Surrogate Alcohol: What Do We Know and Where Do We Go?

Dirk W. Lachenmeier, Jürgen Rehm, and Gerhard Gmel

Background: Consumption of surrogate alcohols (i.e., nonbeverage alcohols and illegally produced alcohols) was shown to impact on different causes of death, not only poisoning or liver disease, and appears to be a major public health problem in Russia and elsewhere.

Methods: A computer-assisted literature review on chemical composition and health consequences of "surrogate alcohol" was conducted and more than 70 references were identified. A wider definition of the term "surrogate alcohol" was derived, including both nonbeverage alcohols and illegally produced alcohols that contain nonbeverage alcohols.

Results: Surrogate alcohol may contain substances that cause severe health consequences including death. Known toxic constituents include lead, which may lead to chronic toxicity, and methanol, which leads to acute poisoning. On the other hand, the role of higher alcohols (e.g., propanol, isobutanol, and isoamyl alcohol) in the etiology of surrogate-associated diseases is currently unclear. Whether other constituents of surrogates have contributed to the high all-cause mortality over and above the effect of ethanol in recent studies also remains unclear.

Conclusions: Given the high public health importance associated with the consumption of surrogate alcohols, further knowledge on its chemical composition is required as well as research on its links to various disease endpoints should be undertaken with priority. Some interventions to reduce the harm resulting from surrogate alcohol could be undertaken already at this point. For example, the use of methanol or methanol-containing wood alcohol should be abolished in denatured alcohol. Other possible surrogates (e.g., automobile products) should be treated with bittering agents to avoid consumption.

Key Words: Surrogate Alcohol, Moonshine, Nonbeverage Alcohol, Illegal Alcohol, Homemade Alcohol, Methanol, Lead Poisoning, Public Health.

RECENTLY, AN ARTICLE in Lancet concluded that consumption of surrogate alcohol ceteris paribus accounted for more than 30% male mortality in the age group of 25 to 54 in the Russian town of Izhevsk in recent years (own calculations based on Leon et al., 2007; formula in Hanley, 2001; see below for details). Drinking of surrogate alcohols impacted different causes of death including cardiovascular disease, not only poisoning or liver diseases. It seems to constitute a major public health problem in this part of Russia.

There have been other reports on mortality associated with surrogate alcohol, especially in relation to consumption of methanol (see below). However, a number of different types of alcohol have been labeled "surrogate alcohol," and the active pathways leading to death are far from clear. For instance, Leon et al. (2007) restricted themselves to nonbeverage alcohol, i.e., manufactured ethanol-based liquids not intended for consumption such as aftershaves, whereas other authors use the term for all forms of illegally produced alcohol (e.g., McKee et al., 2005) or only for forms of nonbeverage alcohol that are not defined in statistics on alcohol (Nordlund and Österberg, 2000). This article will try to give an overview of the different definitions of "surrogate alcohol." Further, we will examine the chemical composition of the different forms of surrogate alcohol and their impact on health outcomes. We conclude with a discussion on potential steps to reduce the harm of surrogate alcohol as well as suggested research to fill the gaps in our knowledge about it.

MATERIALS AND METHODS

The current knowledge about surrogate alcohol was compiled by a computer-assisted literature search in the following databases: PubMed (U.S. National Library of Medicine, Bethesda, MD), Web of Science (Thomson Scientific, Philadelphia, PA), Food Science and Technology Abstracts (International Food Information Service, Shinfield, UK), and Scopus (Elsevier B.V., Amsterdam, Netherlands). The following terms were searched: "surrogate alcohol," "nonbeverage alcohol," "denatured alcohol/spirits," "methylated alcohol/spirits," "moonshine," "illegal alcohol/spirits," "illicit alcohol/spirits," "methanol intoxication/poisoning," and "lead

From Chemisches und Veterinäruntersuchungsamt (CVUA) Karlsruhe, Karlsruhe, Germany (DWL); CAMH, Toronto, Ontario, Canada (JR); the Alcohol Treatment Center, Lausanne University Hospital, Lausanne/Switzerland (GG); and the Swiss Institute for the Prevention of Alcohol and Drug Problems, Lausanne, Lausanne/Switzerland (GG).

Received for publication March 27, 2007; accepted June 22, 2007.

Reprint requests: Dirk W. Lachenmeier, Dr. rer. nat., Chemisches und Veterinäruntersuchungsamt (CVUA) Karlsruhe, Weißenburger Street 3, D-76187 Karlsruhe, Germany; Fax: +49-721-926-5539; E-mail: Lachenmeier@web.de

DOI: 10.1111/j.1530-0277.2007.00474.x

intoxication/poisoning." The references including abstracts were imported into Reference Manager V.11 (Thomson ISI Research Soft, Carlsbad, CA) and the relevant articles were manually identified and purchased in full text. The reference lists of all articles were checked for relevant studies not included in the databases.

DEFINITION OF SURROGATE ALCOHOL

Surrogate alcohol has not been consistently defined in the literature. Under this broad heading, some authors include illegally produced alcohol intended for consumption as well as alcohols that are not initially intended for consumption (McKee et al., 2005). It should be noted that homemade alcohols are usually illegally produced, but there are exceptions where home production is not illegal, but would be part of unrecorded consumption. We subsume legally homemade alcohols under the category of surrogate alcohol. Others more strictly define surrogate alcohol as substances that contain ethanol or possibly other alcohols, but are "not intended for consumption," such as medicinal compounds, aftershaves, industrial spirits, or fire lighting liquids (Lang et al., 2006). Nordlund and Österberg (2000) in their overview for the Nordic countries, even split up alcohols "not intended for consumption" into those that appear in alcohol statistics and those that do not. The former category comprises alcohols produced for industrial, technical, and medical purposes. The latter category, which they define as "surrogate alcohol," is made up of denatured alcohol or other products such as medicine or car chemicals that contain alcohol but are meant for other purposes such as car washing. Denaturing of alcohol occurs in many countries and is undertaken for the purposes of exemption from excise duty that is applied to nondenatured forms. In Russia (e.g., Savchuk et al., 2006), surrogate alcohols are differentiated based on the type of alcohol that the liquid contains. There are 2 classes of surrogates here: true surrogate alcohols (i.e., solutions and liquids manufactured from ethanol or containing large amounts of ethanol) and false surrogate alcohols (i.e., ethanol-free liquids such as methanol, propanol, and ethylene glycol). Thus, we can distinguish 3 distinct meanings of the term "surrogate alcohol" in the literature:

1. As a synonym for all nonbeverage alcohols, i.e., all alcohols not intended for human consumption;
2. Denoting only nonbeverage alcohols outside production data;
3. Denoting both nonbeverage alcohols and illegally produced or homemade alcohols.

In this review, we will use the last definition, as in some instances alcohols illegally produced for human consumption contain nonbeverage alcohols, e.g., to increase alcohol concentration. Thus, beverage alcohol that is offered for consumption on the illegal market is often adulterated by nondrinkable alcohol (e.g., sold as aquardiente in Mexico; Medina-Mora, 1999), and consumers may not be aware of the potential risks. Similarly, in Russia, it appears that denatured industrial ethanol is used for producing illegal alcohol for consumption as it is possible to eliminate the common denaturing agent diethyl phthalate through simple distillation (Savchuk et al., 2006). There is also evidence that some heavy drinkers, commonly the economically most disadvantaged, mix beverage alcohol with industrial denatured alcohol themselves. In addition, as argued by McKee et al. (2005), in some, mainly eastern European countries it is speculated that the production of surrogate alcohol is actually intended for consumption, e.g., medicinal alcohols sold in much larger bottles than in western Europe with colorful labels or aftershaves without a discernibly pleasant scent or warning labels such as "for external use only." While we will include such illegal beverages in the below review, we will exclude beverages that are produced in the same factories as "normal recorded" alcohol (i.e., beer factories, distilleries, and wineries), but then are not recorded in order to evade taxation.

