Reduction of Drinking in Problem Drinkers and All-Cause Mortality

J. Rehm1,2,3,4,5 and M. Roerecke1,2,*

1 Social and Epidemiological Research (SER) Department, Centre for Addiction and Mental Health (CAMH), 33 Russell Street, Toronto, ON, Canada M5S 2S1, 2 Dalta Luna School of Public Health, University of Toronto, Toronto, Canada, 3 Department of Psychiatry, Faculty of Medicine, University of Toronto, Toronto, Canada, 4 PAHO/WHO Collaborating Centre for Mental Health & Addiction, Toronto, Canada and 5 Epidemiological Research Unit, Klinische Psychologie & Psychotherapie, Technische Universität Dresden, Dresden, Germany

* Corresponding author: Tel.: +41-416-535-8501; E-mail: m.roerecke@web.de

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BACKGROUND: AGGREGATE-LEVEL STUDIES OF REDUCTION OF DRINKING AND ALL-CAUSE MORTALITY

Alcohol consumption has been linked with considerable mortality as a result of being causally related to >200 International Classification of Diseases codes (Rehm et al., 2009; Lim et al., 2012). Most risk curves for disease and injury outcomes suggest a monotonic dose–response relationship between average volume of alcohol consumption and all-cause mortality where higher levels of volume of alcohol consumed are associated with higher risk for mortality (Rehm et al., 2010a). The most notable exceptions are ischaemic conditions (Patra et al., 2010; Roerecke and Rehm, 2012) and diabetes (Baliunas et al., 2009). Overall, the detrimental effects of alcohol outweigh by far the beneficial effects, and as a result, the risk of mortality increases steadily after 10 g average daily consumption in European populations and is more accelerated with heavy drinking (Rehm et al., 2011; Nichols et al., 2012). As a consequence, reduction of drinking, and especially of heavy consumption, has been suggested as the main measure to reduce alcohol-attributable mortality (Anderson and Baumberg, 2006; Babor et al., 2010), with most of the underlying evidence stemming from aggregate-level studies.

There has been a tradition in alcohol research to study the aggregate-level relationship between per capita consumption of alcohol and mortality, both all-cause mortality and various cause-specific mortality such as liver cirrhosis, suicide or categories of unintentional injury (for an overview see Norström and Ramstedt, 2005). In summary, this literature showed that overall changes in aggregate consumption in a country were associated with changes of all-cause mortality of the same direction; i.e. if per capita consumption went down, the rate of all-cause mortality went down, and if consumption went up, the rate of all-cause mortality went up (Her and Rehm, 1998; Norström, 2001; Norström and Ramstedt, 2005).

While these data have shown consistent associations, causality cannot be established in ecological studies like this (Morgenstern, 1995, 2008). For instance, it cannot be ruled out that economic conditions led to both a change in the level of drinking and mortality. Natural experiments, defined as empirical studies in which an often abrupt change in conditions occurred, such as a change in alcohol policy or by other factors out of the control of an experimenter and the drinkers concerned, allow better determination of causal direction than simple associations over time. However, alternative explanations such as concurrent secular trends during the time of the natural experiment cannot be ruled out (Cook and Campbell, 1979).

A number of natural experiments, most importantly the so-called Gorbachev reform (Leon et al., 1997), but also the 12-fold rise of taxes on distilled spirits during World War I in Denmark (Thorsen, 1990; Skog, 1993), prohibition in the USA (Dills and Miron, 2004) or the effect of German seizure of wine in France during World War II (Zatonski et al., 2010) all indicate that reduced consumption led to decreases in mortality rates. For illustration, we will just elaborate on the most prominent example, the Gorbachev reform of the 1980s, during which state-owned legal alcohol production was drastically reduced in the Soviet Union. Even though unrecorded alcohol consumption increased, the overall annual consumption of pure alcohol fell from 14.2 l per capita in 1984 to 10.7 l in 1987—a decrease of some 25% (Shkolnikov and Nemtsov, 1997). In that period, all-cause mortality rates in Russia in the 40–44 age range decreased by 39% for men and by 29% for women.

Between 1987 and 1994, after the alcohol ban was rescinded in 1987, alcohol consumption increased again to 14.5 l per capita, slightly more than its former level. In this period, all-cause mortality rates more than doubled for men and almost doubled for women in the age group 40–44 years (Leon et al., 1997). There are few alternative explanations for the decrease in mortality, as most other changes during that time would have led to an expectation of increased mortality. Of course, for the second period between 1987 and 1994, other changes associated with the dissolution of the Soviet Union could easily be hypothesized as responsible for
the increase in mortality rates, but newer research seems to exclude many of these alternative explanations and establish the end of the Gorbachev reforms as the main relevant cause (Bhattacharya et al., 2012).

