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COMMENTARY ON REPLICATIONS OF MARLATT'S TAXONOMY

Lest taxonomy become taxidermy: a comment on the relapse replication and extension project

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Abstract

In this commentary on the Relapse Replication and Extension Project, several points are addressed. First, the selection of the pretreatment "baseline relapse" as the key predictor variable for post-treatment relapse in the predictive and construct validity studies is questioned. It is doubtful whether retrospective accounts of drinking episodes after 4 days of abstinence qualify as relapse episodes for the following reasons: clients may not have been committed to abstinence (e.g. weekend binge drinkers); treatment may have included coping skill training for pretreatment high-risk situations (i.e. treatment intervention is viewed as a "nuisance variable"). By assuming that a client's retrospective report of a poorly defined pretreatment "relapse" is predictive of the type of relapse episode experienced during the post-treatment follow-up assessment is to commit an error of "taxidermy" in evaluating the reliability and validity of the taxonomy; i.e. the tendency for researchers to "stuff and permanently mount" distal baseline episodes as static predictor variables, thereby depriving them of their dynamic and fluid role as proximal determinants in the relapse process. The difference between researchers who assess distal trait predictors of relapse and clinicians who focus more on proximal state predictors in conducting relapse prevention therapy is discussed and illustrated by means of a case study. Finally, the metaphor of relapse as "falling off the wagon" is described as an example of a systems approach to relapse prevention.

Having now read all the papers in this relapse taxonomy replication project, I would like to take the liberty of offering a few concluding comments. Although I am pleased and flattered that our early work on the relapse process has received such attention and is deemed worthy of replication by the US National Institute on Alcohol Abuse and Alcoholism, I am also concerned that some basic assumptions of the model have been misconstrued and may therefore lead readers to some faulty conclusions based on the findings presented. I make these remarks as the originator of the taxonomy and as a reviewer of these papers. With two exceptions, neither I nor my colleagues at the University of Washington have been personally involved in the Relapse

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Replication and Extension Project. At the beginning of the replication in 1992, I was asked to train researchers from three project sites in the coding of relapse precipitants based on our original classification system (Marlatt, 1996a, this issue). At the end of the replication, I was invited to provide comments on completed studies in 1995 symposia and in this supplement.

Many comments that occurred to me while reading through the papers in the relapse replication project have already been covered in the discussion papers by Donovan (1996, this issue) and Kadden (1996, this issue), and so I will not belabor the points they raised. Rather, I would like to take issue with what I consider to be a critical methodological flaw that runs across several papers that attempt to address the validity of the taxonomy. In my view, it was a mistake to use the so-called "baseline relapse" as the critical predictor variable in studies designed to assess the predictive validity (Stout, Longabaugh & Rubin, 1996, this issue) and construct validity (Maisto, Connors & Zywiak, 1996, this issue) of the relapse taxonomy. By assuming that a participant's retrospective report of a poorly defined pretreatment "relapse" episode should predict the type of relapse episode experienced during the treatment follow-up is to commit an error of "taxidermy" in evaluating the taxonomy. By taxidermy, I refer to the tendency of these researchers to "stuff and permanently mount" distal baseline relapse episodes as static predictor variables, thereby depriving them of their dynamic and fluid role as proximal determinants in the relapse process.

The first problem concerns the definition and assessment criteria for establishing the "baseline relapse" for the predictive and construct validity studies. Here is how the baseline relapse was defined:

> At baseline, relapse was defined as an episode that included a heavy drinking day, i.e., a day on which the subject consumed enough alcohol to achieve a BAL of 10 or higher, based on gender and body weight. This relapse had to be preceded by four days of abstinence (Stout, Longabaugh & Rubin, 1996, this issue, p. S100).