Overall, data on the amount of surrogate alcohol used in different parts of the world is scarce. Based on the available data from the Global Alcohol Database of the World Health Organization (http://www.who.int/globalatlas/default.asp), the estimates of unrecorded consumption are summarized in Table 1. As explained in the last paragraph, surrogate alcohols do not constitute the total of unrecorded consumption, it is fair to say, that they constitute a considerable part

Table 1. Estimates of Unrecorded Alcohol Consumption From the Global Alcohol Database of the World Health Organization

WHO Regions	Per capita recorded + unrecorded alcohol consumption (in liters of pure alcohol)	Per capita unrecorded alcohol consumption (in liters of pure alcohol)	Percentage of unrecorded consumption to total consumption (%)
Africa	7.03	2.48	35.3
America A: Canada and United States	9.42	1.13	12.0
Central and South America	8.02	2.53	31.5
Eastern Mediterranean	0.7	0.52	74.3
Western Europe (Europe A)	12.15	1.32	10.9
European B: Central and Eastern Europe	7.51	2.83	37.7
European C: Russia and surrounding countries	14.91	6.07	40.7
South East Asia (including India)	2.01	1.49	74.1
Western Pacific A: Australia, Japan, and New Zealand	9.38	1.70	18.1
Western Pacific B: China and Pacific	6.02	1.13	18.8
World	6.16	1.72	27.9

of this category. The exact proportion of surrogates as defined above in unrecorded consumption is unclear; however, as for most parts of the world we do not have quantifiable information about the composition of unrecorded consumption. In some parts of the world, such as in some African countries, homebrew may constitute the largest part of unrecorded consumption, in other parts, for instance Sweden, legally imported alcohol via travel allowance may constitute the largest part (see Global Alcohol Database).

Clearly, unrecorded consumption is mainly a problem of countries that are economically least or medium resourced. In the highest-resourced countries, unrecorded consumption amounts to less than 15% of the overall consumption, whereas globally, it accounts for about 28%. Unrecorded consumption per capita is highest in Africa, Central and South America, and Central and Eastern Europe. Proportionally, it is highest in Africa and in South East Asia.

CHEMICAL COMPOSITION OF DIFFERENT TYPES OF SURROGATE ALCOHOL

Moonshine From the United States

Analyses of North American illicitly distilled spirits, also known as moonshine, bootleg, white lightning, corn liquor, or hooch, were generally focused on ethanol and heavy metal contaminations. The trace element content of 12 samples of moonshine made in Georgia were analyzed by Gerhardt et al. (1980b). Four elements (arsenic, copper, lead, and zinc) were present in appreciable quantities. One sample had a potentially toxic concentration of arsenic (415 μg/l), copper was found in high concentrations up to 14 mg/l (90% of the samples were above the drinking water standard for copper). Seven samples had detectable lead concentrations in a range between 35 and 5,300 μg/l. High concentrations of zinc were found in 2 samples (2,900 and 6,350 μg/l).

A total of 48 different moonshine samples were analyzed by Holstege et al. (2004). The samples were confiscated by law enforcement agencies during raids on various stills. The samples were analyzed for ethanol, isopropanol, acetone, methanol, ethylene glycol, and lead content. Ethanol content ranged from 10.5 to 66.0%/vol with a mean value of 41.2%/vol (SD 15.9%/vol). Lead was found in measurable quantities in 43 of 48 samples with values ranging from 5 to 599 μg/l with a mean value of 81 μg/l (SD 123 μg/l). Methanol was found in only 1 sample at a level of 0.11%. No samples were found to contain measurable levels of ethylene glycol, isopropanol, or acetone.

Moonshine is typically produced in ground stills using barrels, automobile radiators, and multiple copper tube units sealed with solder as condensers. During the production of moonshine, the leaching of lead from solder or other lead-containing materials in the radiators can result in lead contamination of the moonshine. In the United States, a lead level of 15 μg/l has been established as the action level for public water supplies. Of the 48 moonshine samples mentioned above, 29 (60%) of the samples had levels equal to or exceeding this cut-point (Holstege et al., 2004). However, it should be considered that the daily consumption of water is much higher than the one of alcoholic beverages. For example, the Codex alimentarius recommends a maximum level of 200 μg/l lead in wine (Codex alimentarius, 2003). Of the analyzed moonshine, 5 (10%) samples exceeded this level. In comparison with legal spirits, the lead levels of the moonshine samples in this study do not appear to be unusual. Nascimento et al. (1999) reported a mean lead concentration of 250 μg/l (SD 120 μg/l) in a selection of international spirits (including whiskey, rum, and vodka) with a range of nondetectable concentrations up to 600 μg/l of lead. Sherry brandies contained a mean lead concentration of 58 μg/l (range: 8 to 313 μg/l) (Cameán et al., 2000) and Scottish whiskies contained a mean of 3 μg/l (range: 0 to 25 μg/l) (Adam et al., 2002).

Given these comparisons, the conclusion of Holstege et al. (2004) that moonshine might lead to serious lead toxicity cannot be derived from the presented data. However, only a small number of samples were analyzed, so that highly lead contaminated moonshine may be on the market anyway. Previous studies with a limited number of moonshine samples determined higher and toxicologically relevant lead concentrations. For example, 70,000, 760,000, and 970,000 μg/l were determined in 3 moonshine samples by Pegues et al. (1993). In a larger study conducted between 1995 and 2002 with 115 moonshine samples from 9 states, lead levels were found ranging from 0 μg/l to 53,020 μg/l with a median of 44 μg/l. Thirty-three samples contained lead levels above 300 μg/l. The median alcoholic strength of the samples was 44.8% vol (range 3.9 to 65.8% vol). No toxicologically relevant amount of methanol was identified in any sample (Morgan et al., 2004). The association between surrogate alcohol and lead poisoning is further discussed in the section Lead Poisoning Related to Surrogate Consumption.

Moonshine From Other Countries

In contrast to moonshine from the United States, the research on moonshine in Central and Eastern Europe, as well as a single study from Africa, concentrated on volatile composition (i.e., products of fermentation besides alcohol). The results are summarized in Table 2. For comparison, the limits of methanol and volatile substances according to European law are shown in Table 3.

Overall, in recent reports from Central and Eastern European countries, there was concern about toxicity of illegally produced beverages. McKee et al. (2005) concluded from a study of Russian Samogons (Russian name for illegally home-distilled alcoholic beverage) that it contains aliphatic alcohol congeners at toxicologically relevant levels.

Lang et al. (2006) went so far as to conclude that illegal products in Estonia contain "toxic long chain alcohols." In a small study (34 homemade spirits and 31 commercial spirits), Szücs et al. (2005) determined that methanol, isobutanol, 1-propanol, 2-butanol, and isoamyl alcohol concentrations in

Table 2. Chemical Composition of Illegally Produced Alcoholic Beverages in Comparison With Data From Legal Products

Alcohol type	Number of samples	Ethanol (% vol)	Methanol (mg/l)	1-Propanol (mg/l)	Isobutanol (mg/l)	Isoamyl alcohol (mg/l)	Reference
Illegal vodka (Russia)	13	32.6 to 87.7	0.95 to 6.95	0 to 2.77	(No data)	(No data)	Savchuk et al., 2006
Samogon (Russia)	11	33.8 to 47.0	(No data)	41 to 200[a]	133 to 1,600[a]	318 to 1,754[a]	McKee et al., 2005
Samogon (Russia)	80	16.5 to 62.2	0 to 655	8 to 566	19 to 3,874	36 to 4,682	Nuzhnyi, 2004
Illegal alcohols (Estonia)	9	31.9 to 56.4	(No data)	0 to 451[a]	0 to 630[a]	0 to 1,404[a]	Lang et al., 2006
Moonshine (Africa)	(No data)	21.0 to 44.0	80 to 152	39 to 75	(No data)	(No data)	Mosha et al., 1996
Moonshine fruit and marc spirits (Germany, Italy)	36	11 to 60	8 to 4,776	6 to 1,298	10 to 664	14 to 1,580	Huckenbeck et al., 2003
Moonshine Palinka (Hungary)	38	31 to 50	0 to 5,772	8 to 4,764	26 to 1,180	62 to 2,332	Huckenbeck et al., 2003
Legally manufactured vodka	29	35.3 to 40.0	0 to 64	0	0 to 6	0 to 7	Lachenmeier and Musshoff, 2004
Legally distilled fruit spirits	219	31.2 to 49.1	101 to 5,556	64 to 5,571	59 to 3,253	13 to 1,822	Lachenmeier and Musshoff, 2004

[a]Recalculated from original data in mM.

