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Comparison of Blood-Ethanol Concentration in Deaths Attributed to Acute Alcohol Poisoning and Chronic Alcoholism

ABSTRACT: Ethanol concentrations were measured in femoral venous blood in deaths attributed to acute alcohol poisoning ($N = 693$) or chronic alcoholism ($N = 825$), according to the forensic pathology report. Among acute alcohol poisonings were 529 men (76%) with mean age 53 years and 164 women (24%) with mean age 53 years. In the chronic alcoholism deaths were 705 men (85%) with mean age 55 years and 120 women (15%) with mean age 57 years. The blood-ethanol concentrations were not related to the person's age ($r = -0.17$ in acute poisonings and $r = -0.09$ in chronic alcoholism). The distribution of blood-ethanol concentrations in acute poisoning cases agreed with a normal or Gaussian curve with mean, median, standard deviation, coefficient of variation, and spread of 0.36 g/100 mL, 0.36 g/100 mL, 0.086 g/100 mL, 24% and 0.074 to 0.68 g/100 mL, respectively. The corresponding concentrations of ethanol in chronic alcoholism deaths were not normally distributed and showed a mode between 0.01 and 0.05 g/100 mL and mean, median, and spread of 0.172 g/100 mL, 0.150 g/100 mL, and 0.01 to 0.56 g/100 mL, respectively. The 5th and 95th percentiles for blood-ethanol concentration in acute poisoning deaths were 0.22 and 0.50 g/100 mL, respectively. However, these values are probably conservative estimates of the highest blood-ethanol concentrations before death owing to metabolism of ethanol until the time of death. In 98 chronic alcoholism deaths (12%) there was an elevated concentration of acetone in the blood (>0.01 g/100 mL), and 50 of these (6%) also had elevated isopropanol (>0.01 g/100 mL). This compares with 28 cases (4%) with elevated blood-acetone in the acute poisoning deaths and 22 (3%) with elevated blood-isopropanol. We offer various explanations for the differences in blood-ethanol and blood-acetone in acute poisoning and alcoholism deaths such as chronic tolerance, alcohol-related organ and tissue damage (cirrhosis, pancreatitis), positional asphyxia or suffocation by inhalation of vomit, exposure to cold coupled with alcohol-induced hypothermia, as well as various metabolic disturbances such as hypoglycemia and ketoacidosis.

KEYWORDS: forensic science, alcohol, alcoholism, ethanol poisoning deaths, toxicity

Opinions differ about the blood-ethanol concentration necessary to cause death. Different authorities cite different fatal blood-ethanol concentrations, such as 0.35 to 0.40 g/100 mL (1), 0.40 g/100 mL (2), range 0.225 to 0.40 g/100 mL (3), and >0.35 g/100 mL (4). These figures probably reflect the personal experiences of the pathologist or toxicologist concerned, and the sample sizes are often fairly limited and reference ranges of blood-ethanol are rarely reported. Moreover, the quality assurance procedures used for sampling blood and for determination of ethanol are not always made perfectly clear, which also complicates interpretation of the results. Furthermore, whether acute alcohol poisoning was the primary cause of death or a contributing factor is not always clear when compilations of fatal blood-ethanol concentrations are published (1–4).

Unacceptably high variations in postmortem blood-ethanol concentrations might indicate drawing blood specimens for analysis from different sampling sites, e.g., heart or peripheral vein (5,6) or postmortem diffusion of ethanol, which is also a confounding factor especially if alcohol remains unabsorbed in the stomach at the moment of death (7–9). A host of other considerations impact on the reliability of postmortem blood-ethanol concentrations (e.g., microbial activity and decomposition) as discussed in detail elsewhere (10,11).

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The purpose of this article is to provide medians and reference ranges for postmortem blood-ethanol concentrations in forensic autopsies where the primary cause of death was reported as either acute ethanol poisoning or chronic alcoholism. The blood specimens analyzed were always taken from a femoral vein, and the analytical method was headspace gas chromatography. The distributions of blood-ethanol concentrations were compared for men and women as a function of their age and the cause of death.

