
Letters to the Editor

WHAT CAME FIRST? COMMENT ON DOM ET AL. (2006)

What came first? The old question about the chicken and the egg is an important theme of the paper by Dom *et al.* (2006). Is greater impulsivity, measured in variety of ways including delay discounting and questionnaire measures of personality, the initiating cause for alcoholism, or is it a downstream consequence of consuming alcohol? In some respects, the answer to this question is the Holy Grail in the study of discounting and drug dependence. We applaud this attempt to add to the current knowledge of the discounting/drug-use relationship as well as the application of the discounting paradigm to different subgroups of a drug-dependent population.

The authors of this paper suggest that impulsive decision-making is at least partially reflective of a stable personality trait and not due solely to alcohol- or drug-use status. The basis of their inference derives from their finding that early-onset alcoholics (EOA) discounted more than late-onset alcoholics (LOA), and LOA did not differ from a control group (cf. Vuchinich & Simpson 1998; Petry 2001). Our understanding of the literature is consistent with this conclusion, suggesting that the exclusive validity of one direction of causation is not tenable; understanding of the relationship between addiction and measures of impulsivity will require a subtle and interactive view.

While we agree with the general principle that impulsiveness as measured by delay discounting is partially a trait variable, there are internal challenges to the group differences observed in the present study. First, we question the use of unmatched EOA and LOA groups. All other things being equal, we would predict that younger, less educated and more drug- and psychiatrically involved individuals (such as EOA in the present study) would discount more than older, more educated and less drug- and psychiatrically impaired individuals (such as LOA in the present study; Green *et al.* 1994; Kollins 2003; Jaroni *et al.* 2004). The observed difference between EOA and LOA is challenged by the competing hypothesis that these demographic variables account for the difference. Statistical control of unmatched variables pales to ensuring matching on those variables. Furthermore, the controls were also unmatched, and strong conclusions cannot be drawn when they are compared to the two alcoholic groups. Additionally, we presume the unmatched controls also drank alcohol (the amount was unreported) and as such this sample is different from prior studies. These previous studies almost always com-

pared drug users with matched controls who were never users. A comparable comparison with alcoholics would require controls that never drank alcohol. Detecting differences between light and heavy users may require more power.

Nonetheless, we believe as this area of research grows, more and more examples will show evidence both that drug use results in greater discounting and impulsivity and that greater discounting and impulsivity results in drug use. For instance, Reynolds (2004) found evidence supporting the former, showing that number of cigarettes consumed was correlated with the rate of delay discounting. On the other hand, Perry *et al.* (2005) found support for the latter, showing that rats who discount more acquire cocaine self-administration sooner. We believe that clarifying the pathways and the processes by which individuals become both addicted and impulsive will inform neuro- and behavioral science, our efforts regarding prevention and treatment and will lead to more nuanced, interactive understanding of behavior, biology and environment as they play out in addictive disorders.

WARREN K. BICKEL & RICHARD YI *Center for Addiction Research, University of Arkansas for Medical Sciences, 4301 W. Markham Street # 843, Little Rock, AR 72205, USA. E-mail: wbickel@uams.edu*

References

- Dom, G., D'haene, P., Hulstijn, W. & Sabbe, B. (2006) Impulsivity in abstinent early- and late-onset alcoholics: differences in self-report measures and a discounting task. *Addiction*, **101**: 50–59.
- Green, L., Fry, A. F. & Myerson, J. (1994) Discounting of delayed rewards: a life-span comparison. *Psychological Science*, **5**, 33–36.
- Jaroni, J. L., Wright, S. M., Lerman, C. & Epstein, L. H. (2004) Relationship between education and delay discounting in smokers. *Addictive Behaviors*, **29**, 1171–1175.
- Kollins, S. H. (2003) Delay discounting is associated with substance use in college students. *Addictive Behaviors*, **28**, 1167–1173.
- Perry, J. L., Larson, E. B., German, J. P., Madden, G. J. & Carroll, M. E. (2005) Impulsivity (delay discounting) as a predictor of acquisition of IV cocaine self-administration in female rats. *Psychopharmacology*, **178**, 193–201.
- Petry, N. M. (2001) Delay discounting of money and alcohol in actively using alcoholics, currently abstinent alcoholics and controls. *Psychopharmacology*, **154**, 243–250.
- Reynolds, B. (2004) Do high rates of cigarette consumption increase delay discounting? A cross-sectional comparison of adolescent smokers and young-adult smokers and nonsmokers. *Behavioural Processes*, **67**, 545–549.