Table 3. Limits of Selected Constituents and Contaminants in Alcoholic Beverages

Compound	Limit	Reference
Methanol maximum level	Neutral alcohol[a]: 50 g/hl p.a.[b] (200 mg/l) Brandy: 200 g/hl p.a. (800 mg/l) Grape marc spirit/Grappa: 1,000 g/hl p.a. (4,000 mg/l) Fruit spirit[c]: 1,000 to 1,500 g/hl p.a. (4,000 to 6,000 mg/l)	European Council (1989)
Volatile substances/higher alcohols[d]	Neutral alcohol: <0.5 g/hl p.a. (<2 mg/l) Rum: >225 g/hl p.a. (>900 mg/l) Brandy: >125 g/hl p.a. (>500 mg/l) Grape marc spirit/Grappa: >140 g/hl p.a. (>560 mg/l) Fruit spirit: >200 g/hl p.a. (>800 mg/l)	European Council (1989)
Lead maximum level	Wine: 200 μg/l	Codex alimentarius (2003)

[a]Highly rectified alcohol (so-called "neutral alcohol" or "ethyl alcohol of agricultural origin"), which is e.g., used for the production of liqueurs, gin, aniseed-flavored spirits but also in pharmaceuticals or in denatured form in cosmetics. [b]The limits are expressed in g/hl of pure alcohol (p.a.) in the regulation. For better comparability, we have calculated the limit in mg/l for an alcoholic strength of 40%/vol. [c]The limit depends on the type of fruit. [d]It should be noted that for neutral alcohol a maximum level for higher alcohols is demanded, whereas for products like rum or brandy a minimum level for such volatile flavor substances is required.

homemade spirits were significantly higher compared with those from legal sources.

In contrast, Huckenbeck et al. (2003), Savchuk et al. (2006) and Nuzhnyi (2004) arrived at the conclusion that spirits from moonshine distillers generally have comparable volatile composition as commercial products.

It may be true that Samogon contains higher levels of aliphatic alcohols than commercial vodka because the home-producers cannot reach the degree of rectification required for vodka production. This may explain the differences obtained in Eastern Europe if homemade products are compared with commercial vodka. In contrast in Central Europe, homemade fruit spirits appear to have very similar composition to commercially made fruit spirits.

However, if the Samogons are compared with products like fruit spirits that are legally produced in the European Union, the composition of aliphatic alcohols was found to be not unusual for a product of alcoholic fermentation. It should be noted that the European law requires a minimum content of higher alcohols for most distilled beverages (because they are important flavor compounds) and no maximum content is provided (Table 3). The food policy so far assumed that the levels of higher alcohols produced during fermentation are generally safe (the toxicity of higher alcohols is further discussed in the section Influence of Higher Alcohols in Surrogate Toxicity). Under regard of the current legal limits, the Samogons with the analytical data presented in Table 2 would be marketable in the European Union.

However, an absence of data on other contaminants in the Samogons and other European moonshine was noted. Presumably, illegal fruit spirits might have problems with ethyl carbamate contamination as do products from legal small distilleries (Lachenmeier et al., 2005). It is also unknown if the lead content constitutes a problem as in some U.S. products.

Nonbeverage Alcohol From Automobile Products

Automobile products like ethylene glycol engine coolants and methanol-based windshield washer products have been described as surrogate alcohol. Obviously, the taste of both pure ethylene glycol and pure methanol was not preventing the consumption of these products. Diluted (30%) solutions of both products were deemed even more tolerable (Jackson and Payne, 1995). Accidental poisonings, especially with methanol have regularly been described (see section Methanol Poisoning). Poisonings were also described from isopropanol, which may be contained in antifreeze preparations (Chan et al., 1993). Automobile products may be rendered intolera-

ble to the human palate by addition of bittering agents like denatonium benzoate (Bitrex®) (Jackson and Payne, 1995).

Nonbeverage Alcohol From Medicinal Compounds

The quality of alcohol in medicines and medicinal compounds is regulated in pharmacopoeias (official compilations of pharmaceuticals including legal standards, issued by a regulated authority in each particular country). For example, the European pharmacopoeia defines "ethanol 96%" and "ethanolum anhydricum" (water-free ethanol). Maximum limits for different other substances including methanol, acetaldehyde, and benzol are also given (Anon, 2005). In principle, the alcohol used in medicines is of food-grade or better quality. The use of denatured alcohol in medicine is not allowed by the European pharmacopoeia.

The chemical composition of medicines used as surrogate alcohol in Russia (McKee et al., 2005) and Estonia (Lang et al., 2006) was determined. The results confirmed the very pure alcohol quality in medicines.

Therefore, the problem with consumption of medicine as surrogate alcohol appears not to be the alcohol quality, but its concentration and/or the active pharmaceutical ingredients that are consumed besides the alcohol. The dosages of the latter ingredients could be significantly higher than intended for the normal therapeutic use if the medicine is misused as surrogate alcohol.

Nonbeverage Alcohol From Cosmetics and Denatured Alcohol

Cosmetic products like hair sprays, after shaves or mouthwashes have been described to be frequently ingested as surrogate alcohol (Egbert et al., 1986, 1985; Khan et al., 1999; Sperry and Pfalzgraf, 1990). In the United States, the use of denatured alcohol in form of hairspray and spray disinfectants (called "Montana Gin") was reported to be widespread among Native Americans in the 1980s (Burd et al., 1987), and still seems to be used (Carnahan et al., 2005) particularly in reservations (Moore, 2005). So-called "denatured alcohol" is usually used as the ingredient in cosmetics. Denatured alcohol is alcohol, which has been rendered undrinkable, and in some cases dyed.

The alcohol is denatured to avoid excise duty payments required for food-grade alcohol. According to the International Nomenclature of Cosmetic Ingredients, the label "Alcohol denat." is required in the ingredients list of such products. In most countries including the United States, Canada, and the European Union such an ingredients list is mandated for every cosmetic product. Besides cosmetics, denatured alcohol may be found in a large range of technical products (e.g., fuel for camping stoves and technical solvent).

Different substances may be used for denaturing ethanol. Traditionally, the main additive was methanol so that methylated alcohol (or meths) is often synonymously used for denatured alcohol. Methanol was commonly used because it has a boiling point close to that of ethanol and cannot be separated by simple processes. Methanol was added in the form of methylene, e.g., 5 l of methylene per 100 l of ethyl alcohol. Methylene is raw methyl alcohol produced from the dry distillation of wood containing at least 10% by weight of acetone or a mixture of methylene and methanol. Other denaturing substances include methylethylketone (~1 l/hl of alcohol) or bitterants like denatonium benzoate (European Commission, 1993). Industrial alcohol is often denatured by adding methanol up to 5% (methylated), a concentration that is toxic (see section Methanol Poisoning). Besides the risk of direct use of methylated alcohol there is the potential for unintentional use of methanol or methylene as part of illegal alcohol "intended for consumption."

HEALTH CONSEQUENCES RELATED TO THE CONSUMPTION OF SURROGATE ALCOHOL

Alcohol consumption has been linked to over 60 different disease conditions, mainly as causing detrimental effects, although some patterns of drinking have been found to convey cardio-protective effects (Rehm et al., 2003). The overall net effect of alcohol consumption is detrimental, however, and alcohol has been identified as a major risk factor for global burden of disease (Rehm et al., 2004). Besides the relationship of alcohol to chronic disease, alcohol has important acute consequences on injury, including, but not restricting to alcohol poisoning (Rehm et al., 2003; Sperry and Pfalzgraf, 1990). These consequences, including the general toxicity of alcohol, will not be discussed in this study, as this section will limit itself to the specific health consequences of surrogate alcohol only. However, it should be kept in mind that part or all of the detrimental effect of surrogate alcohol may be entirely due to the effect of ethanol. In addition, given the fact that surrogates might contain higher alcohol concentrations than legal products (e.g., McKee et al., 2005; Lang et al., 2006), this may also have a detrimental effect, especially for alcohol poisoning and other injuries. Due to the lack in labeling of such products, the necessity of dilution to drinking strength might also be unknown in most cases. Most likely, many products are consumed in their original, highly alcoholic strength.