Materials and Methods

The Swedish National Board of Forensic Medicine (Rättsmedicinalverket) has created a forensic pathology database (RättsBase), which contains, among other things, information about the cause of death in forensic postmortem examinations made throughout the country (12). This database along with the forensic toxicology database (ToxBase) was searched to find instances where ethanol was the only drug present in femoral venous blood (>0.01 g/100 mL) regardless of the cause of death (13). We started with over 7000 cases in which postmortem blood-ethanol concentration exceeded 0.01 g/100 mL and from these identified cases when the forensic pathologist found that the underlying or primary cause of death was either acute alcohol poisoning ($N = 693$) or chronic alcoholism ($N = 825$). Both primary and secondary causes of death are coded by the forensic pathologists based on information in each case file including the police report, hospital records, other history, autopsy findings, and toxicology results. Abnormalities relating to alcohol abuse and chronic alcoholism were carefully considered,

such as alcohol psychoses, alcoholic cardiomyopathy, pancreatitis, as well as various liver disorders such as large accumulations of fat, hepatitis, cirrhosis, jaundice, and ascites fluid. In reaching a diagnosis of acute alcohol poisoning, the blood-ethanol concentration and events surrounding the death are the major factors considered, as well as asphyxiation through compromised breathing or suffocation by inhalation of vomit as verified by histological examination of the lungs.

All blood specimens used for toxicological analysis were taken at autopsy from femoral veins (13) and placed in a container containing potassium fluoride (1 to 2%) as preservative before being shipped to the toxicology laboratory for quantitative determination of ethanol by headspace gas chromatography (HS-GC). This method of analysis is accurate, precise, and highly specific for the determination of ethanol and other low-molecular weight volatiles in body fluids (14). Besides ethanol, the concentrations of methanol, acetone, and isopropanol in blood were also determined if present. The limits of quantitation of these substances in routine postmortem casework were 0.01 g/100 mL, and concentrations below this were reported as negative. Analytical results were reported with two decimal places for concentrations less then 0.10 g/100 mL and one decimal place for concentrations above this threshold. The mean of duplicate determinations done with different HS-GC instruments, each fitted with a different column and stationary phase, was reported and used for the statistical analysis.

Results

Table 1 shows the age, gender, and blood-ethanol concentrations of individuals when the death was ascribed to either acute alcohol poisoning or chronic alcoholism. The number of men exceeded the number of women by about 3:1 in acute poisoning deaths, although their mean age was about the same, being in their mid-fifties with 38% between 50 to 59 years. The average blood-ethanol concentration was slightly higher in women (0.373 g/100 mL) compared with the men (0.356 g/100 mL) for the acute poisonings. In the alcoholism deaths, 85% were men and 15% were women and both sexes were in their mid-fifties with 39% aged 50 to 59 years. The mean blood-ethanol concentration in chronic alcoholism deaths was slightly higher in men (0.174 g/100 mL) compared with women (0.157 g/100 mL) but considerably less by about 0.2 g/100 mL on average compared with acute alcohol poisonings. There were no associations between postmortem blood-ethanol concentrations and age at death, neither in acute poisonings ($r = -0.17$) nor in chronic alcoholism deaths ($r = -0.09$)

Figure 1 shows a frequency distribution of blood-ethanol in the acute alcohol poisoning deaths, and this showed a good fit to a Gaussian curve (coefficient of skewness −0.043 and kurtosis 0.71), and the mean and median values were the same (0.36 g/100 mL). The minimum and maximum blood-ethanol concentrations were 0.074 and 0.68 g/100 mL, and 90% of all cases were between 0.22 and 0.50 g/100 mL. By contrast, the blood-ethanol distribution in the alcoholism deaths was not shaped like a Gaussian curve (Fig. 2), and 221 cases (27%) were between 0.01 and 0.05 g/100 mL. The mean, median, minimum, and maximum values were 0.17 g/100 mL, 0.15 g/100 mL, 0.01 and 0.56 g/100 mL, with 90% of cases being between 0.013 and 0.41 g/100 mL.

Figure 3 compares the cumulative frequency distributions of blood-ethanol concentration in acute poisoning and chronic alcoholism, and the expected normal distributions are shown as superimposed dotted lines. There was a marked deviation from normality in the alcoholism deaths, although in the acute alcohol poisoning deaths an excellent fit to the sigmoid curve was found.