Vuchinich, R. E. & Simpson, C. A. (1998) Hyperbolic temporal discounting in social drinkers and problem drinkers. *Experimental and Clinical Psychopharmacology*, 6, 292–305.

EARLY-ONSET ALCOHOLISM: A SEPARATE OR UNIQUE PREDICTOR OF DELAY DISCOUNTING? COMMENT ON DOM ET AL. (2006)

Dom *et al.* [1] report on differences in discounting rates between patients with early and late onset of alcoholism. Individuals with early-onset alcoholism discounted delayed rewards more rapidly than their late-onset counterparts, who evidenced discount rates similar to controls. Differences between the groups were also noted with respect to a variety of other self-report indices of impulsivity and sensation seeking.

These findings add to the growing literature on discounting rates and substance abuse. Published reports now demonstrate that virtually every substance-abusing population discounts delayed rewards more rapidly than controls. In addition, more and more studies are emerging that examine factors within drug-abusing populations that are related to discounting, such as the effects of drug abstinence or withdrawal [2,3], antisocial personality disorder [4] and severity or multiplicity [5,6] of problems. A study from our laboratory [7] even found that paternal history of alcoholism was related to high discounting rates in non-substance-abusing women.

Early onset of alcoholism is related to many of the above factors, including heritability, increased number and severity of substances used and antisocial personality disorder. It is not surprising that this subgroup of early-onset alcoholics discounted more rapidly than late-onset alcoholics, but this study is, nevertheless, a useful demonstration of another factor that relates to discounting.

This is an exciting field of research. The first studies of delay discounting were conducted with laboratory animals, and the concept was applied initially to drug dependence in 1997 [8]. Since then, publications on this topic have increased exponentially. Measurement and task features have been refined, but they are not perfected. In fact, counterintuitive data are available demonstrating, for example, reductions in discounting during acute administration of drugs [9]. A report from another group of investigators [10] actually found no differences in discounting rates between early- and late-onset alcohol-dependent patients, although alcohol-dependent patients as a whole did discount more rapidly than controls.

The reasons for differences across studies and populations remain to be determined but the overarching

theme, that substance abusers evidence increased discounting, remains. Hypotheses related to why this relationship exists are in a nascent stage. Only limited data address discounting and development of substance use problems. Concordance between this measure of impulsivity and other behavioral and personality indices are not well explored, and whether discounting rates uniquely predict substance abuse, or other psychiatric conditions, is unclear. Strong evidence is not yet available that treatment, be it pharmacological or behavioral, can alter the rapid discounting of delayed rewards in drug abusing populations.

As more researchers begin and continue to explore delay discounting, perhaps some of these questions can be answered. Further understanding of these issues and their biological, psychological and societal underpinnings may lead ultimately to novel prevention and treatment of drug abuse, and perhaps other impulse control disorders as well.