The health consequences related to the consumption of surrogate alcohol can be divided into toxicity specifically due to other compounds found in surrogate alcohol besides ethanol and other, more general, consequences associated with surrogate use (e.g., cardiovascular disease). The most common form of toxicity associated with surrogate alcohol is accidental poisoning, which can be classified into 2 groups: chronic toxicity with contaminants such as lead and acute poisonings with compounds like methanol. In fact, methanol and lead toxicity seem to make up the vast majority of toxicity from surrogate alcohol. Other toxicity can be found, but rarely; for instance, only a single instance of moonshine-related arsenic poisoning had been reported so far (Gerhardt et al., 1980a).

Table 4. Summary of Accidental Methanol Poisoning Outbreaks and Fatalities Associated With the Consumption of Surrogate Alcohol

Country	Year	Type of surrogate alcohol consumed	Methanol poisoning cases (*n*)	Methanol-related deaths (*n*; % of cases)	Reference
United States (Atlanta)	1951	Bootleg whiskey containing 35 to 40% methanol	323	41 (13%)	Bennett et al., 1953
Canada	1955	Duplicating fluid containing methanol	49	0 (0%)	Tonning, 1956
India	1967	Denatured alcohol	89	32 (36%)	Krishnamurthi et al., 1968
United States (Kentucky)	1968	Thinner (diluted to 37% vol methanol)	18	8 (44%)	Kane et al., 1968
Malaysia	1977	Alcoholic drinks of unknown comp.	20	15 (75%)	Seng, 1978
Papua New Guinea	1977	Pure methanol	28	4 (14%)	Naraqi et al., 1979
India	1988	Spirits adulterated with methanol	97	28 (29%)	Mittal et al., 1991
Brazil	1997	Cachaça blended with industrial alcohol	No data	13	Laranjerai and Dunn, 1998
Ontario	1986 to 1991	Mainly antifreeze	No data	22	Liu et al., 1999
New Zealand	1995 to 1996	Methylated spirits	24	8 (33%)	Meyer et al., 2000
United States (42 States)	1993 to 1998	Mainly windshield wiper fluids	13524[a]	74 (0.5%)[a]	Davis et al., 2002
United States (State Prison of Southern Michigan)	1979	Methanol diluents used in photocopy machines (4% mas methanol)	44	3 (7%)	Swartz et al., 1981
Turkey (Aegean Region)	1996 to 2000	Homemade beverages containing methanol	No data	44	Duman et al., 2003
Turkey (Izmir)	1993 to 2002	Mainly Eau-de-colognes	113[a]	–	Kalkan et al., 2003
Turkey (Istanbul)	1992 to 2001	Unknown	No data	271	Yayci et al., 2003
Turkey (Adana)	1997 to 2003	Homemade raki	No data	41	Gülmen et al., 2006
Turkey (Edirne)	1992 to 2003	False raki and cologne	No data	18	Azmak, 2006
Norway	2002 to 2004	Illegal product containing 20% methanol and 80% ethanol	51	17 (33%)	Hovda et al., 2005

[a]Including all cases of methanol intoxication (e.g., suicides and unintentional ingestion by children).

Methanol Poisoning

Methanol occurs naturally at a low level in most alcoholic beverages without causing harm. However, illicit drinks made from industrial methylated spirits (containing 5% of methanol) can cause severe illness or even fatalities. Assuming that an adult consumes 4 × 25 ml of a drink containing 40%/vol of alcohol over a period of 2 hours, the maximum tolerable concentration of methanol in such a drink would be 2%/vol (Paine and Dayan, 2001). The current EU limit for naturally occurring methanol in certain fruit spirits of 1,000 g/hl of pure ethanol (which equates to 0.4%/vol methanol at 40%/vol alcohol) provides a greater margin of safety (Paine and Dayan, 2001).

The first large outbreaks of methanol poisoning were documented during the Second World War in the German army. During postmortem examinations, over 100 deaths were associated with methanol-containing surrogate alcohols during that time period. For example, a mass poisoning in 1941 with methanol-containing alcohol led to 95 poisonings with severe effects and 10 deaths (Steinkamp, 2006).

Further outbreaks of methanol poisoning reported in the scientific literature since 1950 are summarized in Table 4. Recent outbreaks without specifying the number of cases were also reported in Papua New Guinea (Marshall, 1999), Mexico (Medina-Mora, 1999), India (Mohan et al., 2001; Saxena, 1999), and Brazil (Miranda et al., 1992). In outbreaks where number of cases were reported, the mortality rates ranged from 0 to 75%, however surviving patients have often been reported as having residual visual problems. Clinical manifestations, diagnosis, and treatment of methanol poisoning were reviewed by Kruse (1992). In spite of improvements in treatment over the past decades, methanol poisoning still has a high mortality, mainly because of delayed admission to hospital and late diagnosis (Hovda et al., 2005).

An interesting methanol epidemic occurred in the State Prison of Southern Michigan, where several inmates obtained a quantity of nearly pure methanol diluents ordinarily used in photocopy machines, and distributed this fluid in small quantities as "homemade" spirits. One specimen, retrieved from an inmate cell revealed a pink fruity liquid with at least 4% methanol by weight. The relatively low incidence of fatalities may be explained by the early recognition of methanol poisoning and prompt institution of a treatment program (Swartz et al., 1981). In Brazil, 13 deaths occurred after consumption of cachaça contanining 17% of methanol. The methanol contamination was due to mixing of stolen industrial alcohol with sugarcane spirits from clandestine distilleries to produce a low quality and extremely cheap form of cachaça (Laranjerai and Dunn, 1998). In Ontario, Canada, 3 major factors for methanol-related deaths were identified: (1) Consumption of methanol- or wood alcohol-labeled products as ethanol substitutes (64%); (2) Illicit sources of alcohol (23%); and (3) Improper storage of methanol in spirit bottles

(13%). The higher incidence of methanol-related deaths in Ontario compared with the United States was speculated to be related to the higher costs of alcoholic beverages in Canada compared with the United States (Liu et al., 1999).

In New Zealand, the abuse of methylated spirits, which contain 5% methanol and between 70 and 90% ethanol, was described to be commonplace. The reported deaths were mainly attributed to binge drinking of methylated spirits (Meyer et al., 2000). In the United States, from 13,524 cases associated with methanol poisoning in the time period between 1993 and 1998, 967 cases were reported having methanol poisoning with moderate effect, major effect, or death. Methanol products were recorded, showing windshield wiper fluids to be 61% of exposures (Davis et al., 2002). However, the study showed no clear distinction between cases of surrogate alcohol use and other accidental methanol intoxications (e.g., in children).

In Turkey, alcoholics with low socioeconomic status consume homemade alcoholic beverages and fatalities may occur due to substitution of methanol for ethanol in those beverages. Between 1996 and 2003, 44 fatalities were reported in the Aegean region of Turkey (Duman et al., 2003). In nonfatal methanol poisonings in Turkey, cheap eau-de-colognes were reported to be the main source of methanol (Kalkan et al., 2003). While in other countries, only several large outbreaks were reported, in Turkey the cases were generally unconnected and appear to be relatively constant over the years (Yayci et al., 2003). In the Adana region of Turkey, similar problems arise from home-produced raki from grapes, figs, or plums. Although the production is illegal, villagers generally use wooden materials and reed pipes during the distillation process meaning that methanol is produced by the equipment accidentally. The villagers do not generally have any intention of selling this product or causing harm to anyone, yet it does cause serious intoxications: 17 deaths were causally related to the consumption of illegal raki (Gülmen et al., 2006). In contrast, other reports from Turkey found that methanol levels were low in illegally produced raki in Turkey and comparable with that produced under the governmental monopoly (Fidan et al., 1996). An explanation might be locally different production conditions in the regions of Turkey.

