

ISSUES IN TESTING LINDESMITH'S THEORY<sup>1</sup>

*Were we attacking "a theory of conscious motivation?"*—Lindesmith's seizing upon our use of the word "motivation" to contend that we mistook his theory for one of *conscious* motivation enables him to raise methodological problems concerning consciousness, to question the relevance of our work to his, and to pave the way for an argument that our "pleasure motive . . . involves a confusion of effects with causes." We deal with the first two issues here and the third in the next section.

Our use of "motivation" at many points followed standard practice in psychology, where the word does not entail consciousness. Although we could easily have dispensed with "motivation" and adhered to a "hard" behaviorism, we did not for two reasons. First, self-reported motives can be valuable clues to the relevant response-consequence contingencies. Therefore, when we presented evidence that euphoria is consciously desired, and so forth, we were alleging the importance and occurrence of *positive reinforcement* rather than arguing that Lindesmith's was a theory of conscious motivation (see our definition of "euphoria," in McAuliffe and Gordon [1974 p. 800]). It is the difference in the role we attach to positive reinforcement that challenges his theory, not any difference concerning the concept of "motive." Second, we used motivational terminology to parallel Lindesmith's use of this language in his own work. For example, ". . . the most obvious characteristic of the addict's behavior is his intense desire . . . for the drug. This desire . . . is a *powerful conscious motive* driving him to seek satisfaction . . ." (Lindesmith 1968, p. 49; our italics). In Lindesmith's work, conscious motives are equated with "craving," which leads to the frequent use of opiates (1968, pp. 55, 56). Finally, he holds that "the craving . . . is fixed by negative rather than by positive reinforcement . . ." (1968, p. 95). It was at this point that we took issue with him.

Lindesmith has also used the concept of "motives" to refer to all reasons individuals give for becoming addicts other than those that stem directly from drug use (1968, pp. 16–17). He has explicitly denied that his is a theory of motives in this sense. The present controversy, however, concerns variations in motives due to variations in drug-derived reinforcement. Our paper dealt with Lindesmith's treatment of motives in the latter category, not the former.

A cognitive theme in Lindesmith's work would also seem to support

<sup>1</sup> The full 50-page version of this rejoinder (abstracted in McAuliffe and Gordon 1975), including two tables, can be obtained by ordering manuscript no. 866, by McAuliffe and Gordon, from the Journal Supplement Abstract Service, American Psychological Association, 1200 Seventeenth Street, N.W., Washington, D.C. 20036, for \$3.00 (payable to American Psychological Association).

his claim that his is not a motivational theory (1968, pp. 8, 73, 80). This theme emphasizes the recognition that opiates have been taken to relieve withdrawal distress as the critical event in the addiction process. Here, we agree with others that even in cognitive psychologies "motivational variables remain hidden" (Murray 1964, p. 4; Scheerer 1954, p. 91). Motivational terminology is not incompatible therefore with criticism of Lindesmith's cognitive analysis, since in his terms we would disagree over what constitutes the full set of cognitions. Recognition that the drug gives pleasure is as much a learning experience as the fact that it brings relief.

*The role of euphoria in chronic addiction.*—Our paper focused on the role of euphoric effects after the user first realized his physical dependence, because Lindesmith himself had identified continued drug use at this stage as "the central theoretical problem of addiction" (1968, p. 33). Our position was that he was mistaken in according no causal significance to euphoria in explaining *continued* drug use. His main argument in immediate support of this position is contained in numerous passages which state or imply that euphoria "vanishes" (Lindesmith 1965, pp. 128, 136; 1968, pp. 9, 31–33, 78, 79, 84, 94–95, 132, 137, 176, 177; Lindesmith and Strauss 1968, pp. 194–95). Those familiar with his works will recognize that no mere count of citations can adequately convey the total effect of these passages in driving home Lindesmith's claim that "addiction is determined by . . . withdrawal symptoms . . . rather than . . . positive euphoric effects often erroneously attributed to its continued use" (1968, p. 94–95).

