Key future research questions on mediators and moderators of behaviour change processes for substance abuse

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Abstract

In summarizing the key themes and results of the second meeting of the German Addiction Research Network 'Understanding Addiction: Mediators and Moderators of Behaviour Change Process', the following concrete steps forward were laid out to improve knowledge.

The steps included pleas to (1) redefine substance abuse disorders, especially redefine the concept of abuse and harmful use; (2) increase the use of longitudinal and life-course studies with more adequate statistical methods such as latent growth modelling; (3) empirically test more specific and theoretically derived common factors and mechanisms of behavioural change processes; (4) better exploit cross-regional and cross-cultural differences.

Funding agencies are urged to support these developments by specifically supporting interdisciplinary research along the lines specified above. This may include improved forms of international funding of groups of researchers from different countries, where each national group conducts a specific part of an integrated proposal. Copyright © 2008 John Wiley & Sons, Ltd.

Key words: substance use disorders, behavioural change, mechanism, research

Introduction and objectives

The objective of this contribution is to summarize key results of the second meeting of German Addiction Research Network 'Understanding Addiction: Mediators and Moderators of Behaviour Change Process'. The contribution is based not only on the formal presentations (see other contributions to this special issue), but also on the discussion and interactions during the meeting.

Key themes

Three recurrent key themes characterized the conference especially in discussion and interaction sections:

- (1) Discussions on the scope of the problem, i.e. relating to the definitions and operationalizations of substance use disorders.
- (2) Discussion on the best strategies to identify common factors and processes/mechanisms.

- (3) Key considerations of gene–environment interactions.

These themes will also structure this presentation.

Definitions and operationalizations of substance use disorders

The concept of substance use disorders (SUDs) has changed considerably over the past 25 years (e.g. Room, 1998). Currently, these disorders are usually defined according to ICD-10 (WHO, 1993; American Psychiatric Association, 2000). The earlier-mentioned definitions of substance use dependence (SD) in ICD-10 and DSM-IV converge and have been shown to be relatively stable across standard assessments and cultures, with the exception of cannabis dependence, (e.g. Grant et al., 2006; Pull et al., 1997; Üstün et al., 1997; Compton et al., 1996; Rounsaville et al., 1993). Unfortunately, the definitions for harmful use according to ICD-10 (WHO, 1993), and for substance abuse (SA) according to DSM-IV (American Psychiatric Association, 2000) are less stable and comparable across cultures and instruments (e.g. Grant et al., 2006; Üstün et al., 1997; Rounsaville et al., 1993). Moreover, the theoretical postulate, that harmful use or SA are necessary precursors to SD has not received much support and leads to seriously distorted prevalence and incidence estimates of SD if operationalized in this way, i.e. when asking symptoms of SD only if SA or harmful use has been asserted. As an example see the prevalence rates of the World Mental Health Survey (Demyttenaere et al., 2004), which are clearly underestimates (Grant et al., 2007). In addition, recent research in alcohol based on the large US representative National Epidemiologic Survey on Alcohol and Related Conditions (NESARC) with more than 20,000 people showed that the item response curves for DSM-IV Alcohol Use Disorder Criteria displayed an order opposite to the theoretically postulated: most symptoms of alcohol abuse loaded on a latent scale of alcohol use disorders as more severe than alcohol dependence (Archibald lecture of T.K. Li on 8 May 2006 on 'Alcohol Dependence Syndrome: 30 Years Later'). Of course, this result may change in different cultures based on patterns of drinking (Rehm et al., 2005a), but clearly a universal classification system cannot uphold the notion of SA or harmful use as precursors to SD based on current knowledge.

Another problem for research is, that in drug abuse research the common medical systems are not accepted

and different classifications with more or less overlap prevail (UNDCP, 2000; EMCDDA, 2003; Rehm et al., 2005b).

Thus, the current definitions of SUDs may hinder some research progress, especially in epidemiological research on initiation, where the concept of early onset SUD should be considered. With better definitions and operationalizations, we could be more effective in identifying mechanisms and processes of change both for natural course and interventions (prevention and treatment). As currently such revised definitions for both ICD-11 and DSM-V are considered, the following additional considerations should be made from a research perspective:

- For both alcohol and drug use disorders it may help to include patterns of use in the definition, especially as such patterns are linked to other disease outcomes (Rehm et al., 2003; Degenhardt et al., 2004).
- The concept of a dimensional instrument rather than a categorical one, or the use of statistical hybrid models such as item response mixture modelling would help improve future research on mediators and moderators of behaviour change processes for substance abuse (see also Muthen, 2006; Muthen and Asparouhov, 2006). Item-response mixture models could combine adequate scaling methodology with path modelling techniques, thus simultaneously testing complex hypotheses on behavioural change. A dimensional system would not consider SA and SD as dichotomous concepts (present/absent), but would assume a continuous variable such as severity of SA or severity of SD without any evident cutoff point. Hasin et al. (2006) could recently show that such a continuous concept was superior for research purposes compared to the current dichotomous concept.