When examining the scientific literature reported above and summarized in Table 4, one should keep in mind that the scientific literature covers only some of the outbreaks. Many others are only reported in the newspaper and other media. A search on March 3, 2007, using the key words "methanol," "poisoning," and "outbreak" revealed 38,400 hits using Google search engine.

Lead Poisoning Related to Surrogate Consumption

Lead exposure associated with moonshine consumption in the United States is well documented (Table 5). One investigation identified 128 adult deaths linked to lead toxicity in the United States between 1979 and 1988. Of the fatal adult cases, moonshine was the cause in 20 of the 25 patients for whom the source of lead was identified (Staes et al., 1995). In an extended time period (1979 to 1998), a trend toward decreasing death rate was detected that might be related to either safer stills or decreased use of moonshine (Kaufmann et al., 2003).

Findings by Ellis and Lacy (1998) suggested that nonfatal lead intoxication associated with moonshine consumption in west Alabama has declined (2.3% of cases in 1989 to 1992, compared with 9.2% of cases in 1979 to 1982). A reason might be the destruction of illegal stills in Alabama, e.g., 94 stills were destroyed in 1991 (Anon, 1992). Modern stills were purported to be built better than stills in the past, so that today's moonshine was found to be free of contaminants (Holstege et al., 2004). It was speculated that copper tubing was replacing automobile radiators in the construction of stills (Gerhardt et al., 1980b). However, Morgan et al. (2001, 2003) reported that the days of lead toxicity and moonshine are not over because elevated blood lead levels were still found in moonshine drinkers.

Influence of Higher Alcohols in Surrogate Toxicity

Alcohols with more than 2 carbon atoms are commonly called higher or fusel alcohols (sometimes volatiles in alcoholic beverages besides ethanol are also called congeners). Most higher alcohols occur as by-products of yeast fermentation and are important flavor compounds. For example, they

Table 5. Summary of Lead Intoxications Associated With the Consumption of Surrogate Alcohol

Country	Year	Type of surrogate alcohol consumed	Total Lead intoxication cases	Cases associated with surrogate alcohol	Reference
West Alabama	1989 to 1992	Moonshine	224	5 (2%)	Ellis and Lacy, 1998
Alabama	1990 to 1991	Moonshine	(no data)	8	Anon, 1992
Noth Carolina	1983	Moonshine	(no data)	10	Reynolds et al., 1983
Atlanta, Georgia	2000	Moonshine	(no data)	4	Morgan et al., 2003
Alabama	1991	Moonshine	9	9 (100%)	Pegues et al., 1993
United States	1979 to 1998	Moonshine	200[a]	28% Alcohol related[b]	Kaufmann et al., 2003
United States	1979 to 1988	Moonshine	139[a]	20[a] (14%)	Staes et al., 1995

[a]Lead poisoning related deaths. [b]No data about cases causally related to moonshine were given.

commonly account for about 50% of the aromatic constituents of wine, excluding ethanol. Quantitatively, the most important higher alcohols are the straight-chain alcohols 1-propanol, 2-methyl-1-propanol (isobutyl alcohol), 2-methyl-1-butanol, and 3-methyl-1-butanol (isoamyl alcohol) (Jackson, 2000). The content of higher alcohols in alcoholic beverages is generally not seen as of toxicological relevance. For example, the Joint FAO/WHO Expert Committee on Food Additives included higher alcohols (1-propanol, isobutyl alcohol, 1-butanol, and isobutanol) in the functional class "flavoring agent" and commented that there was no safety concern at current levels of intake when used as flavoring agent (JECFA, 1997). For certain groups of spirits, the European Union even demands minimum volatile substance content (i.e., the quantity of volatile substances other than ethanol and methanol, which are mainly higher alcohols). For example, fruit spirits must have at least a content of volatile substances of 200 g/hl of pure ethanol (see Table 3) (European Council, 1989).

Higher alcohols are found in both legal alcoholic beverages and surrogate alcohols (Table 2). Some authors attributed a possible higher toxicity of surrogates to their content of higher alcohols. For example, compared with consumers of mainly licit alcohol, higher rates of alcoholic liver disease among consumers of homemade "country liquor" have been reported in India (Narawane et al., 1998), and an animal study on rats suggests that "toddy" (an Indian country liquor) had an increased toxicity compared with the same dose of pure ethanol (Lal et al., 2001). Aliphatic alcohols and other hepatotoxic substances have also been found in Brazilian rhum (Mincis et al., 1993) and in Tanzanian beverages (Nikander et al., 1991).

So far, it is unclear if the relatively low contents of higher alcohols in combination with high concentrations of ethanol have a consequence on the etiology of surrogate-derived diseases. Only limited and contradictory information about the toxicity of higher alcohols was found in the literature. Gibel et al. (1969) reported severe hepatic damage occurring in rats treated with high doses of corn fusel oil-containing aldehydes, esters, and a large number of higher alcohols. Peneda et al. (1994) confirmed those results and suggested that the hepatotoxicity of ethanol may be enhanced by interaction with its congeners and acetaldehyde; they also suggested that alcoholic beverages are not equivalent in their potential to cause liver damage.

In contrast, Siegers et al. (1974) administered 4 alcoholic congeners orally to guinea pigs at doses up to 100-fold higher than those which can be expected at the most by human binge drinking and detected no hepatotoxic activity. The experiments of Hillbom et al. (1974), feeding rats with 1 M solutions of ethanol, *n*-propanol, or 2-methyl-1-propanol over 4 months also failed to produce a hepatotoxic response. The no-effect level of isoamyl alcohol in rats was determined to be 1,000 mg/kg/d, a level estimated to be 350 to 400 times the maximum likely intake in man (Carpanini et al., 1973).

Hepatotoxicity may be assessed by assaying liver cytosol-derived enzymes such as lactate dehydrogenase (LDH), glutamate-pyruvate-transaminase (GPT), or glutamate dehydrogenase (GLDH). McKarns et al. (1997) evaluated the release of LDH by rat liver epithelial cells in vitro after acute exposure to 11 short-chain alcohols and found a correlation between the hydrophobicity of these alcohols and their ability to alter plasma membrane integrity. Strubelt et al. (1999) studied 23 aliphatic alcohols in the isolated, perfused rat liver. The capacity of the straight chain primary alcohols (methanol, ethanol, 1-propanol, 1-butanol, and 1-pentanol) to release GPT, LDH, and GLDH into the perfusate was strongly correlated with their carbon chain length. The secondary alcohols (2-propanol, 2-butanol, 2-pentanol, and 3-pentanol) were less active in this respect, whereas branching of the carbon chain (2-methyl-1-butanol and 3-methyl-1-butanol) did not consistently change alcohol toxicity. Alcohol-induced hepatotoxicity was primarily due to membrane damage induced by the direct solvent properties of the alcohols. Strubelt et al. (1999) concluded that the consequences and relative contributions of alcohol metabolization to the overall hepatotoxicity of higher alcohols required further study.

In consideration of the sparse toxicological data of higher alcohols, it appears to be impossible to evaluate their potential in the hepatotoxicity of surrogate alcohol.

Other Health Consequences of Surrogate Consumption

Overall, literature on health consequences of surrogate consumption is limited. To our knowledge, only the above cited population-based case-control study of Leon et al. (2007) gives estimates on population bases rather than reporting the number of affected cases of an outbreak. The basis for this study were all deaths in men between October 2003 and October 2005 in the Russian town of Izhevsk in the age groups of 25 to 54. Proxy interviews were used to assess exposure. Leon et al. (2007) found that 42% of the deceased and 8% of the controls consumed surrogate alcohol in the past year. The authors found an age-adjusted odds ratio (OR) of 9.2 (95% confidence interval: 7.2 to 11.7) that was attenuated slightly by adjustment for volume of alcohol consumed (OR: 8.3; 95% confidence interval: 6.5 to 10.7). The magnitude of consumption happened in people with lower socioeconomic status and further adjustment for education and smoking reduced the OR to 7.0 (95% confidence interval: 5.5 to 9.0). Based on the latter OR, the population attributable fraction based on the exposure in controls can be calculated as 31.9%; i.e., 32% of all the deaths in this age group for men would disappear if surrogate consumption was to be removed without any substitution by other alcohol. This clearly indicates a potential public health importance of surrogate consumption, which goes far beyond the poisonings of methanol and lead described above. Surrogate seems to be linked to many causes of death, including cardiovascular disease, liver disease, and infectious disease mortality.

