Blue Mood, Blackened Lungs

Depression and Smoking

The notion of “self-medication” is one of the most intuitively appealing theories about drug abuse. According to this hypothesis, drug abuse begins as a partially successful attempt to assuage painful feelings. This does not mean seeking “pleasure” from the use of drugs. Rather, individuals predisposed by biological or psychological vulnerabilities find that drug effects corresponding to their particular problems are powerfully reinforcing. For example, clinical observations suggest that persons who have difficulty regulating mood may be particularly vulnerable to abuse of cocaine or other stimulant drugs, persons who have difficulty controlling anger to abuse of heroin and other opioids, and persons who have high anxiety levels to abuse of alcohol and other sedatives. Many drug abusers report that their drug helped them to feel “normal,” even before they developed physiological dependence with continued drug use necessary to prevent withdrawal symptoms. See also pp 1541 and 1546.

Such clinical observations are supported by the recent discoveries of specialized neuronal membrane receptor sites for several classes of drugs with abuse potential, suggesting that the abused drugs are substituting for inadequately functioning normal biological processes. Evidence for genetic vulnerability to certain forms of drug abuse, eg, alcoholism, also fits the hypothesis. The tragedy, of course, is that exogenous drugs overwhelm the normal function, and the beneficial effects sought by the user become outweighed by the adverse effects of drug abuse. Eventually, avoidance of withdrawal symptoms may become the major factor in continuing drug use.

Although the self-medication hypothesis is gaining considerable support (New York Times. June 26, 1990:C1), several aspects of it need careful evaluation:

First, there is the problem of distinguishing among causes, consequences, and the co-occurrence of unrelated disorders. There is increasing evidence of high rates of diagnosable psychiatric disorders among adult and adolescent patients in treatment for drug abuse. Since treatment samples might be biased toward persons with the increased clinical problems resulting from “dual” diagnoses, it is even more impressive that emerging results from carefully designed surveys indicate a comorbidity rate for mental disorders about 50% among persons with “primary” alcohol abuse diagnoses (Goodwin FK. ADAMHA exp abuse efforts. US Med. January 1990:54). However, determining what constitutes a “primary” diagnosis can be a difficult proposition. For example, the relationship between depression and alcoholism. Depression during alcoholism varies from 30% to 70% depending on the stringency of diagnostic criteria, but symptoms usually clear if abstinence is maintained only 5% to 10% of alcoholics with primary affective disorders. Thus, the acute and chronic effects of substance produce the symptoms of mental disorders; sorting from secondary disorders, whether based on development or relative importance, can be very difficult, usually requires longitudinal observations.

A second, clinically relevant problem is that the self-medication hypothesis can lead to an unproductive question of why the individual is abusing drugs, sometimes as a rationale to continue the abuse until the “why stopped. To the contrary, successful treatment of drug patients with other psychiatric symptoms usually addressing the drug abuse initially to achieve, then carefully assessing and treating whatever symptoms remain. Returning to the example of depressed alcoholics, a recent study found that antidepressant treatment is not appropriate for depressed alcoholics whose depressive symptoms persist after 4 weeks of abstinence.

Finally, if self-medication does play a role in when and how does it operate? Do vulnerable individuals make drug effects initially, or are they just more pronounced from experimental use through regular use? Once abuse is full-blown, does self-medication play a role in drug craving beyond the attempt to withdraw symptoms? Is self-medication always drug abuse?

Important empirical results pertaining to self are provided by two studies in this issue of The General report, it is the single most important;
cause of death in the United States, constituting the biggest public health problem in our country.

Analyzing data from a diagnostic interview community survey in the area of St Louis, Mo, Glassman et al 1 found a strong association between cigarette smoking and a lifetime diagnosis of major depression, defined as persistent depressed mood for 2 weeks or more plus at least four additional depressive symptoms (eg, appetite and sleep disturbance).2 Except for alcoholism, no other psychiatric diagnoses were associated with smoking, though not all disorders (eg, schizophrenia) were tested. Smoking cessation rates were also significantly lower for persons with a history of major depression.

From a national database including a self-report scale of depressive symptoms, Anda et al 3 found that smoking rates increased and success in quitting decreased as depression symptom scores increased. Considered together, these two studies make a strong case for an association between persistent cigarette smoking and depression, an association that may become increasingly apparent as the social acceptability of smoking continues to wane and evidence of adverse health effects from both active smoking and passive smoke exposure continues to mount.

As previously suggested by Hughes,4 the following explanations could account for a depression-smoking association: (1) self-medication of depression with nicotine, (2) a common predisposition (eg, low self-esteem) to both depression and smoking, or (3) linkage of predisposing genes. A fourth possibility, that smoking leads to depression, is not ruled out by these studies in the absence of chronological data, but it seems less plausible, even though failed attempts to quit could certainly lead to depressed mood in some smokers. The self-medication hypothesis fits these data well and is also supported by a previous report that depressive symptoms in adolescence predict higher cigarette-smoking rates in young adulthood.5 Further research will be required to determine whether depression increases smoking initiation (perhaps, as suggested by Anda et al, abetted by advertisements implying that smoking improves well-being), but the current results show clearly that depression makes smoking cessation more difficult.

These two studies have important implications for both depression and smoking. They provide further documentation of the pervasive health effects of depression, adding to recent findings from the RAND Corp’s Medical Outcomes Study 6 that patients with either a depressive disorder or depressive symptoms had impairments in physical, social, and role functioning as bad or worse than those reported by patients with chronic physical conditions. The results of Anda et al suggest that persons with depressive symptoms not meeting the full criteria for a depressive disorder are at increased risk for smoking; the prevalence of smoking increased in a "dose-response" fashion as depressive symptoms increased. This also fits with the finding of the Medical Outcomes Study that depressive symptoms, as well as full-blown depressive disorders, were associated with functional impairments. The association with smoking (along with high morbidity, 7 risk of mortality from suicide, and an increasing prevalence in the United States) adds yet another reason to improve the poor detection of depression by primary care physicians, particularly since effective treatments are available.8

The two studies also cast new light on the huge public health problem of cigarette smoking and the difficulties of smoking cessation. A July 1990 Gallup poll found that 27% of American adults continue to smoke, though 7% said they would like to quit, mainly because almost all of the smoke (93%) agreed that smoking is harmful to health (Am J Prev Med 1990;6:190-5). Applying the finding of Anda et al that about 20% of smokers have high depressive symptom scores, this means that depression may be a contributing factor to the smoking of more than 10 million Americans, or 40% less likely to quit smoking than are nondepressed smokers.9

The scope of the problem is staggering. Given the enormous value of smoking cessation for prevention of morbidity and mortality, controlled trials are needed to test the clinical observations of Glassman et al that antidepressant drugs can moderate the adverse impact of a history of depression on smoking cessation by preventing the development of depressive symptoms.

At this point, it would be premature to recommend routine use of antidepressant treatment to assist smoking cessation for smokers with a history of depression. However, physicians should now be alerted to inquire about depressive symptoms and to consider appropriate treatments for depression when smoking patients are unable to maintain a smoking-free lifestyle. Evaluation and treatment of depression may become essential aspects of the role physicians need to play in the campaign to achieve a smoke-free society.10

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