Best strategies to identify common factors and processes and mechanisms

There are empirical as well as theoretical ways to identify common factors and mechanism. The papers presented at the conferences showed quite a lot of techniques of data reduction such as factor or cluster analysis. However, to achieve progress, different and more sophisticated methodological approaches will need to be used. First and foremost, life course approaches will help gain better insight in mechanisms. The seminal

work of Vaillant (1995, 1998, 2003) for alcohol, and Hser et al. (2001) for narcotic addicts lead the way also for shorter time periods. These kind of approaches should be combined with modern statistical techniques like latent growth modelling (Singer and Willett, 2003; Muthen, 1997); for an example see Muthen and Muthen (2000), to improve our knowledge. The currently used techniques of factor analysis in cross-sectional slices, however, are bound to produce limited or no insights into common factors and mechanisms, as they are by definition reflecting only sample characteristics at a certain time point (Arminger et al., 1995). In contrast, we need to look at individual developments over time, and in distinguishing groups or clusters of people, in many cases the developmental trajectories over time should be grouped rather than a bundle of co-variations at an arbitrary time point. And since the statistical techniques now exist to allow to undertake these kind of theoretically driven analyses, we should incorporate them (Muthen, 1997).

Other important strategies discussed concern the separation of potential mechanisms at least in a 2×2 matrix into pharmacological versus behavioural mechanisms, and by initiation versus extinction (the for instance with the help of treatment). In looking at these factors, we have to overcome the current situation of a large pool of so called predictors and mechanisms for initiation of SA as well as for treatment success, which appear to be very unspecific and become significant in different studies almost randomly. This is not surprising given the fact, that the pool of factors used to predict is rather large, and some of these factors should become significant just by chance, if most of them are tested in the current research paradigm. Instead of these unspecific factors, specific and theoretically derived mechanisms should be focussed on. And since a lot of these mechanisms are specific for initiation versus extinction, or with respect to modality (pharmacological versus behavioural), the above matrix makes sense.

Finally, the role of environment and societal factors should be included as well. Following Campbell's plea to more systematically implement social reforms as experiments (Campbell, 1969; Campbell, 1976), there are tremendous possibilities not only to increase theoretical knowledge, but also to gain insight in best ways to actually change behaviour on a society level (for overviews see also Rehm, 2003; Babor et al., 2003; Chisholm et al., 2004). The plea to include

environmental and societal factors should not be limited to treatment interventions, but this sector, as Babor has convincingly demonstrated (Babor, this issue), will profit considerably.

Key considerations of gene–environment interactions

The last specific area from the conference to be discussed here concerned the gene–environment interactions in behavioural change. Very clearly, SUDs and substance related problems are impacted by genetics (Lachman, 2006), and when exploring behavioural change, we should not limit ourselves to environmental factors *per se*, but examine their interaction with genetics. This will involve interdisciplinary teams with specialists from both sides. While the plea for more interdisciplinary research has degenerated into an almost meaningless motherhood and apple-pie statement without any consequences, it is all the same right. For exploring gene–environment interactions as well as for most of the research laid out earlier, interdisciplinarity is as much key as it is for modern research involving genetics.

Specifically for SUDs, we need to systematically exploit cross-regional and cross-cultural differences. Substance use is quite often culturally formed, and thus there is natural variation of antecedent conditions in this respect for exploring gene–environment interactions. For example, whereas the level of drinking alcohol and the prevalence of alcohol use disorders peaks in early adulthood in North America (Grant et al., 2006), for several European countries the peak is in mid adulthood (Rehm et al., 2005a). Comparing gene–environment interactions underlying this phenomenon would be very interesting. A basic requisite in this exploitation is the use of common methodology to assess genotypes as well as phenotypes. Unfortunately, there are in part historic traditions of using different methodologies in this assessment, which have to be overcome. But even if they are overcome and research teams from two or more countries agree on one protocol, there is the problem of funding. Often funding agencies restrict funding to the home country. Thus, the conference closed with a plea to funding agencies to increase possibilities to conduct such cross-regional and cross-cultural research, e.g. by enabling new forms of funding for multinational teams.

Finally, again a small methodological point. Usual statistical modelling of interactions may not reflect true

interactions in the data, but rather properties of the statistical method (Greenland, 1993). Again, remedies are possible, but they require more statistical sophistication (see Muthen et al., 2006, as an example).

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Declaration of interest statement

The author has no competing interests.

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