Table 2 shows the concentrations of acetone and isopropanol in blood specimens if these exceeded 0.01 g/100 mL for the two causes of death. Blood acetone was elevated in 98 deaths (12%) among chronic alcoholism cases, and the mean concentration was 0.017 g/100 mL (range 0.01 to 0.14 g/100 mL). In this same material, 50 bloods (6%) contained isopropanol concentrations above 0.01 g/100 mL (mean 0.017 g/100 mL, range 0.01 to 0.069 g/100 mL). There were 28 cases (4%) with elevated blood-acetone identified in the acute poisoning deaths at approximately the same mean concentration of 0.017 g/100 mL (range 0.01 to 0.068 g/100 mL). In the ethanol poisoning deaths, the blood-isopropanol concentration was elevated in 22 cases (3%) with a mean of 0.016 g/100 mL and range 0.01 to 0.085 g/100 mL. No correlations were found between blood-ethanol concentration and blood acetone concentration, neither in the acute poisoning deaths nor chronic alcoholism cases.

Discussion

Many factors need to be considered when postmortem blood-ethanol concentrations are interpreted and a cause of death assigned either to acute alcohol poisoning or chronic alcoholism (10,11). The mechanism by which alcohol kills depends on circumstances and events surrounding the death, and any trauma associated with gross alcohol intoxication and drunkenness needs to be excluded. The selection of cases for the present study was based on the pathologists report, i.e., whether the coding indicated either acute alcohol intoxication or chronic alcoholism as the underlying cause of death.

Ethanol is a central nervous system depressant drug exerting its effects, in, among others ways, via the $GABA_A$ inhibitory receptor (15–17). At very high blood-ethanol concentrations, the respiratory center at the brain stem is paralyzed and the person stops breathing owing to oxygen deficiency (15). The rate of increase in blood-ethanol concentration is seemingly an important determinant of acute impairment effects of alcohol and the narcotic action on the respiratory center. This effect might, however, be secondary to car-

TABLE 1—*Age, gender, and blood-ethanol concentrations in deaths attributed to acute alcohol poisoning and chronic alcoholism.*

				Blood Ethanol g/100 mL	
Cause of death	*N*	Gender	Age (SD)*	Mean (SD)	90% Range
Acute alcohol poisoning	693	Men 529 (76%)	53.7 (10.7)	0.356 (0.087)	0.21 and 0.49
		Women 164 (24%)	52.8 (11.9)	0.373 (0.083)	0.25 and 0.51
Chronic alcoholism	825	Men 705 (85%)	55.4 (10.2)	0.174 (0.133)	0.013 and 0.41
		Women 120 (15%)	56.9 (8.7)	0.157 (0.126)	0.015 and 0.41

*Standard deviation.

