} When Native Americans are included, they are often portrayed stereotypically (e.g., the savage warrior, the alcoholic).\textsuperscript{49} Social marketing may be a key way to reach Native Americans with nutrition messages: previous research with the Pima has shown that a less direct and less structured intervention—working through cultural and holistic principles—is preferable to a direct and highly structured approach to modify diet and activity.\textsuperscript{46}

Treatment of obesity, even among children, is very often ineffective,\textsuperscript{50} and childhood obesity easily transitions to adulthood obesity, especially among children with a familial history of weight problems.\textsuperscript{47,51} Prevention and education programs that focus attention on pregnant women, parents of infants, and very young children, might be the key to preventing these weight problems, before large amounts of adipose tissue develop. Using novel routes of communication that focus on healthier beverage choices and other lifestyle behaviors among Native Americans is one strategy deserving of further research.


50. Alaniz ML, Wilkes C, Pro-drinking messages and message environments for young adults: the case of alcohol industry advertising in African American,