In addition, the distribution of cause of death further strengthens the interpretation that alcohol was a major cause of the change in mortality rates: both the decrease and increase in mortality were strongest in fully alcohol-attributable causes of death such as alcohol poisoning (Leon et al., 1997). There was almost no change in cancer death rates; however, a change in cancer rates would not have been expected based on the long-time interval for development of this disease [(Rehm et al., 2007); for lag times in general see Holmes et al. (2012)].

These aggregate-level experiences lead to the hypothesis that on an individual level as well, reductions in consumption would lead to reduction in mortality. In the rest of this article, we examine this hypothesis based on individual-level studies.

METHODS

Meta-analysis

We will first present a narrative review of cohort studies of reduction of drinking and mortality and then a recent Cochrane review of this topic based on brief interventions (McQueen et al., 2011). To illustrate the effects of reduction on mortality, we extracted data from all the six randomized controlled trials (RCTs) in this Cochrane review (McQueen et al., 2011) with a mortality follow-up after 1 year (Chick et al., 1985; Gentilello et al., 1999; Saizt et al., 2007; Freyer-Adam et al., 2008; Tsai et al., 2009; Liu et al., 2011), and one study with a mortality follow-up of 6 months (McManus et al., 2003), which also had data on changes in consumption during the follow-up time. We pooled the estimates across studies using inverse-variance weighted DerSimonian-Laird random-effects models to account for between-study heterogeneity (DerSimonian and Laird, 1986) (see Fig. 1).

Modelling mortality risk by drinking

Based on these results, we modelled the effects of risk reduction on mortality (see Fig. 2). The probability of a 40-year-old French man was taken as the basis for the illustration of effects (http://www.ined.fr/en/everything_about_population/graph_month/age_mortality/), with the additional assumptions that drinking 8 drinks containing 12 g of pure ethanol each per day was associated with a relative risk of 9, based on the different relative risks from meta-analyses (Rehm et al., 2010a). The shape of the mortality curve was taken to be exponential as best approximation of the accelerated risk curves between the level of alcohol consumption and mortality (White et al., 2002; Rehm et al., 2010a).

INDIVIDUAL-LEVEL STUDIES OF REDUCTION OF CONSUMPTION AND MORTALITY

On an individual level, there are a number of cohort studies, which examined the impact of reduction of drinking (excluding those who quit drinking altogether) on mortality in the general population. However, heavy drinking has been defined differently and often quite widely in these studies, sometimes starting with more than two drinks or 24 g of pure alcohol per day defined as heavy alcohol consumption levels. Using this definition, Fillmore et al. (2003) found that heavy drinkers in a US nationally representative survey who reduced their drinking over 10 years of follow-up also reduced their risk of mortality compared with those who continued heavy drinking. This result was seen only in men, however. Other cohorts found overall mixed results varying with definitions of reduction, age and comparison groups (Goldberg et al., 1994; Grønbaek et al., 2004; Ermerson et al., 2005); among these, the study with the highest threshold for heavy drinking with six drinks or 48 g of pure alcohol in the UK (Emerson et al., 2005) found a substantial decrease in mortality risk between those who reduced their consumption to less than heavy drinking levels compared with those who continued heavy drinking.

The comparison group is important here. In the general population in high-income countries, a sizable proportion of people who quit drinking do so because of illness due to their drinking (‘sick quitter effect’), for instance if their physician told them. Of course, these people have higher mortality risks than lifetime abstainers or former light drinkers (Shaper et al., 1988; Marmot and Brunner, 1991; Rehm et al., 2010a). The effect of a reduction of drinking can be...
involve one to four short (5–15 min) counselling sessions with a trained interventionist (e.g. physician, psychologist, social worker). They generally aim to moderate a person’s alcohol consumption to sensible levels, and to eliminate harmful practices such as single occasion heavy drinking. For people with more severe problems such as alcohol dependence, more intensive therapy is recommended (Raistrick et al., 2006). In total, all-cause mortality in these studies was reduced on average by 43% (95% CI: 12–63%) in the experimental group compared with the control group [data taken from (McQueen et al., 2011)]. Between 1 and 16 deaths were recorded in each trial group. Across all studies, 113 deaths were recorded, 45 in the experimental groups and 68 in the control groups. The corresponding reduction in average alcohol consumption per day from a mean level of 91.2 g was 43.7 g pure alcohol (95% CI: 23.5–63.9 g) in the experimental group and 25.4 g (95% CI: 15.8–35.0 g) in the control group. In other words, the reduction in the experimental group amounted to 47.9% and in the control group 27.9%. Overall, a difference in reduction of 18.3 g of pure alcohol per day between experimental and control group was associated with a 43% reduction in mortality.