A number of questions and concerns are raised by this definition. Clearly, this is not a prospectively assessed "baseline" event; rather, it is based on retrospective recall of a drinking event that may or may not represent a relapse episode. Given the general concerns about the validity of retrospective self-reports expressed in the literature, it is surprising to find that these investigators relied so heavily on reports of relapses that may have occurred a year or more prior to the intake interview. Recent research on time effects in retrospective reports of relapse episodes confirms this potential source of error (McKay, Rutherford & Alterman, 1996); these authors conclude:

> These findings indicate that the amount of time between the onset of a relapse and when the subjects interviewed can influence reports of experiences prior to relapse and attributions for relapse (p. 37).

Problems associated with such a long period of retrospective recall for baseline relapse episodes is further emphasized by Kadden (1996, this issue) in his comments on the current validity studies:

> In addition, the relapse after four days of abstinence may have occurred long before the interview at which the baseline data were collected, in some cases a year or more in the past. With such variability in the prior occurrence of the baseline relapse incident, there may have been substantial differences in the accuracy or detail of recall of relapse episodes, between those occurring relatively recently and those that may have occurred many months prior the research interview (Kadden, 1996, this issue, p. S142).

Another critical problem with the selection of the "baseline" relapse is that no attempt was made to determine if the patient was in fact committed to abstinence at the time the so-called "relapse" occurred. Recall the assumptions we made stating the conditions for assessing relapse in our model:

> The model applies to cases in which the individual has made a commitment to a period of abstinence, following voluntary termination of the use of a substance such as alcohol ... An important condition of the theory is that it applies only to those cases in which the individual has made a voluntary choice or decision

to change a target behavior (e.g. to stop drinking...). (Marlatt & Gordon, 1980, p. 424).

No attempt was made in the current validity studies to assess the patient's prior commitment or voluntary decision to abstain as a condition of defining the baseline relapse episode. Relapse was thus defined as any heavy drinking day preceded by 4 days of abstinence. Several questions arise concerning the validity of these selection criteria: (1) patients who drink continuously without 4 days of abstinence are necessarily excluded by this definition—in one paper, for example, it was noted that "... 70 subjects were dropped from these analyses because their relapses occurred more than 6 months prior to intake ... the excluded subjects appear to be very severe drinkers, unable to stay abstinent for 4 days in a row before drinking" (Rubin *et al.*, 1996, this issue, p. S115); (2) patients who drink in a weekend binge pattern after abstaining from Monday to Thursday each week would be misclassified as relapsing every weekend; (3) patients who were unable to drink for at least 4 days because of illness, hospitalization or incarceration, and who then drank heavily at the end of this period would also be classified as relapsed, despite absence of any prior voluntary intention to abstain. These methodological shortcomings are noted in passing by the authors of the construct validity paper:

> There are, of course, numerous possible explanations of the general failure to find support for the predictions of this study besides a lack of construct validity of the Marlatt typology ... [one] hypothesis is that the baseline relapse, which was central to the analyses reported in this paper, was not a relapse at all. Therefore, any distribution of precipitant frequencies based on such an event would not be a sensitive indicator in evaluation of a relapse precipitant typology's construct validity. In this regard, it is unlikely that many subjects were making a concerted effort to change their drinking patterns during the period they reported as part of the baseline assessment that the relapse had occurred (Maisto, Connors & Zywiak, 1996, this issue, p. S95).

Too bad that this retrospective observation was not utilized in prospective plans for the research design of the validity studies. At least the authors show a degree of reliability in their selection of the critical baseline relapse episode: they keep making the same mistake over again (Stout *et al.*, 1996, Maisto *et al.*, 1996; Rubin *et al.*, 1996, this issue).

Another faulty assumption is that the pretreatment relapse precipitant should in some way predict the same precipitant for the first posttreatment relapse. This is like a garage mechanic making a prediction that your next flat tire should be caused by an identical puncture in the same tire as the one he just fixed. Let's not get hung up on this level of specificity! First, as noted by Donovan (1996), patients are often vulnerable to a hierarchy of high-risk situations for relapse, and that relapse risk is better predicted by a profile of a particular patient's likely precipitants and a measure of readiness for change. Secondly, this prediction ignores the intervening impact of treatment itself on the patient's improved capacity to cope with high-risk situations experienced after treatment. To ignore the potential impact of treatment on vulnerability to future relapse precipitants is equivalent to defining clinical intervention as some kind of "nuisance variable", a source of error variance. Is it any wonder that many clinicians believe that research is conducted by "ivory tower" academics who have never set foot in the clinical arena?

