Primary Prevention: Should We Support Both Practice and Research?

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The February 16, 1983 debate on Primary Prevention in which Albee and Goldston met Lamb and Zusman underlined a number of issues that are repeatedly discussed in such forums. In response to an invitation to comment on the debate by the Florida Mental Health Institute, I would like to focus on five of these issues. I should state at the outset that my comments are far from unbiased, as I am clearly an advocate for prevention intervention and prevention intervention research (Munoz & Kelly, 1975; Munoz, 1976; Christensen, Miller & Munoz, 1978; Munoz, Snowden & Kelly, 1979; Munoz, 1980; Munoz, 1982). In my discussion, I will try to: 1) identify some premises which were not sufficiently emphasized; 2) make such premises clear so that, hopefully, any further discussion is based on shared (or at least well-identified) starting points; and 3) add my perspectives in each of these areas. I will use examples dealing with affective disorders throughout my discussion, because they are among the most prevalent of disorders, and because they are present in various levels of severity.

The Target of Prevention Intervention:
Severe Disorders vs. Psychological Dysfunction

Are prevention efforts in mental health only to be directed toward diagnosable and severe disorders, or can one have true preventive interventions which are targeted to a reduction of symptomatology which may not meet criteria for a diagnosis? Lamb and Zusman advocate a focus only on the most severe disorders, such as schizophrenia. Albee and Goldston imply that DSM III is not only a temporary diagnostic system, but that there are many diagnoses within it which are not as severe in their consequences as some conditions which may not be diagnosable. In the affective disorders area, it has been pointed out by epidemiologists that there are at least three groups of identifiable expressions of depression: self-reported

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depressive symptomatology, non-bipolar depression, and bipolar disorder (Boyd & Weissman, 1982; Weissmann & Boyd, 1983). Lamb and Zusman would limit our efforts to the severe non-bipolar and the bipolar disorders, Albee and Goldston would address the entire range of depression.

It should be pointed out that the prevalence of the severe affective disorders is relatively small, while mild to moderate levels of depression are quite prevalent. Thus, ongoing, low level depressed mood states are certainly more widespread in the population, and are likely to have a more pervasive influence on communities than the less frequent, if more individually disruptive, major episodes. The state of the empirical evidence regarding prevention of depression is as follows:

1) There are no controlled, experimental studies completed to date testing whether the most severe depressive disorders can be prevented;
2) Prevention practitioners reason that if levels of depression are kept low, people are less likely to develop full-blown depressive episodes. There are encouraging studies showing that the symptomatology of depression can be reduced in non-clinical populations (Muñoz, Glish, Soo-Hoo & Robertson, 1982; Tableman, Marciniak, Johnson & Rodgers, 1982);
3) There are no studies comparing the differential effects on a community of programs focused on only the severe end of the spectrum versus programs focused on the entire range.

Given this state of affairs, more research is needed in all three areas. However, if funds are to be limited, then it would seem most prudent to pursue research in the area which early results indicate has the greatest probability of producing positive results. If preventive research is to be constricted and focused, I recommend that further studies be carried out to further document the early positive findings on intervention focused on depressive symptomatology, and that attempts be made to measure the effects of such interventions much more widely that on just the mood of the individuals who take part in these programs (that is, the effects on their families, job, health, legal problems, and so on). In addition, such programs should also include measures of differential incidence of diagnosable levels of depression, to test the assumption that controlling depressive symptoms will reduce the number of new “cases” that meet DSM III criteria.