Now Lindesmith asserts that our claim that he stated addicts do not experience euphoria is false, because he had acknowledged that they experience pleasure from the impact effect. But this inclusion of the impact effect as euphoria represents a substantial change from his prior stance. Although he did indeed indicate that impact effects were gratifying, he consistently distinguished euphoria from the "physical thrill" of the impact effect (1968, p. 33; see also pp. 34, 38), argued that this gratification was merely a mistaken interpretation of relief from oncoming withdrawal (1968, pp. 40–41, 89, 91), and omitted mention of the impact effect when discussing positive reinforcement. Consequently, we had to establish in our article that it actually was a euphoric effect (pp. 801–2, 814–15). Thus, either he was inconsistent in his previous work or we were justified in our claim concerning his treatment of euphoria.

The presence of any positive effects in the postdependent stage is damaging to a theory which freely grants their causal efficacy in the predependent stage (Lindesmith 1968, p. 84) while insisting that postdependent use depends only on "negative rather than positive reinforce-

ment" (1968, p. 9). This potential contradiction undoubtedly accounts for Lindesmith's separate treatment of the troublesome impact effect.

Although he now concedes the postdependence presence of euphoria in the form of the impact effect, Lindesmith still denies its causal role by arguing that causality operates in the wrong direction: "Causal significance cannot be attached to experiences that follow from addiction instead of preceding it." (His complaint about the nod is dealt with in our next section.) His argument harks back to earlier work, where he suggested that the addict's reports of pleasurable impact effects are "simply a reflection of the addict's craving; . . . he enjoys the drug . . . because he desires [craves] it so intensely rather than because it is inherently enjoyable" (1968, p. 41; see also 1965, pp. 123-24). Lindesmith's contention (which may actually be merely a definition) that craving is present only after physical dependence is already "established and recognized" confines the impact effect to the postdependent period and implies that the causal ordering is opposite to what an explanation involving positive reinforcement requires, since addiction already exists.

His argument concerning the causal role of craving derives from what he considers the requirements of "a general theory," which are implicit here but explicit in his book (1968, chap. 1). There he states: "It was assumed that the essential causal process of addiction would be found in all cases of addiction and that it would not be found in any case of non-addiction" (1968, pp. 14-15), and "the assumption made at the outset of this investigation [was] that the craving for drugs is generated in one identifiable, unitary type of experience" (1968, p. 13). Since there are some cases of addiction in which euphoria may be absent, Lindesmith apparently inferred ("This reasoning led me to the conclusion . . .") that the "craving" must be connected with negative reinforcement because this is the only source recognized as universal in his definition of "addiction," that is, the only one meeting his requirements for a general theory. Hence, when he refers to cases of addiction following medical treatment, the supposedly parochial nature of contemporary street addicts, the supposed absence of the impact effect under other than intravenous administration, and the sometimes noneuphoric reactions to opiates by normal subjects, he is relying more than ever on those arguments against the universality of euphoria that remain in the aftermath of our demonstration that euphoric effects were experienced by postdependent addicts to a far greater degree than he had granted. In Lindesmith's eyes, these arguments disqualify euphoria from meeting his requirements for a general theory.

A notable feature of this use of "craving" in Lindesmith's work is the lack of any empirical effort to determine whether it is entirely a unitary phenomenon unaffected by the degree to which euphoria is or has been

experienced, and whether it is present to any degree prior to physical dependence. For example, he could have investigated whether impact effects are present prior to physical dependence, since he regards them as caused by craving. In the absence of any such efforts, Lindesmith's inferred causal ordering is simply too self-serving to command assent (as well as inconsistent with facts described below). Moreover, by its very logic, his inferential model does not admit the possibility of a second cause no matter how influential it might be in any proportion of cases less than the total. The "universality" argument, therefore, begs the question concerning the potential relevance of the kind of cause that is at issue. However, it may be of interest to point out that the generality he demands is achieved at the next higher level of abstraction by the concept of "reinforcement," which embraces both positive and negative effects.

Positive effects have been shown to reinforce opiate use (Claghorn, Ordy, and Nagy 1965), and since it can be shown that they are present before and after dependence, it would follow that the reinforcement they produce is also present at these times, contributing to continued use and other forms of addict behavior. When euphoric effects are not felt, consequent behavioral patterns are either diminished or absent, but drug use itself may not cease if there is reinforcement from some other source. The discovery of cases in which drug use persists without any euphoria thus proves nothing about the power of positive effects. The question is not whether positive effects can cause drug use by addicts but, rather, in what proportion of cases and to what extent they are important.