As a clinical psychologist trained in the scientist–practitioner model, and who operates both as a researcher and therapist in the addictive behaviors field, I wear two hats: the hat of the scientist and the hat of the therapist. When wearing my scientist hat, I can ask a number of questions about predictors of treatment outcome that have been reported in the scientific literature. Most published studies of determinants of relapse rely on statistical comparisons between groups of subjects who differ on the basis of relatively stable traits such as gender, ethnic group, age, family history of addiction or type of treatment received. From the point of view of the objective scientist or statistician, such stable traits can be used to make general predictions about treatment outcome (e.g. young men with a positive family history of alcoholism are less likely to remain abstinent after treatment). In the context of designing and evaluating alcoholism treatment programs, assessment of such stable trait variables is helpful to the extent that it provides useful information in terms of predicting the stability of treatment outcome (assessed

at some fixed interval after completion of treatment, e.g. 6 and 12 months post-treatment). Assessment of historical and demographic traits and other stable background traits provides information about potential distal determinants of outcome.

When I am wearing my practitioner hat, however, my orientation shifts from a consideration of general traits and distal predictors of treatment outcome to the ongoing assessment of individual state variables that help me understand and work with my client's unique proximal determinants of the process of change over time. In the therapy session itself, my focus shifts from consideration of distal trait determinants based on statistical studies of group differences to one of trying to assess proximal state determinants that are specific to my client's ongoing therapeutic process. Proximal state determinants (unlike stable trait predictors) are much more likely to fluctuate and change on the basis of day to day experiences (e.g. exposure to a series of high-risk situations over time, fluctuations in commitment to treatment goals, modification in the practice of adaptive coping skills, changes in environmental or social support and shifts in cognitive variables such as self-efficacy and outcome). As a scientist–practitioner who is formulating treatment plans for a particular client, I often rely on the scientific literature for information about distal trait predictors of treatment outcome; but once treatment is under way, I rely more on assessment of proximal states that are often more accurate in predicting immediate changes in treatment process (e.g. predicting lapses from one therapy session to another). In terms of the ongoing therapy process, assessing such proximal determinants of relapse gives me the information I need to help my client cope more effectively with pending high-risk situations (e.g. planning to handle "what is coming up next week") or to help the client deal with actual relapse crises when they occur (e.g. cognitive restructuring to cope with the "abstinence violation effect").

As an example, I recently served as a therapist for a client, a married woman with mixed problems of depression and alcohol dependence. In a published commentary on this case (Marlatt, 1996b), I described several setbacks my client experienced in her attempts to stop drinking. On one occasion, she accepted a drink (one glass of wine) that was offered to her at a women's literary discussion group. This lapse, associated with a social pressure precipitant did not eventuate in any further drinking. Some months later, however, she drank to the point of intoxication after speaking on the phone to her husband who was out of town on a business trip. During the conversation, her husband criticized her because he mistakenly assumed that she had been drinking during his absence. Upset and angry by this false accusation by her husband, she reacted by drinking almost a whole bottle of sherry and was intoxicated when her husband returned home that same evening. Why did the first precipitant (social pressure to drink) trigger a lapse while the second episode (interpersonal conflict) triggered a more serious relapse?

One hypothesis that predicts this distinction between a lapse and relapse episode is provided by behavioral choice theory (Vuchinich & Tucker, 1996). According to this model, alcoholics are more likely to relapse in situations that signal reduced availability of future rewards in significant life areas (e.g. health and personal relationships):

> This general analysis yields two empirical predictions. First, the likelihood of posttreatment drinking after an event should be directly related to the degree of alcohol-related disruption in the life-health area to which the event is relevant. Second, drinking episodes associated with events should be more severe than those not associated with events. This would be the case because drinking unrelated to events does not involve a change in the availability of rewards in the life-health areas, whereas drinking related to events involves such a change ... lapses and relapses may be differentiated by the environmental conditions (e.g., whether or not the availability of valuable non-drinking activities has been altered) that exist before and when drinking starts (Vuchinich & Tucker, 1996, pp. 21, 25).