NANCY PETRY *Department of Psychiatry, University of Connecticut Health Center, 263 Farmington Avenue, Farmington, CT 06030–3944, USA. E-mail: petry@psychiatry.uhc.edu*

References

1. Dom G, D'haene P, Hulstijn W, Sabbe B. Impulsivity in abstinent early- and late-onset alcoholics: differences in self-report measures and a discounting task. *Addiction* 2006;101: 50–59.
2. Mitchell SH. Effects of short-term nicotine deprivation on decision-making: delay, uncertainty and effort discounting. *Nicotine Tobacco Res* 2004;6: 819–28.
3. Bickel WK, Odum AL, Madden GJ. Impulsivity and cigarette smoking: delay discounting in current, never, and ex-smokers. *Psychopharmacology* 1999;146: 447–54.
4. Petry NM. Discounting of delayed rewards in substance abusers: relationship to antisocial personality disorder. *Psychopharmacology* 2002;162: 425–32.
5. Alessi SM, Petry NM. Pathological gambling severity is associated with impulsivity in a delay discounting procedure. *Behav Proc* 2003;64: 345–54.
6. Petry NM. Pathological gamblers, with and without substance use disorders, discount delayed rewards at high rates. *J Abnorm Psychol* 2001;11: 482–7.
7. Petry NM, Kirby KN, Kranzler HR. Effects of gender and paternal history of alcoholism on discount rates for delayed rewards in healthy subjects. *J Stud Alcohol* 2002;63: 83–90.
8. Madden GJ, Petry NM, Badger GJ, Bickel WK. Impulsive and self-controlled choices in opioid-dependent and non-drug using controls: drug and monetary rewards. *Exp Clin Psychopharmacol* 1997;5: 256–62.
9. de Wit H, Enggasser JL, Richards JB. Acute administration of d-amphetamine decreases impulsivity in healthy volunteers. *Neuropsychopharmacology* 2002;27: 813–25.
10. Bjork JM, Hommer DW, Grant SJ, Danube C. Impulsivity in abstinent alcohol-dependent patients: relation to control subjects and type 1/type 2-like traits. *Alcohol* 2004;34: 133–50.

DELAY DISCOUNTING AND EARLY ONSET ALCOHOLISM: REFLECTIONS ON THE COMMENTS BY BICKEL AND YI (2006) AND PETRY (2006)

In their comments Bickel and Yi (2006) and Petry (2006) emphasize the importance of neurobehavioural research with respect to addictive processes and highlight some critical issues in this line of work (Dom *et al.* 2006). In addition, they raise important questions not only concerning the complex interaction between impulsiveness and substance use disorders, but also with respect to the complexity of the interpretation of behavioural findings within naturalistic patient populations.

Bickel & Yi (2006) question the use of unmatched early onset (EOA) and late-onset alcoholism (LOA) groups. Although we fully agree with them that from a statistical point matching on variables is to be preferred over statistical controlling, one may question the (im)possibility of matching two (sub)groups on exactly those variables that are considered to be clinical characteristics of one of the two. A low educational level, comorbid illicit drug abuse, and psychiatric disorders have all been reported to typify EOA. In this respect, it is important to note that in prominent multidimensional classification systems (see, e.g. Babor *et al.* 1992) an early start of alcoholism clusters with a higher problem severity, poorer educational and professional status, and more psychiatric impairments (i.e. personality disorders).

Indeed, and as suggested in our study, high impulsivity may be a specific (cognitive) endophenotypic factor reflective of an underlying vulnerability not only with respect to early initiation of substance use and consequent early substance use disorders, but also towards poor peer association, low educational level due to early learning problems and school drop-out, disinhibitory behavioural problems, and the development of more and more severe psychiatric problems in adulthood. In this respect we follow the line of reasoning by Petry (2006) who emphasises that it remains an open question whether discounting rates uniquely predict substance abuse or if they reflect an underlying vulnerability with respect to other psychiatric disorders. Clearly, additional research is needed to further elaborate the role of impulsive choice and other behavioural dimensions of impulsivity (i.e. response inhibition) within other psychiatric disorders both independently and within the context of their comorbidity with substance abuse.

In view of these comments, we reanalysed our data. After having statistically adjusted the data for the confounders comorbid cluster B personality disorder, parental alcoholism, number of years of alcohol abuse, comorbid illicit drug abuse, age, and education, the difference in discounting between the EOAs and LOAs

remained (marginally) significant ($F_{1,75} = 3.912$; $P = 0.052$), while none of the confounders had a significant effect. Although the additional analysis sustained our initial conclusion, i.e. higher discounting is characteristic of early onset alcoholics, the adjustment for multiple variables did substantially reduce the statistical power (observed power 0.497), thus warranting a careful interpretation of the findings and replication in a larger sample.