DISCUSSION AND CONCLUSIONS

Surrogate alcohol use was shown to be a potential threat to public health. Two pathways have been identified: first, components other than ethanol in surrogate alcohol may lead to poisoning. In this respect, methanol poisoning and lead poisoning outbreaks have been documented in the recent past. Secondly, several health effects over and above those of ethanol ingestion including organ damage have been identified with the consumption of methanol, e.g., effects on the central nervous system, liver, retinal, and renal damage. High lead blood levels through illicitly produced moonshine have also been linked with damages of the central nervous system, the peripheral nervous system, the hematopoietic system, the renal system, and the gastrointestinal system. Long-chain aliphatic alcohols contained in products not intentionally produced for consumption (e.g., antifreeze) but also in homemade products intended as drink alcohol have been linked with a higher hepatotoxicity. However, the occurrence and severity of detrimental health outcomes clearly depends on the concentration of these substances. The Russian case-control study of Leon et al. (2007) showed a strong link between use of surrogate alcohols and all-cause mortality in men. Unfortunately, the exact pathways underlying this link are far from clear, but ethanol itself is strongly related to different causes of mortality (Rehm et al., 2004; see below).

There are a number of general limitations in the study by Leon et al. (2007) that need addressing. First, only the frequency of surrogate alcohol drinking, and not the ethanol content of surrogate alcohol was measured. Thus, it was not clear whether the higher mortality was just due to higher intake of ethanol among those consuming surrogate alcohols. Secondly, because the surrogate alcohols could not be analyzed, it is unknown whether toxic agents other than ethanol were responsible for the higher mortality. Thirdly, there might be some residual confounding due to other life circumstances. Surrogate drinkers are often at the margins of the society, where poorer housing, less healthy diets, etc. might be responsible for a higher mortality. For example, proxy information used to estimate ORs for cases was less often obtained from wives and partners compared with controls, pointing to less social support or less stable living situations. Taking into account only proxy reports from wives and partners reduced ORs for surrogate drinking. Given the high public health importance of this findings, research on these pathways should be undertaken with priority. Also, the findings of Leon et al. (2007) should be replicated in other jurisdictions with high proportion of surrogate alcohol consumption.

However, some interventions to reduce the harm resulting from surrogate alcohol could be undertaken already at this point. Meyer et al. (2000) judged the complete removal of methanol from denatured spirits to be the most significant measure to reduce methanol-attributable morbidity and mortality. Other denaturing agents such as denatonium benzoate are available and there is no need for the use of methanol in denatured alcohol (Meyer et al., 2000). Some countries, including Australia, have abolished the use of methanol to denature alcohol, limiting the availability of this substance for abuse, with a subsequent significant reduction in cases of toxicity (Meyer et al., 2000). Many European countries also do not allow methanol (or methanol-containing wood alcohol) to be used as denaturing agent (European Commission, 1993). Today, methanol is generally judged as unsuitable for denaturing alcohol: methanol cannot be distinguished by taste from ethanol and the use appears to be unsafe from a toxicological standpoint. According to Savchuk et al. (2006), diethyl phthalate also appears to be unsuitable as denaturation agent as it has no effect on the organoleptic properties of ethanol and can be separated by distillation. Nowadays, other substances such as bittering agents appear to be the denaturing agents of choice: only low amounts are necessary to make alcohol undrinkable. For cosmetics, the most elegant way is to use the perfume oils that are part of the recipe anyway as denaturing agent. Thus, methanol should be prohibited globally as a means of denaturation. Other surrogate alcohols e.g., for automobile products, could also be treated with bittering agents to avoid consumption.

In addition to measures on the supply side, research is necessary to better understand the demand side of surrogate alcohol consumption in order to develop preventive programs. Clearly, lower price per unit of pure ethanol is a strong reason why people use surrogate alcohols. But many of the users of surrogate alcohol also consume other forms of alcohol (e.g., Leon et al., 2007). Under which circumstances are different forms of alcohol purchased? Are surrogate alcohols only purchased, when there are no more resources for more expensive other forms of alcohol? Currently, we know little about the reasons and circumstances for obtaining surrogate alcohols beyond the fact, that they are less expensive. What role does alcohol dependence play in the purchasing decision? For example, it could be the case that tolerance and the need for higher quantities of alcohol per day, in some cases coupled with less available resources, may lead specifically to the purchase of surrogate alcohols.

Surrogate alcohol comprises very many different products. Medicinal alcohol is commonly pure ethanol and its detrimental effects are thus due to alcohol poisoning also related to a lack of diluting it to drinking strength. Rigorous control of selling of medicinal alcohol and the selling of only small container sizes have been shown to reduce potential harm from medicinal alcohols to a marginal problem in the Nordic countries (Nordlund and Österberg, 2000). As shown in a recent report of the International Center for Alcohol Policies homemade (moonshine) products are not always illegal and are often deeply rooted in the culture (Haworth and Simpson, 2004). In other countries such as in the Eastern Mediterranean region, where alcohol is prohibited on religious grounds, most of the available beverage alcohol is illegally (home) produced (WHO, 2006). Conclusions from a WHO report (2006) actually question even more rigid alcohol control policies because of the need to counterbalance them against even more harmful consumption patterns or consumption of more

harmful substances. Given the fact that people are ready to drink even nonbeverage alcohol, often in regions where commercially produced alcohol is difficult to obtain, such as in Indian U.S. reservations, one may wonder whether harm reduction strategies, which may even relax availability restrictions of beverage alcohol, may be more promising. Thailand for example has legalized homemade spirits recently (Assunta, 2001). Other harm reduction measures may be, comparable with early drug warning systems, the testing of illegally produced but available products (such as counterfeit vodkas) and the information of consumers about its potential toxicities. According to an unknown referee of an earlier version of this paper, local Public Health Departments (Sanitary Epidemiological Service) conduct a large number of tests of alcoholic beverages in the Siberian Region of the Russian Federation. However, we could not identify any scientific article on the potential or effectiveness of these departments for informing the public. Given the scale of the problems associated with the consumption of counterfeit and poor quality vodkas in Russia, all the available time and resources of these departments may have been used in testing and related consequences, so that preventive activities may not have been feasible yet.

Given the current lack on research it is difficult to recommend preventive actions. A major recommendation, as for example outlined in the resolution EM/RC52/R.5 (WHO, 2005) of Regional Committee for the Eastern Mediterranean is the establishing of an information system that fosters the evidence base on alcohol consumption including surrogate alcohol consumption. It is clear that there cannot be any simple generalization of preventive measures that meets the differences in cultural and religious norms. In many countries of the world, surrogate alcohol only plays a minor role and prevention should be focused on prevention of harms due to ethanol consumption in general. In other parts of the world, however, surrogate alcohol consumption may create additional problems over and above that of ethanol consumption alone and needs to be addressed by other strategies than e.g., taxation, which have shown to be effective but cannot address consequences related to alcohol that is simply missed by the taxation system. Preventive measures for reducing the harm attributable to the use of surrogate alcohols can only be conceptualized if we develop better insight into the phenomenon, i.e., if we can answer the question of who will buy surrogate alcohols under what circumstances for what reason.

ACKNOWLEDGMENT

The authors thank Benjamin Taylor for his excellent language editing of the manuscript.

REFERENCES

Adam T, Duthie E, Feldmann J (2002) Investigations into the use of copper and other metals as indicators for the authenticity of Scotch whiskies. J Inst Brew 108:459–464.