Returning to the case example above, my client's lapse did not escalate further relapse because the event (taking a glass of wine at a social gathering) did not signal decreased access to subsequent rewards in important life-health areas. In the second episode, drinking was more severe, presumably because the precipitating event (an interpersonal conflict in which she was accused by her husband of drinking) signaled to

her that she was unlikely to gain any future rewards for abstinence upon her husband's return from the business trip. Here I am in agreement with the conclusion drawn by Vuchinich & Tucker: "Most likely the lapse-relapse distinction depends both on the contextual initiating conditions and on internal processes after drinking begins" (Vuchinich & Tucker, 1996, p. 25).

When wearing the practitioner's hat, the therapist's responses to such "contextual initiating conditions" and on subsequent "internal processes after drinking" are critical. For therapists applying the relapse prevention model in the consulting room, the assessment of dynamic state changes is often more predictive of outcome than static trait variables. Scientist–practitioners can perform "mini-experiments" in the course of conducting therapy. Hypotheses about a particular client's ability to cope with specific high-risk situations can be tested as therapy proceeds. If one coping strategy fails to make a difference in preventing relapse for a particular client, another coping skill can be introduced and its effectiveness can be ascertained. Therapists can also keep track of their clients' progress on a more continuous, session-to-session basis, rather than on relying on periodic outcome assessments conducted at fixed follow-up intervals. In a parallel move, addiction treatment researchers are beginning to turn to "real time" assessment of clinical process variables, including the use of mini-computers to track daily occurrences of urges, lapses and relapses almost as soon as they occur in the client's natural environment (e.g. Stone & Shiffman, 1994).

The popularity of relapse prevention in the addictions treatment arena can be explained, in part, by the clinical utility of the model (Carroll, 1996). Because the model provides a focus and a framework for therapists to help their clients cope more effectively with ongoing changes in the "here-and-now" flow of present experience, it is a flexible and adaptive tool in the clinical consulting room. Therapists can benefit from the knowledge of trait predictors of treatment outcome (e.g. what the general prognosis and criteria are for treatment-matching for an adolescent African-American male who shows a mixed pattern of alcohol and cocaine abuse) obtained during the intake and treatment formulation stage. Once treatment has been initiated, however, clinicians are more likely to rely on proximal state predictors (such as the cognitive and behavioral variables documented in the relapse process model) to guide ongoing treatment.

Clinical utility is not the same thing as predictive and construct validity, particularly as defined by several papers in this replication series. In attempting to test the predictive validity of the taxonomy (i.e. to what extent does a client's "pretreatment" relapse category predict the same category for a post-treatment relapse) the authors are assuming that the taxonomy categories represent stable trait determinants, in and of themselves, to the exclusion of all other essential elements in the cognitive–behavioral model, including the client's coping capacity and associated cognitive and affective responses. It is as though these studies were designed and conducted by statisticians who are oblivious of the dynamic interplay of cognitive and behavioral factors in the ongoing process of relapse and recovery. Sometimes the exclusive wearing of one hat serves as a blindfold if the brim is too low.