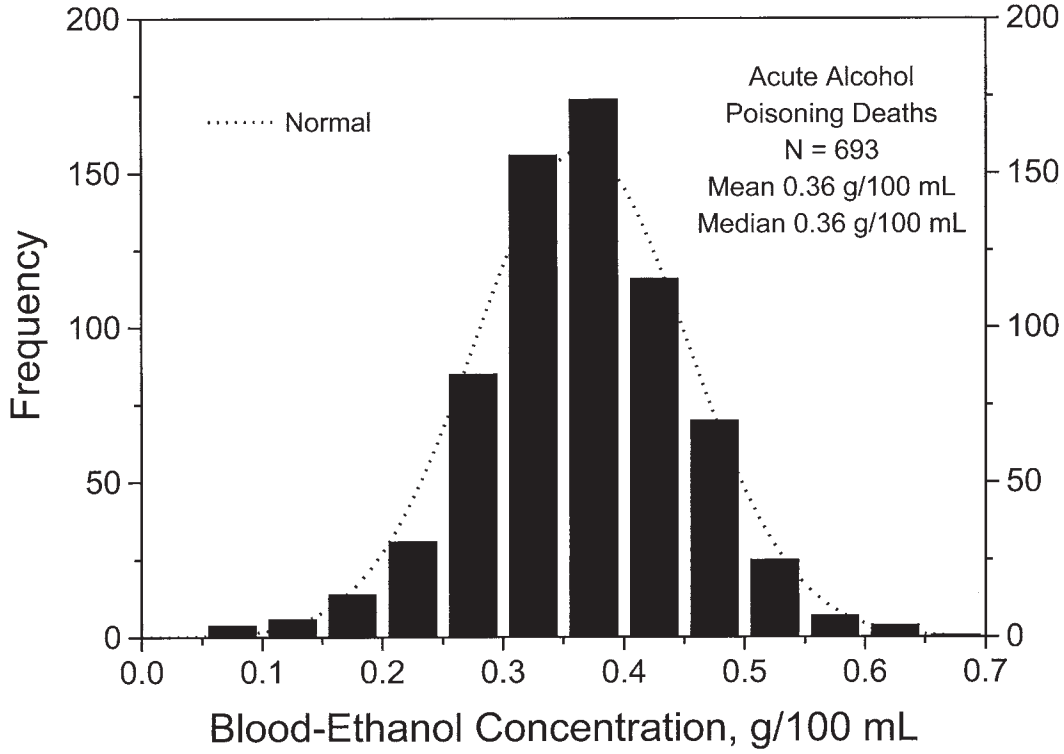

FIG. 1—*Frequency distribution of blood-ethanol concentration measured in femoral venous blood taken at autopsy in deaths attributed by the pathologist to acute alcohol poisoning. The bell-shaped dotted curve is for a normal or Gaussian distribution.*

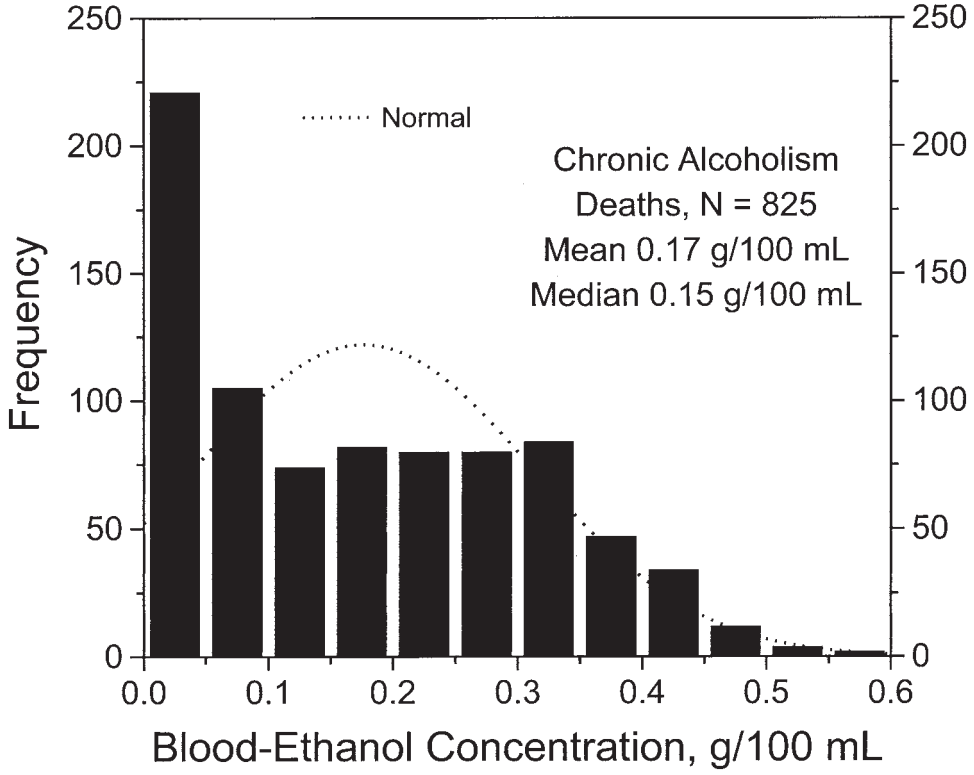

FIG. 2—*Frequency distribution of blood-ethanol concentration measured in femoral venous blood taken at autopsy in deaths attributed by the pathologist to chronic alcoholism. The bell-shaped dotted curve is a normal or Gaussian distribution.*