Anon (1992) Elevated blood lead levels associated with illicitly distilled alcohol—Alabama, 1990-1991. MMWR Morb Mortal Wkly Rep 41:294–295.

Anon (2005) Ethanol 96%. Eur Pharma 5:2099–2104.

Assunta M (2001) Impact of alcohol consumption on Asia. The Globe 2001(Spec. Issue):4–8. Available at: http://www.ias.org.uk/resources/publications/theglobe/globe200103-04/gl200103-04_p4.html. Accessed July 16, 2007.

Azmak D (2006) Methanol related deaths in Edirne. Leg Med (Tokyo) 8: 39–42.

Bennett IL Jr, Cary FH, Mitchell GL Jr, Cooper MN (1953) Acute methyl alcohol poisoning: a review based on experiences in an outbreak of 323 cases. Medicine (Baltimore) 32:431–463.

Burd L, Shea TE, Knull H (1987) "Montana gin": ingestion of commercial products containing denatured alcohol among native Americans. J Stud Alcohol 48:388–389.

Cameán AM, Moreno IM, López-Artíguez M, Repetto M, González AG (2000) Metallic profiles of sherry brandies. Sci Aliments 20:433–440.

Carnahan RM, Kutscher EC, Obritsch MD, Rasmussen LD (2005) Acute ethanol intoxication after consumption of hairspray. Pharmacotherapy 25:1646–1650.

Carpanini FM, Gaunt IF, Kiss IS, Grasso P, Gangolli SD (1973) Short-term toxicity of isoamyl alcohol in rats. Food Cosmet Toxicol 11:713–724.

Chan KM, Wong ET, Matthews WS (1993) Severe isopropanolemia without acetonemia or clinical manifestations of isopropanol intoxication. Clin Chem 39:1922–1925.

Codex alimentarius (2003) Maximum levels for lead (CODEX STAN 230-2001, Rev. 1-2003). Available at: http://www.codexalimentarius.net. Accessed July 16, 2007.

Davis LE, Hudson D, Benson BE, Jones Easom LA, Coleman JK (2002) Methanol poisoning exposures in the United States: 1993-1998. J Toxicol Clin Toxicol 40:499–505.

Duman E, Akgür SA, Öztürk P, Sen F (2003) Fatal poisonings in the Aegean region of Turkey. Vet Hum Toxicol 45:106–108.

Egbert AM, Liese BA, Powell BJ, Reed JS, Liskow BI (1986) When alcoholics drink aftershave: a study of nonbeverage alcohol consumers. Alcohol Alcohol 21:285–294.

Egbert AM, Reed JS, Powell BJ, Liskow BI, Liese BS (1985) Alcoholics who drink mouthwash: the spectrum of nonbeverage alcohol use. J Stud Alcohol 46:473–481.

Ellis T, Lacy R (1998) Illicit alcohol (moonshine) consumption in West Alabama revisited. South Med J 91:858–860.

European Commission (1993) Commission Regulation (EC) No 3199/93 of 22 November 1993 on the mutual recognition of procedures for the complete denaturing of alcohol for the purposes of exemption from excise duty. Off J Europ Comm L288:12–15.

European Council (1989) Council Regulation (EEC) No 1576/89 laying down general rules on the definition, description and presentation of spirit drinks. Off J Europ Comm L160:1–17.

Fidan I, Denli Y, Anli RE (1996) A research on the methanol content of raki produced in Turkey. Gida 21:415–418.

Gerhardt RE, Crecelius EA, Hudson JB (1980a) Moonshine-related arsenic poisoning. Arch Intern Med 140:211–213.

Gerhardt RE, Crecelius EA, Hudson JB (1980b) Trace element content of moonshine. Arch Environ Health 35:332–334.

Gibel W, Wildner GP, Lohs K (1969) Experimental studies on hepatotoxic effect of higher alcohols (fusel oil). Z Gastroenterologie 7:108–113.

Gülmen MK, Meral D, Hilal A, Akcan R, Çekin N (2006) Methanol Intoxications in Adana, Turkey. Toxicol Mech Methods 16:353–357.

Hanley JA (2001) A heuristic approach to the formulas for population attributable fraction. J Epidemiol Community Health 55:508–514.

Haworth A, Simpson R (2004) Moonshine Markets. Issues in Unrecorded Alcohol Beverage Production and Consumption. Brunner-Routledge, New York.

Hillbom ME, Franssila K, Forsander OA (1974) Effects of chronic ingestion of some lower aliphatic alcohols in rats. Res Commun Chem Pathol Pharmacol 9:177–180.

Holstege CP, Ferguson JD, Wolf CE, Baer AB, Poklis A (2004) Analysis of moonshine for contaminants. J Toxicol Clin Toxicol 42:597–601.

Hovda KE, Hunderi OH, Tafjord AB, Dunlop O, Rudberg N, Jacobsen D (2005) Methanol outbreak in Norway 2002-2004: epidemiology, clinical features and prognostic signs. J Intern Med 258:181–190.

Huckenbeck W, Freudenstein P, Jeszenszky E, Scheil HG (2003) Congeners in spirits produced by moonshine distillers. Blutalkohol 40:294–301.

Jackson RS (2000) Wine Science: Principles, Practice, Perception. 2nd ed. Academic Press, San Diego, CA.

Jackson MH, Payne HA (1995) Bittering agents: their potential application in reducing ingestions of engine coolants and windshield wash. Vet Hum Toxicol 37:323–326.

JECFA (1997) Summary of Evaluations Performed by the Joint FAO/-WHO Expert. Committee on Food Additives, World Health Organization, Geneva.

Kalkan S, Cevik AA, Cavdar C, Aygoren O, Akgun A, Ergun N, Tuncok Y (2003) Acute methanol poisonings reported to the Drug and Poison Information Center in Izmir, Turkey. Vet Hum Toxicol 45:334–337.

Kane RL, Talbert W, Harlan J, Sizemore G, Cataland S (1968) A methanol poisoning outbreak in Kentucky. A clinical epidemiologic study. Arch Environ Health 17:119–129.

Kaufmann RB, Staes CJ, Matte TD (2003) Deaths related to lead poisoning in the United States, 1979-1998. Environ Res 91:78–84.

Khan F, Alagappan K, Cardell K (1999) Overlooked sources of ethanol. J Emerg Med 17:985–988.

Krishnamurthi MV, Natarajan AR, Shanmugasundaram K, Padmanabhan K, Nityanandan K (1968) Acute methyl alcohol poisoning. (A review of an outbreak of 89 cases). J Assoc Physicians India 16:801–805.

Kruse JA (1992) Methanol poisoning. Intensive Care Med 18:391–397.

Lachenmeier DW, Musshoff F (2004) Volatile congeners in alcoholic beverages. Retrospective trends, batch comparisons and current concentration ranges. Rechtsmed 14:454–462.

Lachenmeier DW, Schehl B, Kuballa T, Frank W, Senn T (2005) Retrospective trends and current status of ethyl carbamate in German stone-fruit spirits. Food Addit Contam 22:397–405.

Lal J, Kumar CV, Suresh MV, Indira M, Vijayammal PL (2001) Effect of exposure to a country liquor (Toddy) during gestation on lipid metabolism in rats. Plant Foods Hum Nutr 56:133–143.

Lang K, Vali M, Szücs S, Adany R, McKee M (2006) The composition of surrogate and illegal alcohol products in Estonia. Alcohol Alcohol 41:446–450.

Laranjerai R, Dunn J (1998) Death by methanol poisoning in Brazil. Addiction 93:1103–1104.

Leon DA, Saburova L, Tomkins S, Andreev E, Kiyanov N, McKee M, Shkolnikov VM (2007) Hazardous alcohol drinking and premature mortality in Russia: a population based case-control study. Lancet 369:2001–2009.

Liu JJ, Daya MR, Mann NC (1999) Methanol-related deaths in Ontario. J Toxicol Clin Toxicol 37:69–73.

Marshall M (1999) Country profile on alcohol in Papua New Guinea, in Alcohol and Public Health in 8 Developing Countries (Riley L, Marshall M eds), pp 115–133. World Health Organization, Geneva.

