The importance of environmental modifiers of the relationship between substance use and harm

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The modifying role of social and environmental determinants

Taking into consideration patterns as well as levels of substance use has improved the understanding of disease consequences and social harm on both the individual and the population levels (alcohol: Rehm et al. 2004; tobacco: Ferrence et al. 2000; illegal drugs: van Ameijden et al. 1994). However, considerable variance in the aetiology of these consequences remains unexplained. It has become apparent that variance in substance use-related harms cannot be explained solely by agent-intrinsic concepts (e.g. level of use or even pattern or mode of use). Both individual characteristics and environmental factors moderate the occurrence and severity of harms from substance use (Evans, Barer & Marmor 1994; Berkman & Kawachi 2000). Here we focus on characteristics of the social or physical environment which are associated with increased risk of negative social or health consequences from use of various substances.

The immediate environment in which substance use occurs has been shown to have a strong relationship with harmful outcomes, such as violence. For instance, studies of licensed premises have found that characteristics of the social and physical environment (decorum, crowding, ventilation and staff behaviours) and characteristics of patrons (ethnicity, intoxication level) account for 40–60% of the variance in predicting frequency and severity of aggression (Graham et al. 1980; Homel & Clark 1994). The wider socio-cultural context in which alcohol is consumed (including such factors as expectations about the effects of alcohol and cultural tolerance for alcohol-related violence) has also been shown to be a critical factor in the strength of the association between alcohol use and violence (MacAndrew & Edgerton 1969). For instance, there is some evidence of an interaction of alcohol consumption and poverty in terms of violent crimes such as homicide, with higher rates when these two risk factors are combined than could be expected from the addition of both individual risk factors (Parker 1993).

In the illicit drug use field, strong evidence has emerged of the crucial role of socio-environmental determinants for drug-related morbidity and mortality. For example, poverty and related forms of social marginalization (i.e. lack of housing, aboriginal status) as well as marginalized use environments (injection in public places, streets, etc.) have been identified as strong predictors of infectious disease transmission and overdose deaths among illicit drug users (e.g. Palepu et al. 1999; Cruib et al. 2003).

Poverty seems to be related crucially to substance use-related harm in at least two ways: first as an independent variable which may lead to harmful substance use, and secondly as a moderator in the relationship between substance use and harm. To illustrate the first point: poverty in established market economies has been associated with several health risks, such as poor nutrition, sedentary life-style and substance use (Adler & Ostrove 1999; Jarvis & Wardle 1999; Frohlich et al. 2002). Several studies have shown disproportionate numbers of individuals from socially disadvantaged groups engaging in potentially harmful substance use behaviours, including heavy consumption of alcohol as well as a higher prevalence of smoking and illicit drug use (e.g. Jarvis & Wardle 1999; Mäkelä 1999). Poverty has also been shown to modify the relationship between substance use and harm, with the same patterns of consumption of alcohol, tobacco and/or other drugs resulting in higher levels of harm among economically disadvantaged populations than among more economically advantaged groups (Cahalan & Room 1974; Lantz et al. 1998; Harrison & Gardiner 1999; Mao et al. 2001). For instance, the same smoking history of 9 or more pack-years was found to result in at least twice the risk of lung cancer in Canada 1994–97 in the lowest social class as in the highest social class (Mao et al. 2001). Similarly, Harrison & Gardiner (1999) found that, for men aged 25–69 years, those in the lowest socio-economic status (SES) category (unskilled labour) had a 15-fold higher risk of alcohol-related mortality than professionals in the highest SES category in England and Wales. This difference could not be explained by overall volume of drinking, which tended to be higher for those in higher SES categories. Thus, SES or characteristics associated with SES such as
drinking patterns or context must have moderated the effects of alcohol on mortality.

In sum, the characteristics of the social and physical environment of the user and the immediate environment in which substance use occurs contribute directly to harmful use patterns as well as modifying the level of harm associated with substance use.

THE IMPORTANCE OF INCLUDING THE SOCIAL AND PHYSICAL ENVIRONMENTAL MODIFIERS IN RESEARCH AND INTERVENTIONS

At the level of research, we need to incorporate general social determinants of health (such as socio-economic position or context) into our causal pathways leading to negative health and social outcomes of substance use (Murray & Lopez 1999). Once these causal pathways are better understood and more clearly delineated additional and more focused interventions could be conceptualized, taking these factors into consideration.

Consider, for example, the current campaign against tobacco smoking in established market economies. Although dramatic success has been achieved in reducing smoking in many countries, the classic and ‘rational choice’ based approaches such as taxation or smoking bans in certain environments have demonstrated ceiling effects, especially for certain segments of the population (Hatsukami 1999; Bondy et al. 2000). Specifically, socio-economically marginalized groups—primarily the bottom third of the SES strata—in the established market economies have continued to smoke despite the increased breadth and intensity of anti-smoking interventions (Zang & Wynder 1998). Moreover, non-compliance of certain population segments with public health measures directed at reducing smoking may contribute to their further social marginalization (Fischer & Poland 1998). In order to develop strategies to reach these groups effectively, it is necessary to have greater knowledge about the interrelationships between the role of distinct determinants, including SES and comorbidity (e.g. depression; Glassman 1993; Pomerleau 1997), which do not seem to be addressed by the current portfolio of antismoking interventions.

Knowledge derived from research on complex interactions will not necessarily translate directly into interventions; however, this knowledge can be used to frame interventions to improve their efficacy or coverage. For instance, knowing that poverty modifies the link between substance use and related harms does not offer any intervention strategy per se, because most interventions which are politically possible will not be powerful enough to change the distribution of wealth. However, if we have more concrete knowledge of the exact causal pathways and the intermediate effects leading from poverty to a negative health outcome in the context of substance use activity, we may be able to develop strategic interventions for addressing certain poverty-related risk factors. For example, it is much easier to influence and alter the use of contaminated injection equipment among poor injection drug users than it is to eliminate poverty in this population. Thus, knowledge of population- or cultural-level environmental risks could have useful applications both at the societal level (e.g. better targeting programmes for high-risk groups) and in the immediate context in which use occurs by high-risk users.

As with interventions that provide safer using environments for injection drug users, interventions with licensed drinking establishments provide another example of how preventive interventions can focus on environmental moderators of the relationship between use and harm. Bars and nightclubs are known to be high-risk environments for alcohol-related problems such as aggression, violence and injury (e.g. Stockwell, Lang & Rydon 1993), and it has been shown that these risks vary according to the physical bar-room environment (Graham et al. 1980; Stockwell et al. 1993; Homel & Clark 1994) and the behaviour and communication skills of bar staff (Hauritz et al. 1998; Wells et al. 1998). Accordingly, interventions that focus on changing the barroom environment (e.g. changes in rules or policies related to games, management of line-ups and re-entry to the bar, modifications of the social or physical environment and improvement in staff communication and intervention skills) have been shown to be effective in reducing harms from drinking in these settings without necessarily altering overall consumption levels (Homel et al. 1997; Graham et al. 2004; see also review by Graham 2000). One additional advantage of interventions directed at the environment may be that they are often more acceptable to politicians than other policies of proven effectiveness such as taxation or rationing.

CONCLUSION

In conclusion, there are some challenges in the prevention of substance-related harm that cannot be solved using approaches that focus predominantly on reduction of overall substance use or the alteration of individual behaviour. A better understanding of the aetiology of substance use-related harms, incorporating the moderating effects of the social and physical environment, needs to be developed and applied to interventions in...
order to maximize the reduction of substance use-related harms.

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References