MODELLING THE EFFECTS OF REDUCED DRINKING ON MORTALITY FOR HEAVY DRINKERS IN A HOSPITAL SETTING

Based on the above numbers (Fig. 1, and assuming that the risk reduction between experimental and control group is based on the differences in alcohol consumption), we modelled the effects of reduction of drinking from high levels (for detailed methods see above). This assumption makes sense, as it is based on RCTs where all other differences should be equally distributed in both experimental and control groups. The results can be seen in Fig. 2. On the y-axis, the 1-year mortality risk per 10,000 is given, and on the x-axis, the reduction in average consumption in grams per day from a baseline level of eight drinks (or 96 g pure alcohol per day). The curve displays the change in 1-year mortality risk of a 40-year-old French man drinking 96 g of pure alcohol per day at baseline based on a reduction from that level.

The level of alcohol consumption has been shown to be approximately exponentially related to mortality for most causes of death, including all-cause mortality [see Rehm et al. (2010a) for an overview, Gmel et al. (2003a) for all-cause mortality; (Rehm et al., 2010b) for liver cirrhosis mortality, which is accounting for most alcohol-attributable mortality in chronic diseases]. Consequently, the reduction is following a decelerated decreasing curve, where reduction in mortality per drink is highest for the heaviest drinking category. For example, in the risk curve displayed in Fig. 2, a reduction of three drinks or 36 g of pure alcohol per day from a baseline level of 96 g results in a reduced mortality risk of 119 per 10,000; however, a similar reduction of 36 g from a baseline level of 60 g per day results in a reduced mortality risk of 38 per 10,000.

DISCUSSION

Both aggregate- and individual-level studies showed that a reduction in alcohol intake led to a reduction in mortality, if compared with continuous drinking at a high level. This was most notable for heavy drinking defined by at least 48 g of pure alcohol per day. In general population cohort studies in high-income countries, the effect may be obscured by a ‘quieter effect’ (Shaper et al., 1988), i.e. comparing those who quit drinking for health reasons with those who continued drinking.

Overall, even though we have cast our net quite widely (including cohort and intervention studies), the evidence base regarding consequences of reduction of drinking for all-cause mortality in individual-level studies is relatively small. There is definitely a need for more research in this area studying reduction with various thresholds for heavy drinking in both natural and intervention settings. This is especially relevant because a reduction of drinking is the main goal for most interventions for problem drinkers, such as BIs (Bertholet et al., 2005; Moyer and Finney, 2005) or motivational interviewing (Miller, 1983). Reduction of drinking is also a possible goal for treatment of alcohol dependence in certain conditions (National Institute for Health and Clinical Excellence, 2011; Rehm et al., 2013a).

Because mortality is arguably the most important distal outcome for interventions regarding problematic consumption levels, the effect of a reduction of heavy drinking levels to sensible drinking levels should also be studied with respect to stability of effects and attractiveness of such interventions. Stability is important and may require multiple smaller interventions or booster sessions of the original intervention, which seems possible, given the new technical possibilities to follow up via internet or text messaging (Moyer and Finney, 2005). As for attractiveness to clients, this factor is important, as many people with alcohol problems fear that abstinence may be the only goal of any intervention, and this is seen as unattractive by many (Heather et al., 2010).

Empirical evidence on reduction has some implications. First, most risk curves for mortality of main causes of death are exponential (Rehm et al., 2010a), and so are the risk curves for all-cause mortality with accelerated risk per each additional unit of alcohol consumed after the nadir of minimal risk (Gmel et al., 2003a). However, this means for risk reduction that more risk is avoided by reducing the same amount of drinks from a higher level than from a lower level. As shown above, a reduction of 36 g of pure alcohol per day from 96 g/day is associated with a 3-fold greater decrease in mortality risk compared with the same reduction from 60 g/day. In other words, from a public health point of view, it is mainly the reduction from high levels of alcohol intake that is important. This is no surprise, as it had been shown that more than 75% of the overall alcohol-attributable
net mortality in Europe is caused by heavy drinking as defined by at least 60 g per day in men or 40 g per day in women (Rehm et al., 2013b).

As a consequence, from a public health point of view, a reduction of heavy drinking occasions is key, both regular heavy drinking occasions (see reasoning above) and episodic heavy drinking occasions (Gmel et al., 2003b, 2011). Any measures to reduce such occasions are likely to result in a proportionally higher decrease of mortality and thus are urgently required. While there needs to be more research, the current knowledge base is large enough to start such measures based on the precautionary principle (Foster et al., 2000; Babor et al., 2010).

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