The purely empirical aspects of Lindesmith's continued argument again the causal importance of euphoria can be summed up in two stages. Predependence euphoria exists only in the form of the nod, and this euphoria is too inconsistent and unimpressive to produce "craving." Post-dependence euphoria exists only in the form of the impact effect, which is strong but of greatly limited generality because supposedly it is present only among users of the intravenous method. Even then, the impact effect is present only after the onset of addiction and therefore out of causal sequence. Finally, the impact effect is probably only a sign of relief. However, there is firm evidence against all of these contentions.

The presence of the impact effect in predependent and currently non-dependent users addresses many of these issues. In Saint Louis one of us had the good fortune to encounter and interview a college student shortly after he had first tried heroin; the first effect he had experienced was an intense "rush." In Baltimore, two nondependent postaddicts who had begun to use opiates again gave the impact effect as an important reason for their current use. Similar observations of effects recognizable as impact effects, occurring in either nonaddicts or postaddicts, have been

reported elsewhere (Aldrich 1969, p. 462; Jacobs 1974, p. 61; Powell 1973, pp. 589, 591; Seevers and Pfeiffer 1936, p. 175; Wikler 1952, p. 277). A description by an addict of her very first opiate dose indicates how potent these effects can be: "The rush was terrific. . . . It practically knocked me off my feet. My head reeled so I could hardly even sit up, let alone stand or walk . . . an overwhelming experience. . . . I thought it was fun, a really wild sensation. . . . My attitude was like, 'Wow, this stuff sure does it to ya! Spectacular'" (Jacobs 1974, p. 61). Finally, injections of cocaine and amphetamine, drugs which produce no physical dependence, nevertheless cause impact effects (Cox and Smart 1972).

Kolb (1925, p. 706) indicates that impact effects can be obtained from other than intravenous administration, contrary to Lindesmith's claims. "Early orgasmic effects" under subcutaneous administration were noted by Martin and Fraser (1961, p. 393); Chessick (1960, p. 545) implicates intranasal sniffing; and Angle et al.'s (1973, p. 439) observations suggest impact effects even from oral ingestion of methadone. Lindesmith himself implies their availability from other routes (1968, p. 255).

Now let us turn to Lindesmith's remaining evidence concerning the degree to which euphoric effects are problematic in early opiate use. He himself has stated (1968, p. 180), "Almost all persons who had had morphine injections with whom I discussed the matter described the effects as pleasant, and some . . . intensely so. . . . If allowances are made for some unpleasant effects with the very first injections, the vast majority experience the initial effects as pleasurable." In the same work (pp. 24-25), he also explained that unpleasant side effects usually disappear after a few doses, and euphoria becomes more intense. If pleasurable effects fail to result from subsequent doses, "such persons . . . do not, of course, become addicted." Now, however, he gives unqualified approval to reports by Beecher "which bear out the conclusion that [normal] subjects are on the whole not impressed by opiate effects and commonly react to them with indifference or . . . dislike."

Actual evidence accords better with Lindesmith's earlier position. It can be shown (McAuliffe and Gordon 1975) that the results described by Beecher do not generalize to addict populations. Pooled data from studies by Chein et al. (1964), Waldorf (1973), Willis (1969), and us show that 65% of 687 addicts experienced euphoria on their first dose. In Waldorf's sample ( $N = 422$ ), 63% felt euphoria on the first dose, 90% by the fifth dose, and 98% eventually. Unfortunately, studies of heroin users not yet physically dependent are rare. But the material known to us fits the pattern. Our interviews with seven college students who had tried heroin only once or twice revealed that five had experienced pleasant effects. In a study by Powell (1973), euphoria emerged spon-

taneously in five of 12 interviews as the respondents' reason for using heroin, although the questioning was not even directly concerned with euphoria (subjects 2, 6, 8, 9, 11). An occasional user similar to Powell's users described heroin to us as the "very best thing I ever experienced" despite extensive use of other drugs (see also Jacobs 1974, p. 68). We have shown (1975) that the differences between these results and Beecher's are explained by interactions among subjects, circumstances, and drugs.