In addition, we agree with Petry (2006) that the reasons for differences across studies and populations remain to be determined. Specifically with respect to alcoholic populations results of studies on delay discounting have been conflicting. Although many aspects could be involved, a key issue in the ongoing research effort is that as a phenotype alcohol dependence is in fact an extremely heterogeneous disorder complicating the delineation of more homogeneous subgroups as well as their matching with specific treatment interventions. Identification of distinctive neurobehavioural characteristics may open up new avenues that could lead towards an enhanced differentiation of alcohol-dependent patients and the detection of those individuals that are more vulnerable to a more severe and relapse-prone form of the disorder. Indeed, neurobehavioural (endophenotypic) characteristics are increasingly reported to predict relapse and as such may prove better candidates for the identification of high-risk patients than the clinical (phenotypical) variables that have traditionally been used. Noel *et al.* (2005), for instance, have suggested that impairments on an 'Alcohol Shifting Task', a variant of the go/no-go paradigm, might be predictive of early relapse in substance abuse in polysubstance abusers with alcoholism. In addition, relapse in gambling proves associated with behavioural (but not self-reported) measures of impulsivity (Goudriaan *et al.* in press). Furthermore, in their neuroimaging study Paulus *et al.* (2005) demonstrated that those methamphetamine-dependent subjects that showed low prefrontal activation during a decision-making task were the ones that relapsed significantly more frequently, while those who showed greater activation remained abstinent.

Finally, and in line with Petry (2006) and Bickel & Yi (2006), we advocate research efforts aimed at determining the underlying biological underpinnings of impulsive choice. Animal studies showed that lesions in the orbitofrontal cortex (OFC), basolateral amygdala, and the nucleus accumbens core (NAC) all result in an increase of impulsive choice (Mobini *et al.* 2002; Winstanley *et al.* 2004; Cardinal & Howes 2005). In human subjects O'Doherty *et al.* (2001) observed OFC activation induced by monetary rewards with the magnitude of activation being related to the magnitude of the reinforcers. Remarkably, OFC and NAC abnormalities have been

reported to be critically involved in addictive processes and have been recently proposed as a final common pathway underlying end-stage addiction (Dom *et al.* 2005; Kalivas & Volkow 2005). However, as with the behavioural findings, at this biological level the chicken-and-egg question also still baffles us but future systematic endeavours may shed some light on which comes first.

GEERT DOM & PETER D'HAENE *Psychiatric Centre Alexian Brothers, Boechout, Belgium. E-mail: geert.dom@fracarita.org*

WOUTER HULSTIJN & BERNARD SABBE *Collaborative Antwerp Psychiatric Research Institute (CAPRI), Antwerp, Belgium*