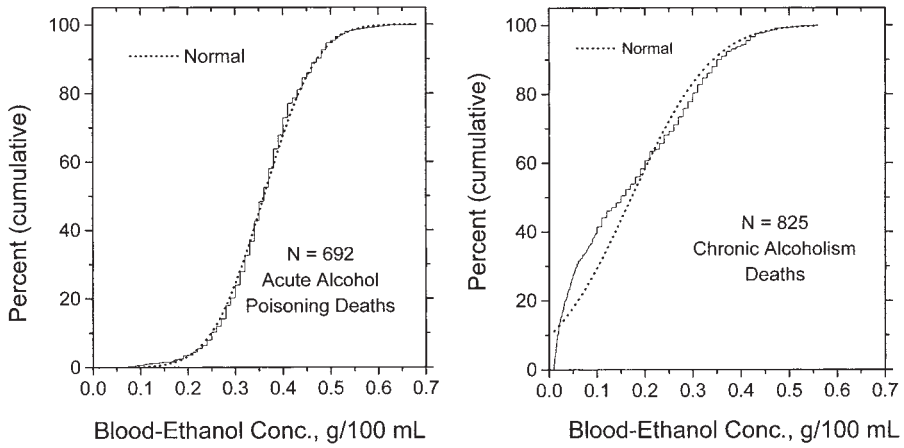

FIG. 3—*Cumulative frequency distributions of blood-ethanol concentration in deaths ascribed to acute alcohol poisoning (left trace) or chronic alcoholism (right trace). The sigmoid-shaped dotted lines are the expected normal or Gaussian distributions.*

TABLE 2—*Occurrence of acetone and isopropanol in femoral venous blood taken at autopsy in deaths attributed to acute alcohol poisoning or chronic alcoholism.*

		Blood Acetone > 0.01 g/100 mL			Blood Isopropanol > 0.01 g/100 mL		
Cause of Death	*N*	*N* (%)	Mean	Min–Max	*N* (%)	Mean	Min–Max
Acute alcohol poisoning	693	28 (4%)	0.017	0.01–0.062	22 (3%)	0.016	0.01–0.085
Chronic alcoholism	825	98 (12%)	0.017	0.01–0.14	50 (6%)	0.017	0.01–0.069

diac and circulatory failure, and such deaths are probably seen at the very highest blood-ethanol concentrations in the upper tail of the distribution (>0.5 g/100 mL) in Fig. 1.

Drinking experience and the development of acute and chronic tolerance are also factors that need to be considered when autopsy blood-alcohol concentrations are considered (18,19). A pronounced central nervous system tolerance to ethanol was reported in patients attending hospital for detoxification, and many individuals were judged only slightly impaired despite blood-alcohol concentrations of 0.3 g/100 mL or more (20,21). This phenomenon of chronic tolerance is also evident in many drunk drivers when examined by a physician and deemed to be sober or mildly under the influence despite a blood-ethanol concentration above 0.2 g/100 mL (22). Some individuals have attempted to drive with blood-ethanol concentrations exceeding 0.40 g/100 mL (23), a concentration considered fatal in most reference texts (1–4).

The blood ethanol concentration necessary to kill was discussed by Johnson (24), and he suggested that in uncomplicated alcohol poisonings 0.25 to 0.30 g/100 mL was potentially lethal ($N = 115$ cases). In deaths with an element of postural asphyxia or inhalation of vomit ($N = 68$) considerably lower blood-ethanol concentrations might be found (24,25). In a useful study, Heatley and Crane (26) investigated the blood-ethanol concentration in 175 alcohol fatalities and reported a mean blood-ethanol concentration of 0.355 g/100 mL, which is in excellent agreement with the our finding of 0.36 g/100 mL. According to Heatley and Crane (26), in deaths that are complicated by aspiration of vomit the postmortem blood-ethanol was 0.326 g/100 mL ($N = 82$) compared with 0.382 g/100 mL ($N = 93$) without an element of asphyxia (26). Teige and Fleischer (27) reported a mean blood-ethanol of 0.36 g/100 mL in 116 cases of acute alcohol poisoning, and in hypothermia-related deaths the blood ethanol concentration was appreciably lower (0.17 g/100 mL). Kaye and Haag (28) cited a terminal blood-ethanol concentration (heart blood) ranging from 0.18 to 0.60 g/100 mL in 94 fatalities attributed to acute alcohol intoxication or alcoholism. More recently, Koski et al. (29) presented a large autopsy material ($N = 615$) and found a median blood-ethanol concentration of 0.348 g/100 mL in cases when ethanol was the only psychoactive substance present.

It is important to recall that the blood-ethanol concentration determined at autopsy is not always a reliable indicator of the concentration prevailing at the time of death or even the maximum concentration existing some time before death (28). Some people might survive for several hours after a bout of heavy drinking and after they become stuporous or comatose, during which time blood-alcohol concentration decreases through metabolism. Thus, the ethanol concentration in blood at autopsy is seldom the highest the central nervous system was exposed to. Furthermore, the concentration of ethanol in blood decreases slightly after death owing to on-going enzyme activity as the body cools and might be 10% below the perimortem concentration (10,11). Ethanol starts to be metabolized from the time drinking starts until the time of death, and the clearance rate is between 0.015 and 0.025 g/100 mL/h for most people (30). With the development of metabolic tolerance, the rate of blood-ethanol decline could increase to 0.035 g/100 mL/h (31), which has been reported in alcoholics during detoxification. These arguments reinforce the conclusion that the blood-ethanol concentration at autopsy is a conservative estimate of the amount of alcohol the person had actually consumed (32).

