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RISK CURVES AND GAMBLING: A REPLY FROM THE AUTHOR

Dr Cunningham [1] raises some excellent points regarding this study [2]. He speaks largely to the limitations of the survey data from which the risk curves were generated, rather than the analytical approach taken. He is correct in pointing out that the frequency data on each type of gambling were collected independently. Our composite measure of reported gambling frequency has limitations and is likely to be an underestimate of actual frequency when one considers that individuals can and often do engage in different gambling activities on different days of the month.

The missing data on gambling consequences for a large proportion of the original sample is another limitation of the survey over which we had no control. Dr Cunningham is correct that many of the excluded respondents reported gambling in the last year, but self-identified as non-gamblers in the first screening question of the gambling consequences section. These individuals were not administered any additional questions on consequences. The rationale for this decision provided by Statistics Canada is that pilot testing of the CCHS-1.2 (Canadian Community Health Survey, Cycle 1.2—Mental Health and Well-Being) revealed that many of the low-frequency gamblers and individuals who self-identified as being non-gamblers (despite having reported some gambling activity in the preceding questions) strongly objected to being asked the consequences questions. Rather than risk having individuals terminate the interview prematurely, the decision was made to administer the consequences questions only to people who identified as gamblers. It is likely that the majority of the excluded people would report zero or few gambling-related problems. However, this is an assumption we cannot verify. We agree that it limits the generalizability of the results.

These limitations speak to the challenge of using survey data for reasons other than its intended purpose. The CCHS-1.2 and other problem gambling prevalence surveys were not developed for the purpose of generating dose–response risk curves. Similarly, population health surveys on alcohol consumption patterns [3] were not developed for the purpose of constructing low-risk drinking guidelines, although data from such surveys were ultimately used for that purpose. We acknowledge the need to cross-validate our findings with other survey data. Our study may also inform the development of future surveys to ensure that accurate data on the dimensions of gambling behavior are collected to compare with risk of gambling-related harm.

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MEASURING ALCOHOL CONSUMPTION IN MAN—IT IS TIME FOR A CHANGE

Recently, Gmel *et al.* have published data on the usefulness of three rating scales for the quantification of alcohol consumption under various conditions, particularly for cross-cultural research [1]. Across regions and cultures, there exist large differences in alcohol-related morbidity [2]. Hence, adapted measures are warranted, enabling scientists to compare ethanol intake between countries and to identify exact cut-offs of risk-relationships. The questionnaires in Gmel *et al.* [1] focus on the amount of consumed 'standard drinks', which are comparable in terms of ethanol content (volume percentages). Depending on the numbers consumed, in a second step data were converted into grams of pure ethanol. Despite this sounding pragmatic, we believe a third step is in order.

In pre-clinical animal research, the standard measure of ethanol intake is g/kg/day (grams pure ethanol per kg body weight per day). Considering the fact that ethanol is equally distributed throughout the body and cellular compartments, the volume of ethanol distribution ($V_D \sim 0.6$ l/kg) is finally dependent on body mass. Thus, ethanol-related behavioural effects as well as organ damage risks are associated with tissue concentration, rather than with absolute intake. The g/kg ethanol measure has been shown to produce comparable results between dif-

ferent strains and different species in terms of ethanol-induced behaviour, organ damage and morbidity [3].

In humans, we also have large differences in body mass (~ volume of distribution), caused mainly by three factors: (i) as an effect of gender, (ii) as an effect of genetically based trait values along climate zones and (iii) as an effect of socio-economic and cultural backgrounds. Thus tallness is directly related to male gender, adaptation to cold (Bergmann's rule) and a high socio-economic background (increased in industrial countries). Given a body mass index (BMI) of 22, the mean body weight of a Danish man is 72.1 kg (woman: 62.8 kg), of an Italian man 68.9 kg (woman: 59.1 kg), of an American man 68.9 kg (woman: 57.7 kg) and of a Chinese man 63.6 kg (woman: 55.6 kg). Hence, the identical absolute intake of 20 g ethanol of a Chinese woman and a Danish man results in pronounced differences in relative concentrations (0.36 g/kg versus 0.28 g/kg).

From a scientific point of view, is it adequate to compare alcohol consumption in man on the basis of absolute measures? We think not! To show that the implementation of the measure g/kg ethanol would markedly influence clinical research, underlining the necessity for a change, we want to clarify this with an example from a recently published clinical trial on the efficacy of pharmacological relapse prevention treatment [4]. In this paper, relapse to drinking was one major outcome criterion and the standard measure of relapse (five standard drinks of 12 g ethanol a day for men and four for women) was applied. Hence, the cut-off was defined as a consumption of 60 g ethanol a day for men and 48 g for women. In this study, one patient fulfilled the criterion of a relapse after an intake of 1.5 l of beer (~ 60 g ethanol) during treatment. Another patient, who consumed 0.7 l of wine, remained in the study, as his ethanol intake (~ 56 g) did not reach the criterion for a relapse. However, because the first patient had a body weight of 88 kg and the second patient had a body weight of 72 kg, the second had a relatively higher consumption (0.77 g/kg versus 0.68 g/kg) experiencing more pronounced alcohol effects.

Undoubtedly, the measure g/kg ethanol would not only largely influence clinical reports or cross-cultural comparisons but would also have a strong implication regarding gender differences. In contrast to pre-clinical animal studies, human data regularly show a lower alcohol intake in females. Is this a proper assumption, taking a relative measure such as g/kg ethanol into consideration? In the above-mentioned study, mean alcohol consumption per day prior to treatment was 214.9 g in women, compared with 268.1 g in men ($P < 0.05$). However, body weight also differed significantly (66.8 kg versus 78.3 kg; $P < 0.05$). Translating these data in g/kg, average alcohol consumption stood at 3.2 g/kg in females and 3.4 g/kg in males, without showing any statistical difference.

These examples suggest that for the comparison of ethanol intake between subjects and, furthermore, for cross-cultural research, translation of absolute intake into a ratio including body weight is necessary, in order to avoid artificial results and misinterpretations. As shown above, body weight is not normally distributed between different countries and cultures. Therefore, a systematic error arises with false to low intake in subjects with low body mass, when ethanol intake is not controlled for body weight. It is easy to do, and we ought to start including this well-proven measure from the pre-clinical settings (animal studies and psychopharmacological experiments that administer alcohol) into our clinical trials.

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MEASURING ALCOHOL CONSUMPTION—IS A REASONABLE CHANGE ALWAYS REASONABLE? RESPONSE TO KIEFER & SPANAGEL (2006)

We would like to thank Kiefer & Spanagel [1] for their thought-provoking letter related to improving alcohol measurement. Their point is provoking, because to our knowledge no cross-cultural research, including meta-analyses to measure relative-risks (e.g. estimates of mortality and morbidity such as those presented in the Report of the World Health Organization [2]), has applied adjustments for body weight. Even the Global Burden of Disease Study [3] did not use weight-adjusted cut-offs to define the three levels of alcohol intake, although they did use different cut-offs for men and women. It would be interesting to see whether the findings from this report would