References

- Babor, T. F., Dolinsky, Z. S., Meyer, R. E., Hesselbrock, V., Hofman, M. & Tennen, H. (1992) Types of alcoholics: concurrent and predictive validity of some classification schemes. *British Journal of the Addictions*, **87**, 23–40.
- Bickel, W. K., Yi, R. (2006) What came first? Comment on Dom *et al.* (2006). *Addiction*, **101**, 291–292.
- Cardinal, N. R. & Howes, N. J. (2005) Effects of lesions of the nucleus accumbens core on choice between small certain rewards and large uncertain rewards in rats. *BMC Neuroscience*, **6**, 37.
- Dom, G., D'haene, P., Hulstijn, W. & Sabbe, B. (2006) Impulsivity in abstinent early- and late-onset alcoholics: differences in self-report and a discounting task. *Addiction*, **101**, 50–59.
- Dom, G., Sabbe, B., Hulstijn, W. & van den Brink, W. (2005) Substance use disorders and the orbitofrontal cortex: systematic review of behavioural decision-making and neuroimaging studies. *British Journal of Psychiatry*, **187**, 209–220.
- Goudriaan, A. E., Oosterlaan, J., de Beurs, E. & van den Brink, W. (in press) Neurocognitive functions predict relapse in pathological gambling. *Addiction*.
- Kalivas, P. W. & Volkow, N. D. (2005) The neural basis of addiction: a pathology of motivation and choice. *American Journal of Psychiatry*, **162**, 1403–1413.
- Mobini, S., Body, S., Ho, M. Y., Bradshaw, C. M., Szabadi, E., Deakin, J. F. & Anderson, I. M. (2002) Effects of lesions of the orbitofrontal cortex on sensitivity to delayed and probabilistic reinforcement. *Psychopharmacology (Berl)*, **160**, 290–298.
- Noel, X., van der Linden, M., d'Acremont, M., Colmant, M., Hanak, C., Pelc, I. *et al.* (2005) Cognitive biases toward alcohol-related words and executive deficits in polysubstance abusers with alcoholism. *Addiction*, **100**, 1302–1309.
- O'Doherty, J., Kringelbach, M. L., Rolls, E. T., Hornak, J. & Andrews, C. (2001) Abstract reward and punishment representations in the human orbitofrontal cortex. *Nature Neuroscience*, **4**, 95–102.
- Paulus, M. P., Tapert, S. F. & Schuckit, M. A. (2005) Neural activation patterns of methamphetamine-dependent subjects during decision making predict relapse. *Archives of General Psychiatry*, **62**, 761–768.
- Petry, N. (2006) Early onset alcoholism: A separate or unique predictor of delay discounting? *Addiction*, **101**, 292.
- Winstanley, C. A., Theobald, D. E., Cardinal, R. N. & Robbins, T. W. (2004) Contrasting roles of basolateral amygdala and orbitofrontal cortex in impulsive choice. *Journal of Neuroscience*, **24**, 4718–4722.

COMMENTS ON ROSSOW & ROMELSJÖ (2006). DISPELLING ILLUSIONS ABOUT THE PREVENTION PARADOX

One prevalent argument in support of the utility of environmental preventive interventions to reduce alcohol-related problems rests upon the observation that these problems are broadly represented in the drinking population. While risks related to drinking at the individual level do increase with increased drinking, most aggregate-level drinking problems are attributable to 'moderate' and 'light' drinkers. Hence, the 'prevention paradox'. While treatment programs reach only indicated problem drinkers (e.g. those entering treatment programs) and educational preventive interventions reach only selected subpopulations of users (e.g. young people in school), environmental prevention programs reach everybody, including those at a low individual-level risk who are responsible for the many drinking problems.

This is a splendid story but for several major problems. First, no one seems to know what attributes signify a 'light', 'moderate' or 'heavy' drinker. Second, attempts to solve this problem using other even more problematic concepts related to individual experiences with alcohol, such as 'intoxication', replace externally defined objective criteria with reports about internally defined subjective states. Third, and most problematic, problems related to alcohol, even among 'light' or 'moderate' drinkers, may have nothing to do with 'light' or 'moderate' drinking. 'Light' and 'moderate' drinkers may only have problems when drinking 'heavily'. Thus, one can see why progress towards understanding the prevention paradox has stalled. The central methodological problems with these studies are not only respondent attrition and subjective bias, noted by Rossow & Romelsjö [1], but also a category error reflecting our persistent focus upon individual drinkers rather than their drinking [2, p. 43]. While characteristics of drinkers, for example frequency and quantity of use, represent dispositions to drink in certain ways, patterns of drinking, actual amounts consumed at specific times and places are the cause of drinking problems.

Against this background, the excellent work by Rossow & Romelsjö has many strengths, not the least of which is dealing directly with the issue of subjectivity in assessments of problem outcomes; so our comments reflect not on their work, but on the larger scientific frameworks in which this work is conducted.