McKarns SC, Hansch C, Caldwell WS, Morgan WT, Moore SK, Doolittle DJ (1997) Correlation between hydrophobicity of short-chain aliphatic alcohols and their ability to alter plasma membrane integrity. Fundam Appl Toxicol 36:62–70.

McKee M, Suzcs S, Sarvary A, Adany R, Kiryanov N, Saburova L, Tomkins S, Andreev E, Leon DA (2005) The composition of surrogate alcohols consumed in Russia. Alcohol Clin Exp Res 29:1884–1888.

Medina-Mora ME (1999) Country profile on alcohol in Mexico, in Alcohol and Public Health in 8 Developing Countries (Riley L, Marshall M eds), pp 75–94. World Health Organization, Geneva.

Meyer RJ, Beard ME, Ardagh MW, Henderson S (2000) Methanol poisoning. N Z Med J 113:11–13.

Mincis M, Mizuta K, Bulhoes E, Andriolo M, Khouri S, Doin P (1993) Hepatotoxic substances in Brazilian rum. Rev Assoc Med Bras 39:234–236.

Miranda MP, Dantas VP, Diez del Corral FS (1992) Ethanol and methanol contents in spirits in Bahia State, Brazil. Boletim da Sociedade Brasileira de Ciencia e Tecnologia de Alimentos 26:104–107.

Mittal BV, Desai AP, Khade KR (1991) Methyl alcohol poisoning: an autopsy study of 28 cases. J Postgrad Med 37:9–13.

Mohan D, Chopra A, Ray R, Sethi H (2001) India, in Surveys of Drinking Patterns and Problems in Seven Developing Countries (Demers A, Room R, Bourgault C eds), pp 103–114. World Health Organization, Geneva.

Moore RS (2005) "Ocean" in the desert: denatured alcohol consumption in a "dry" southwestern reservation. Alcohol Clin Exp Res 29:82A.

Morgan BW, Barnes L, Parramore CS, Kaufmann RB (2003) Elevated blood lead levels associated with the consumption of moonshine among emergency department patients in Atlanta, Georgia. Ann Emerg Med 42:351–358.

Morgan BW, Parramore CS, Ethridge M (2004) Lead contaminated moonshine: a report of bureau of alcohol, tobacco and firearms analyzed samples. Vet Hum Toxicol 46:89–90.

Morgan BW, Todd KH, Moore B (2001) Elevated blood lead levels in urban moonshine drinkers. Ann Emerg Med 37:51–54.

Mosha D, Wangabo J, Mhinzi G (1996) African traditional brews: how safe are they? Food Chem 57:205–209.

Naraqi S, Dethlefs RF, Slobodniuk RA, Sairere JS (1979) An outbreak of acute methyl alcohol intoxication. Aust N Z J Med 9:65–68.

Narawane NM, Bhatia S, Abraham P, Sanghani S, Sawant SS (1998) Consumption of 'country liquor' and its relation to alcoholic liver disease in Mumbai. J Assoc Physicians India 46:510–513.

Nascimento RF, Bezerra C-WB, Furuya S-MB, Schultz MS, Polastro LR, Lima-Neto BS, Franco DW (1999) Mineral profile of Brazilian cachacas and other international spirits. J Food Comp Anal 12:17–25.

Nikander P, Seppala T, Kilonzo GP, Huttunen P, Saarinen L, Kilima E, Pitkanen T (1991) Ingredients and contaminants of traditional alcoholic beverages in Tanzania. Trans R Soc Trop Med Hyg 85:133–135.

Nordlund S, Österberg E (2000) Unrecorded alcohol consumption: its economics and its effects on alcohol control in the Nordic countries. Addiction 95:S551–S564.

Nuzhnyi V (2004) Chemical composition, toxic, and organoleptic properties of noncommercial alcohol samples, in Moonshine Markets. Issues in Unrecorded Alcohol Beverage Production and Consumption (Haworth A, Simpson R eds), pp 177–199. Brunner-Routledge, New York.

Paine AJ, Dayan AD (2001) Defining a tolerable concentration of methanol in alcoholic drinks. Hum Exp Toxicol 20:563–568.

Pegues DA, Hughes BJ, Woernle CH (1993) Elevated blood lead levels associated with illegally distilled alcohol. Arch Intern Med 153:1501–1504.

Peneda J, Baptista A, Lopes JM (1994) Interaction of the constituents of alcoholic beverages in the promotion of liver damage. Acta Med Port 7(Suppl 1):S51–S55.

Rehm J, Room R, Graham K, Monteiro M, Gmel G, Sempos CT (2003) The relationship of average volume of alcohol consumption and patterns of drinking to burden of disease: an overview. Addiction 98:1209–1228.

Rehm J, Room R, Monteiro M, Gmel G, Graham K, Rehn N, Sempos CT, Frick U, Jernigan D (2004) Alcohol use, in Comparative Quantification of Health Risks. Global and Regional Burden of Disease Attributable to Selected Major Risk Factors, *Vol. 1* (Ezzati M, Lopez AD, Rodgers A, Murray CJL eds), pp 959–1108. World Health Organization, Geneva.

Reynolds PP, Knapp MJ, Baraf HS, Holmes EW (1983) Moonshine and lead. Relationship to the pathogenesis of hyperuricemia in gout. Arthritis Rheum 26:1057–1064.

Savchuk SA, Nuzhnyi VP, Kolesov GM (2006) Factors affecting the accuracy of the determination of diethyl phthalate in vodka, ethanol, and samples of illegal alcoholic products. J Anal Chem 61:1198–1203.

Saxena S (1999) Country profile on alcohol in India, in Alcohol and Public Health in 8 Developing Countries (Riley L, Marshall M eds), pp 37–60. World Health Organization, Geneva.

Seng NT (1978) Methyl alcohol poisoning: a report of 20 cases. Med J Malaysia 33:13–16.

Siegers CP, Strubelt O, Breining H (1974) The acute hepatotoxic activity of alcoholic beverages and some of their congeners in guinea pigs. Pharmacology 12:296–302.

Sperry K, Pfalzgraf R (1990) Fatal ethanol intoxication from household products not intended for ingestion. J Forensic Sci 35:1138–1142.

Staes C, Matte T, Staeling N, Rosenblum L, Binder S (1995) Lead poisoning deaths in the United States, 1979 through 1988. JAMA 273:847–848.

Steinkamp P (2006) Methyl alcohol poisoning in the German Armed Forces during the Second World War. Schweiz Rundsch Med Prax 95:1997–1999.

Strubelt O, Deters M, Pentz R, Siegers CP, Younes M (1999) The toxic and metabolic effects of 23 aliphatic alcohols in the isolated perfused rat liver. Toxicol Sci 49:133–142.

Swartz RD, Millman RP, Billi JE, Bondar NP, Migdal SD, Simonian SK, Monforte JR, McDonald FD, Harness JK, Cole KL (1981) Epidemic methanol poisoning: clinical and biochemical analysis of a recent episode. Medicine (Baltimore) 60:373–382.

Szücs S, Sárváry A, McKee M, Ádány R (2005) Could the high level of cirrhosis in central and eastern Europe be due partly to the quality of alcohol consumed? An exploratory investigation Addiction 100:536–542.

Tonning DJ, Brooks DW, Harlow CM (1956) Acute methyl alcohol poisoning in 49 naval ratings. Can Med Assoc J 74:20–27.

WHO (2005) Resolution EM/RC52/R.5. World Health Organization, Regional Committee for the Eastern Mediterranean. Available at: http://www.emro.who.int/rc52/media/pdf/EMRC52R5.pdf. Accessed July 16, 2007.

WHO (2006) Technical paper on public health problems of alcohol consumption in the region (EM/RC53/4). World Health Organization, Regional Committee for the Eastern Mediterranean. Available at: http://www.emro.who.int/rc53/media/pdf/EMRC5304En.pdf. Accessed July 16, 2007.

Yayci N, Agritmis H, Turla A, Koc S (2003) Fatalities due to methyl alcohol intoxication in Turkey: an 8-year study. Forensic Sci Int 131:36–41.