Excessive use of alcohol is a major cause of morbidity and mortality in modern society, and many accidents and trauma deaths are a direct result of drunkenness, especially among younger age groups (33,34). Deaths occurring at fairly low blood-ethanol might have several explanations including age, gender, and experience

with alcohol. Whether resuscitation attempts or any hospital treatment was given would tend to prolong the time available for elimination of ethanol from the blood (28). Another common alcohol-related death is aspiration of vomit, thus blocking the airways and resulting in asphyxia by impairing the gag reflex (26). Positional asphyxia is another likely scenario if a drunken person is placed in an awkward position to "sleep it off"; this might lead to compromised breathing and potential serious consequences. One of the many effects of heavy drinking is to lower the core body temperature (35), which can become exaggerated if the person also falls asleep in a cold environment such as outdoors in the winter (36,37).

Alcohol withdrawal following cessation of an episode of heavy drinking represents a dangerous condition depending on the severity of seizures, delirium, and hallucinations; many such deaths have been reported (38). Chronic heavy drinking also leads to metabolic disturbances such as hypoglycemia, hyperlipidemia, and hyperlactemia (39). Lack of a proper diet in alcoholics combined with the empty calories derived from combustion of ethanol exaggerates the potential for hypoglycemia. Under these conditions, the main energy source switches from glucose to catabolism of fats (39), which produces excess ketone bodies in the blood (acetone, acetoacetate, and β-hydroxybutyrate) and in many instances a dangerous state of metabolic ketoacidosis (40).

It is noteworthy that alcoholic ketoacidosis was suggested as a likely cause of death in alcoholics found at home with low or zero blood-ethanol concentration and nothing more remarkable at autopsy than a fatty liver (41,42). We found more deaths in the chronic alcoholism group (12%) with elevated concentrations of acetone in blood compared with the acute alcohol poisoning deaths (4%). However, the average concentrations of acetone and isopropanol (~0.017 g/100 mL) were about the same and cannot be considered toxic per se. Indeed, acetone might have arisen from the metabolism of fats as a consequence of inadequate supply of food during a drinking binge. The reduction of acetone to give isopropanol is facilitated when the hepatic $NADH/NAD^+$ ratio is high as it is during the oxidation of ethanol (39,44). Otherwise, the presence of elevated concentrations of acetone and isopropanol in blood might reflect use of denatured alcohol preparations laced with various organic solvents. Studies have shown that the concentrations of endogenous acetone in blood are very low, and a median of 0.0003 g/100 mL was reported in 500 drunk drivers with the highest concentration being 0.006 g/100 mL (45). After people drink isopropanol, this alcohol is rapidly converted into acetone, which has a much longer half-life (~17 to 27 h) compared with isopropanol itself (~1 to 3 h) so that acetone is detectable in blood long after isopropanol has disappeared (46).

Chronic alcoholics often suffer from serious medical conditions such as cardiomyopathy, pancreatitis, hepatitis, and cirrhosis, all of which might have contributed to or accounted for their demise (38). In short, there are many contributory factors that need to be considered when cause of death is assessed in chronic alcoholics besides the prevailing blood-ethanol concentration (47–50). This probably explains, at least in part, the lower median blood-ethanol concentration of 0.15 g/100 mL in this group compared with 0.36 g/100 mL in acute alcohol poisoning deaths.

In conclusion, we report that the median blood-ethanol concentration in deaths attributed to acute alcohol poisoning was 0.36 g/100 mL and the 5^{th} and 95^{th} percentiles were 0.22 and 0.50 g/100 mL. These values can be compared with a median blood-ethanol of 0.15 g/100 mL in deaths attributed to chronic alcoholism with 5^{th} and 95^{th} percentiles of 0.014 and 0.41 g/100 mL. These concentrations are probably underestimates of the highest blood-ethanol concentration reached before deaths and likewise the total amount of alcohol consumed owing to metabolism of alcohol taking place up until the time of death.

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