Two unanswered questions

Standing on the shoulders of Strauss & Bacon [3], alcohol epidemiology has failed to ask two central questions: 'How does drinking affect problems?' and 'How do problems affect drinking behavior?' Despite many years

of study of statistical associations between measures of drinking and problems [4–7], these questions have remained unanswered. The general impression given by this literature is that it is enough to correlate harms related to drinking with characteristics of drinkers (e.g. their frequencies and quantities of use). However, because harms related to drinking shape drinking behaviors, and vice versa—a negative feedback loop—estimates of these correlations are biased [8]. These biases are not ameliorated with increased sample sizes or refinements of measurement procedures. Rather, they require detailed attention to (a) the theoretical and mathematical foundations of models that explicitly relate drinking to problem outcomes and (b) statistical issues in the assessment of dynamic systems relating drinking-to-problems-to-drinking (i.e. non-recursive equation models), a point that has not seeped into epidemiological argument [9,10].

Drinkers and drinking

The question implied by the prevention paradox is not ‘What drinkers produce the bulk of alcohol problems?’, but ‘What drinking produces the bulk of alcohol problems?’. A well-developed theoretical and mathematical treatment of the relationships between drinking and problems, and suitable data, can be used to answer the second question, teasing out specific drinking acts (e.g. occasions of use at different levels) and their relationships with problems (e.g. dose–response models). This is not accomplished by correlating standard self-report measures of drinking (e.g. frequency and quantity) with problem outcomes. These measures represent the dispositions of drinkers (i.e. how much each is likely to drink), not drinking (e.g. number of days drinking at specific levels). Nor do such correlations constitute a theoretical model of the manner in which drinking leads to problems. In order to understand drinking and problems we must study drinking, not drinkers. In order to properly characterize the prevention paradox we need to know, at the population level, how drinking contributes to harmful outcomes.

PAUL J. GRUENEWALD & ANDREW TRENO *Prevention Research Center, Berkeley, CA 94704, USA. E-mail: paul@prev.org*

References

1. Rossow I, Romelsjö A. The extent of the ‘prevention paradox’ in alcohol problems as a function of population drinking patterns. *Addiction* 2006;**101**: 84–90.
2. Ryle G. *The concept of mind*. New York: Harper & Row; 1949.
3. Strauss R, Bacon S. *Drinking in America*. New Haven: Yale University Press; 1953.

4. Cahalan DI, Cisin I, Crossley H. *American drinking practices: a national study of drinking behavior*. New Brunswick, NJ: Rutgers Center for Alcohol Studies; 1969.
5. Clark WB, Hilton ME. *Alcohol in America: drinking practices and problems*. New York: State University of New York Press; 1991.
6. Babor T, Caetano R, Casswell S, Edwards G, Giesbrecht N, Graham K, et al. *Alcohol: no ordinary commodity. Research and public policy*. New York: Oxford University Press; 2003.
7. Stockwell T, Gruenewald PJ, Toumbourou JW, Loxley W. *Preventing harmful substance use: the evidence base for policy and practice*. New York: John Wiley; 2005.
8. Duncan OD. *Introduction to structural equation models*. New York: Academic Press; 1975.
9. Heather N, Peters TJ, Stockwell T. *International handbook of alcohol dependence and problems*. New York: Wiley; 2001.
10. Vuchinich RE, Heather N. *Choice, behavioural economics and addiction*. New York: Pergamon Press; 2003.