*Evidence that addicts nod.*—Because addicts sometimes use "high" in a loose way to refer to euphoria generally, that is, to include both "rush" and "nod," Lindesmith accuses us of "lumping" the impact and continuing effects when we questioned addicts using the word "high." He claims, therefore, that our findings cannot be used to test the presence of the nod. We are surprised that Lindesmith fails to recognize that, although addicts occasionally use the term "high" in this more general way, it refers primarily to the continuing effect. "High" may mean either the impact effect and the nod together or just the nod, but it does not refer to the impact effect alone, as Lindesmith implies. Lingeman's dictionary indicates that "high" means the continuing effect and explicitly distinguishes it from the "rush" (1969, pp. 102–3, 214). Lindesmith himself has used it this way (Lindesmith and Strauss 1968, p. 195).

In part, the semantic association between the two terms follows from the empirical association between the two effects themselves, since a shot sufficient to produce a satisfying impact effect will also produce nodding. The opposite does not hold. Oral doses, for example, often produce nodding without an appreciable impact effect. These considerations afford little basis for Lindesmith to challenge the import of our data. Nevertheless, since we failed to anticipate this ambiguity by asking addicts about the continuing effect explicitly in terms of the nod, we present empirical evidence that this major effect, whether called "nod" or "high," does indeed occur.

First, the long-term addicts we studied have often nodded heavily before our eyes. Addicts we have known personally and even lived with were observed nodding deeply. Sometimes nodding occurred during our interviews. Other interviewed addicts occasionally employed the term "nod" spontaneously to describe their experience.

Second, four separate laboratory studies of addicts give evidence of their nodding and enjoying euphoria (Fraser et al. 1963; Martin et al. 1973; Martin and Fraser 1961; Wikler 1952).

Third, two studies report nodding by chronic addicts outside the laboratory (Bloom and Sudderth 1970, p. 469; Rathod, de Alarcon, and Thomson 1967).

Fourth, our data from two surveys of chronic addicts from the streets of Baltimore also support the presence of the nod. In these, addicts

rated the "importance" of the continuing ("To get high") and impact ("For the rush") effects as reasons for their taking heroin or for the deluxe ratio, where the term "high" was employed to represent the continuing effect. Consistent, significant differences favoring the importance of "To get high" indicate clearly that, even if all addicts construed "high" to refer to *both* effects, there must be something more involved in getting high than the impact effect alone. Only 5.4% ( $N = 55$ ) and 7.9% ( $N = 63$ ) of addicts in the two surveys rated the impact effect more important to any degree than the "high." Cells in which the addict could rate one of these items "very important" but the other "not important" are of special interest. Since shots that produce a rush will also produce a nod, whereas methods for producing a nod may not produce a noticeable impact effect, one combination of these extreme responses would seem more likely than the other. Indeed, 9.1% and 9.5% of addicts responded in the pattern favoring the extreme importance of getting high, but no addict in either survey ever said the impact effect was very important while claiming that the high was not important at all. Lindesmith's insinuation that the high referred only to the impact effect in our data is severely strained by these patterns. If the meaning of "high" embraced both the impact and continuing effects for some or all addicts but responses were actually based only on the presence of the impact effect, the cell representing the extreme pattern favoring the high and devaluing the impact effect ought not to contain any responses. As it turns out, it is the opposite pattern that yields no responses—twice.

*Understanding Martin and Fraser (1961).*—Lindesmith calls attention to passages from this study that appear to invalidate our claim that it "verified . . . heroin is . . . more euphorogenic than even morphine" (McAuliffe and Gordon 1974, p. 809), which we used to underscore a point concerning the preference for heroin over methadone. To assess whose interpretation is correct, it is necessary to know that the study was concerned with determining whether heroin provided a *qualitatively* different kind of euphoria, in view of the fact that it also established heroin as being 2.26 times as potent as morphine in producing various effects, including euphoria. Accordingly, doses of the two drugs were adjusted so as to be equipotent (and thus, in effect, equi-euphoric), in order to control for differences in euphoria due to differences in potency. Any remaining profile differences over various reactive measures would represent interactions or "side effects." This design is standard in medical evaluation of alternative therapeutic agents.