If exposure to the same high-risk trigger pre- and post-treatment does not predict relapse, what does? According to our original model, it is not the mere exposure to high-risk situations, but the individual's capacity to cope with these situations without drinking that is the key predictive variable. As such, assessment of the patient's actual coping abilities (e.g. Chaney, O'Leary & Marlatt, 1978) or ratings of self-efficacy (perceived capacity to cope with upcoming high-risk situations) have been found to be the most reliable proximal determinants of posttreatment relapse (Marlatt, Baer & Quigley, 1995). The finding that coping skills, and not mere exposure to high-risk situations, best predicts subsequent relapse is also supported by findings reported in this series by Miller and his colleagues, who tested the predictive validity of six domains described in the Marlatt & Gordon (1985) model:

> In examining six domains of predictor variables, we found that the occurrence of negative life events was unrelated to the likelihood of subsequent relapse. All five other domains were significantly predictive of relapse, and among these the clients' coping resources proved to be most predictive (85% hit rate in predicting relapse from this domain alone). This suggests that it is not the mere occurrence of potentially stressful events, but the

client's resources for coping with them that predict whether or not the client will relapse to drinking" (Miller *et al.*, 1996, this issue, p. S169).

I would like to close this paper with a final brief commentary on Miller's paper in this series, entitled "What is a relapse? Fifty ways to leave the wagon" (Miller, 1996, this issue). Although the title draws upon Paul Simon's lyric, "There must be fifty ways to leave your lover", the real topic is alcohol relapse, or "falling off the wagon" as the old saying goes. The source of this colloquialism dates back to the American civil war, when troops carried their water supply in a horse-drawn wagon, the "water-wagon". To "fall off the wagon" implied that the soldier had a "fall from abstinence" (drinking only water) and started drinking alcohol, presumably obtained from a wagon containing alcohol (the whisky or beer wagon).

It is helpful in this regard to imagine the water-wagon as a system, consisting of the wagon and its water content, a horse to pull the wagon, and a soldier in charge of driving the wagon to accompany thirsty troops in battle. Although there may be "fifty ways" to leave the wagon, these ways would seem to fall into three main sets of determinants: something breaks down in the wagon itself (e.g. the wagon is driven into a pot-hole and a wheel axle is broken); the horse is unable or unwilling to pull the wagon; or the driver "falls off" the wagon or is otherwise impaired. Putting this in terms of the relapse prevention, the goals here are to (a) do what can be done to keep the water-wagon, horse and driver on course and in good running order; (b) if the wagon does break down and the driver falls off, to do what is necessary to fix the problem and to help the driver get the wagon back on track and under way again.

The route of the water-wagon takes it through unknown territory marked by a variety of high-risk situations that may cause either a transitory problem (lapse), a more lasting and serious setback (relapse) or a total breakdown (collapse). The key to successful water-wagon maintenance is to work with the driver—to provide him with route maps that chart out difficult terrain ahead and provide navigational tips (e.g. documenting alternative routes), to train him in basic mechanical skills to repair problems with the wagon should they occur en route (e.g. teaching him to mend a broken wheel) and to help him work with unmotivated or uncooperative horses (e.g. if there is no "horsepower" to keep the wagon moving).

What is most helpful for the driver (and for those trainers and therapists who provide assistance) is a combination of elements, including information and knowledge (e.g. road maps indicating dangerous and safe routes), motivational strategies (e.g. getting the horse moving again by employing a "carrot-and-stick" strategy) and coping skills to prevent or cope with breakdowns (e.g. experience in keeping the axles oiled or fixing a broken wheel). Helping the driver learn from his mistakes and to get back on the water-wagon instead of deserting it for the whiskey-wagon is another critical element in successful wagon recovery.

No experienced wagon-master would predict that whatever caused a breakdown in the past (be it a problem with either the wagon, horse or driver) would be exactly the same factor responsible for all future breakdowns. What is most helpful is awareness of potential breakdown precipitants (so as to prevent lapses), a flexible coping repertoire that can adapt to changing circumstances on the road, and an optimistic "can do" attitude (although exposure to future high-risk situations is inevitable, self-efficacy remains high). These successful maintenance factors depend on the driver's ability to respond to the ever-changing conditions of the road, the horse, and the wagon. In other words, ongoing maintenance or relapse is governed more by the driver's adaptive ability to respond to changes in proximal states than by the impact of distal traits such as the driver's genetic endowment, the color of the horse or the wagon's prior breakdown history. The bad news is that there may indeed be fifty ways to leave the wagon. The good news is that there are fifty corresponding ways to get back on.

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