COMMENTS ON ROSSOW & ROMELSJÖ (2006). THE PREVENTION PARADOX

‘Spring forward—fall back’: this little mnemonic helps me to remember how to set my watch when going from wintertime to summertime and vice versa. There appear to be similar seasons of the prevention paradox. The spring of research on the prevention paradox in the alcohol field began in the late 1980s, with Kreitman (1986) applying Rose’s work (Rose 1981) on alcohol consumption and related harm. The preventive paradox was accepted with enthusiasm, because it helped to circumvent the discussion about whether reducing per capita consumption would also reduce the proportion of heavy drinkers: it stated simply that all drinkers were at risk and thus all had to reduce consumption (Saunders & de Burgh 1998). The excellent paper by Rossow & Romelsjö (2005) is a step forward in research in that it addresses some earlier shortcomings that might have incorrectly favoured the prevention paradox. They not only count cases with at least one incident in their Norwegian data sets, but also look at frequencies of incidents. In their record linkage study of Swedish conscripts they overcome many of the shortcomings of other studies, mainly low response rates and reliance on measures of self-reported harm. In general, their findings seem to confirm most of the earlier studies, namely that alcohol-related harm is an issue not only among high-volume drinkers but that low- to moderate-volume drinkers, being more numerous, contribute even more to overall harm. As Skog (1999a, b) mentioned, there is nothing paradoxical in this. Why should 10% of the drinkers, as in the example of Rossow & Romelsjö, be responsible for the entire burden? In Switzerland, 10% of the population drinks about 50% of all the alcohol consumed, as in other countries (for the United States, for example, see Greenfield & Rodgers

1999) and hence it might be expected that 50% of the burden stems from this group if all that counts is volume of drinking, and risk functions are more or less linearly increasing (Skog 1999a). This is about the percentage of harm (quarrels and fights) given in the Rossow & Romelsjö study (their Table 2).

Regrettably, the Rossow & Romelsjö study adds less to the debate on the prevention paradox—its hot summer. It has been argued that most of the harm caused by low- to moderate-volume drinkers is due to occasions of heavy drinking (Stockwell *et al.* 1996; Skog 1999a; Gmel *et al.* 2001), and therefore preventive measures focusing on the population's level of volume of drinking may have little effect and should be accompanied by harm reduction approaches. Rossow & Romelsjö look at the 10% of drinkers with highest volume and the 10% with the most frequent intoxication. Although the two groups do not overlap perfectly, frequent bingers should theoretically have a high volume (without necessarily being among the 10% with the highest volume) and high-volume drinkers will probably have occasions of intoxication (without necessarily being among the 10% with the most frequent intoxication). It is of interest that Table 3 of Rossow & Romelsjö's study shows that the 15.4% of drinkers that are either among the 10% with highest volumes or among the 10% most frequent binge drinkers clearly contribute to most of the consequences.

One central question remains, however. Why do the 84.6% remaining drinkers with low-risk-volume drinking and low risk from intoxication still account for 42% of all quarrels and 33% of all fights (Table 3). Is their mean consumption still too high and should it be reduced further? It could be that most of these consequences are related to binges, although not binges of the most frequent bingers. This question cannot, however, be answered from the study by Rossow & Romelsjö ('fall back'). Skog (1999b), actually using the 1994 Norwegian survey, showed that about three-quarters of bingers were found among low-volume drinkers; they may just not be the most frequent bingers.

At any rate, research on the prevention paradox should be understood from its consequences for prevention. Rossow & Romelsjö show that the group at highest risk contributes substantially to overall harm, and thus prevention strategies targeting high-risk groups seem valuable. However, even if the harm caused by low-volume drinkers is due to binge drinking, prevention strategies would still have to target the majority of drinkers, because most bingers are found among the majority of low volume drinkers (Skog 1999a, b; Gmel *et al.* 2001).

Heading towards the 'fall' of research on the preventive paradox, we should reap its harvest. Rossow & Romelsjö demonstrate clearly that it makes no sense to continue to use research on the prevention paradox to

argue for or against a population strategy or a high-risk-group strategy. Both are needed and each has its justification. We need to identify the best practices of prevention (Babor *et al.* 2003) and evaluate them in terms of burden that can be avoided. In estimating avoidable burden, we should abandon the idea that alcohol consumption can be reduced completely to zero; we should focus instead on achievable consumption distributions and feasible minimum risks that would minimize harm resulting from both chronic heavy use and episodic heavy drinking (WHO 2002).