Despite this equating for potency, results showed that heroin still elicited from addicts free responses indicative of euphoria more often than did morphine (82 reports versus 57). The authors' own conservative interpretations fail to take into account the special nature of their design

and fail to recognize the full number of responses in their table 4 that could count as "euphoric" ones. In the passage quoted by Lindesmith, they should have added that preference for heroin on the street may be due to its greater potency, since their own results confirm this greater potency.

Lindesmith's misleading reference to an ostensible five-to-three "vote" barely in favor of heroin fails to mention that this was a repeated-measures design, with each drug given blindly for 19 days. Each day, subjects were asked, "Would you like to take the drug daily?" Two subjects indicated maximum acceptance toward both drugs for all 19 days, a third subject did so for 18 days, and the remaining five "showed a decided preference for heroin" (1961, p. 393), a difference the authors themselves were respectful of. Thus, there was practically no sign of preference for morphine over heroin but considerable evidence of preference for heroin over morphine. Other differences from equipotent doses were also slightly but consistently in favor of heroin. Consequently, they would suffice to support a preference for heroin by the experienced user, just as a slight difference in odds justifies consistently taking the more favorable gamble.

*Concerning animal studies.*—Since Lindesmith gives no reason why our reinterpretation of key animal studies is "unconvincing," we rest with our earlier statement. His complaint concerning Wikler stems from misreading our reference to Nichols cited as "Nichols, in Wikler 1968," on our page 835.

*Concerning historical shifts and what we actually said.*—Lindesmith attributes to us the claim that our theory is superior to his because ours accounts for historical shifts from morphine to heroin and to intravenous use. We claimed nothing of the sort (see our pp. 801, 809–11), and it clearly was not our intention to give an account of the appearance of either heroin or the intravenous method. Instead, our use of historical materials was limited to establishing that the impact effect, which is most salient under intravenous administration, is a euphoric effect (p. 801) and that preference for heroin is due to its greater euphoric potential rather than to an autonomous subcultural value (p. 809). However, we are now pleased to acknowledge that the most widely accepted accounts of the adoption of the intravenous mode and of heroin by American addicts contain the assumption that addict behavior is reinforced by positive drug effects—the main theme in our criticism of Lindesmith. Thus, in accounting for the diffusion of the intravenous method, O'Donnell and Jones (1968, p. 127) reason that "the addict who was consciously using drugs for pleasure would be most likely to note and adopt a more pleasurable technique." As for the diffusion of heroin, there are key omissions in Lindesmith's version of the explanation he attributes to Terry and

Pellens. First of all, they distinguish a number of factors leading to the spread of heroin (1970, pp. 84–86, 473, 484–85). Convenience to the peddler, which Lindesmith stresses, is but one of these. Second, this became a consideration only after passage of the Harrison Act in 1914 (p. 86). Previously, heroin use had spread in underworld circles because heroin “seemed to fill a place not easily duplicated by other preparations in the desires of the vicious user. The stimulating effect of heroin is known to be greater than that of morphin and this quality . . . is a sufficient reason for its preference as a drug of dissipation” (Terry and Pellens 1970, p. 484; see also Brown 1966, p. 43).

*Typographical error.*—On page 825, the word “hope-friends” should read “hope-fiends.”

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## RACE, CLASS, AND SUCCESSION: REPLY TO MAGUBANE

In his review of *The Colour of Class on the Copper Mines* (AJS [September 1974]), Mr. Magubane consistently fails to address the contents of the book. As soon as he approaches such a confrontation, he descends into unsubstantiated assertions followed by abuse and sloganeering. While I find his dogmatism vacuous and his radical invective harmless, I cannot ignore his penchant for distortion and fabrication. We have, in short, yet another masterpiece of Magubane's idiosyncratic brand of criticism.

Magubane's method is based on the assumption that most of his readers have not read very carefully the works of those he attacks, or, with luck