GERHARD GMEL *Swiss Institute for the Prevention of Alcohol and Drug Problems, Lausanne, Switzerland. E-mail: ggmel@sfa-isp.ch*

References

- Babor, T. F., Caetano, R., Casswell, S., Edwards, G., Giesbrecht, N., Graham, K. *et al.* (2003) *Alcohol: No Ordinary Commodity Research and Public Policy* Oxford: Oxford University Press.
- Gmel, G., Klingemann, S., Müller, R. & Brenner, D. (2001) Revisiting the preventive paradox: the Swiss case. *Addiction*, **96**, 273–284.
- Greenfield, T. K. & Rodgers, J. D. (1999) Who drinks most of the alcohol in the US? The policy implications. *Journal of Studies on Alcohol*, **60**, 78–89.
- Kreitman, N. (1986) Alcohol consumption and the preventive paradox. *British Journal of Addiction*, **81**, 353–363.
- Rose, G. (1981) Strategy of prevention: lessons from cardiovascular diseases. *BMJ*, **282**, 1847–1851.
- Rossow, I. & Romelsjö, A. (2006) The extent of the 'prevention paradox' in alcohol problems as a function of population drinking patterns. *Addiction*, **101**, 84–90.
- Saunders, J. B. & de Burgh, S. (1998) Distribution of alcohol consumption. In: Grant, M. & Litvak, J., eds. *Drinking Patterns and Their Consequences*, pp. 129–152. Washington, DC: Taylor & Francis.
- Skog, O. J. (1999a) The prevention paradox revisited. *Addiction*, **94**, 751–757.
- Skog, O. J. (1999b) Alcohol policy: why and roughly how? *Nordisk Alkoholisk- and Narkotikatidskrift [Nordic Studies on Alcohol and Drugs]*, **16**, 21–34 [English supplement].
- Stockwell, T., Hawks, D., Lang, E. & Rydon, P. (1996) Unravelling the preventive paradox for acute alcohol problems. *Drug and Alcohol Review*, **15**, 7–15.
- World Health Organization (WHO) (2002) *The World Health Report 2002—Reducing Risks, Promoting Healthy Life*. Geneva: WHO.

THE CORE MESSAGE IN THE PREVENTION PARADOX: RESPONSE TO COMMENTS

We are grateful for the commentaries by Gmel [1] and Gruenewald & Treno [2] on our paper [3] and would like to express our thanks to all three. Both letters deal in some way with the relevance of research on the prevention paradox. Gmel states that this research should be

understood from its consequences for prevention, whereas Gruenewald & Treno state that the question implied by the prevention paradox should not be what drinkers produce the bulk of the problems, but rather what drinking produces the bulk of problems. Having followed the public debates on alcohol policy in our two countries over some years, it is still evident that many politicians and important actors in policy formation are convinced that most alcohol problems are caused by a very small fraction of drinkers who need treatment, not taxation or reduced availability. We will assume that such views are also expressed in public debates on alcohol policy in many other countries as well, and that research on the prevention paradox is relevant for counter-arguments in that respect.

INGEBORG ROSSOW, *Norwegian Institute for Alcohol and Drug Research, POB 565 Sentrum, N-0105 Oslo, Norway.*
E-mail: ir@sirus.no

ANDERS ROMELSJÖ, *Centre for Social Research on Alcohol and Drugs, Stockholm University, Stockholm, Sweden*

References

1. Gmel, G. (2006) Comments on Rossow & Romelsjö (2006). The prevention paradox. *Addiction*, **101**, 295–296.
2. Gruenewald, P. J. & Treno, A. (2006) Comments on Rossow & Romelsjö (2006). Dispelling illusions about the prevention paradox. *Addiction*, **101**, 294–295.
3. Rossow, I. & Romelsjö, A. (2006) The extent of the 'prevention paradox' in alcohol as a function of population drinking patterns. *Addiction*, **101**, 84–90.