

Smoking and Stimulant Abuse in Schizophrenia:
An Examination of the Causes and Effects

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ABSTRACT:

Examines the causes and effects of stimulant and nicotine usage in schizophrenics. Compares three schools of thought on the smoking-schizophrenia connection: smoking causes schizophrenia; smoking out of boredom; smoking to ameliorate side effects of medications. Considers the shortcomings of current treatment modalities in drug abuse and smoking cessation where psychotic clients are concerned. Discusses possible solutions for problems related to smoking and stimulant abuse in schizophrenics for the future, including medication advances.

INTRODUCTION:

It has been well established that tobacco smoking is the most preventable cause of death in the United States (Watkins, et al, 2000). Lung cancer is the most common type of fatal cancer, and cigarette smoking is the primary cause of that. Changes in societal norms, taxes and laws aimed at reducing smoking, and media and school messages aimed at smoking prevention have helped to reduce the prevalence of smoking down to about 25% of people today (Watkins, et al, 2000; Eden Evins, et al, 2001).

Yet, among the schizophrenic, the rate of smoking approaches 90% (Eden Evins, et al, 2001; Tidey, et al, 1999; Tracy, et al, 2000). In addition, most schizophrenics smoke high-tar, high-nicotine cigarettes, smoke a far higher number of cigarettes than non-smokers, and tend to smoke in a fashion that extracts greater nicotine from cigarettes (McChargue, et al, 2002).

This is unique among the mentally ill. The rate of cigarette smoking is higher among most classes of the mentally ill (approximately double the norm, 50% or more), including those dealing with depression, anxiety, bipolar disorder, and most other mood disorders. The rate is such that half of all cigarettes may actually be used by the mentally ill (Petit-Zeman, 2002). However, in no other psychiatric disorder is the rate anywhere near what it is with schizophrenics (Meadows, et al, 2001; Tidey, et al, 1999).

Substance abuse in general is also much higher among the schizophrenic. Pencer & Addington (2003) suggest that as many as 60% of schizophrenics use illicit drugs. Yet while their symptoms tend to be ameliorated by the use of tranquilizers, schizophrenics

generally abuse stimulants, some abuse hallucinogenics (including marijuana), and few abuse opiates (Wheatley, 1998).

Considering that nicotine is a stimulant and that stimulants are by far the most commonly abused substances by schizophrenics, it would appear that the abuse of stimulants would result in a worsening of symptoms, and in many cases, this is true. However, there are other effects of these stimulants, and particularly of nicotine, that may actually improve the functioning of the schizophrenic, and as such, some psychiatrists are now even questioning the need for a schizophrenic to quit smoking.

Rosemary Campos (1993), the director of a medium security inpatient mental hospital in Pomona, California, admitted that the extremely high incidence of methamphetamine addiction among her patients initially baffled her. Methamphetamine use in southern California was extremely high at the time, but among her clientele, it was almost ubiquitous and yet, few other substances were used alongside them (save, of course, for nicotine). She considered the possibility that what she was actually seeing was methamphetamine-induced psychosis, but cessation of the methamphetamine did not ease the psychosis, and signs of psychosis existed prior to documented methamphetamine usage.

While on the surface, such an abuse of stimulants might not appear to make sense, upon closer examination it may become clearer. While there are still no definitive answers, there are three basic schools of thought explaining the reasons for the use and abuse of stimulants and nicotine by schizophrenics:

1. In accordance with Zuckerman's Diathesis-Stress Model (1999), smoking and stimulant abuse trigger schizophrenia in susceptible individuals.
2. Stimulant abuse and smoking relieve monotony in the day-to-day lives of schizophrenics, who are frequently unable to hold jobs or do anything that requires a continuous day-to-day commitment and thus frequently live in poverty in subsidized housing.
3. Stimulant abuse and smoking counteract certain unpleasant effects of the neuroleptic medication one takes for schizophrenia.

NEUROLOGY OF SCHIZOPHRENIA:

The current belief of the cause of schizophrenia is the dopamine hypothesis, which states that schizophrenia is the result of an overactivity of dopamine systems in the brain (Baumeister & Francis, 2002; Angrist, et al, 2001). This was discovered from a chain of events beginning with the study of Rauwolfia Serpentina, a shrub that has been used as a folk medicine in India for diarrhea, snake bites, and delayed labor. Western researchers found that it could be used in treating insanity with violent maniacal symptoms, as well as lowering blood pressure and sedating in general. Ciba Laboratories synthesized reserpine from Rauwolfia Serpentina and marketed it under the name of Serapsil.

Soon a similarity between chlorpromazine (Thorazine) and reserpine was noted, specifically that both acted as a sedative without hypnotic effects, which differed greatly

from the barbiturates largely in use at the time. The term "tranquilizer" was coined to describe this course of action in 1953 (Baumeister & Francis, 2002).

Serotonin was discovered in 1948 and linked to mental illness in 1954 when LSD was found to be a serotonin antagonist. Since LSD appeared to cause insanity, it was reasoned, then insanity was due to a suppression of the action of serotonin, whether caused by drugs or by natural means. At the time, physical evidence was lacking, but this hypothesis was soon supported through sleep studies showing that reserpine potentiated the hypnotic actions of barbiturates and was antagonized by LSD. It was concluded that reserpine thus enhances the actions of serotonin. It was also noted, however, that this action in excess also leads to serotonin depletion, thus limiting the use of reserpine in the treatment of mental illness (Baumeister & Francis, 2002).

What was most significant about this research was that it showed for the first time the role of brain chemistry in behavior. Soon reserpine's effects on other brain chemicals and neurotransmitters were researched, and depletion of catecholamines and norepinephrine was discovered, and that inhibition of monoamine oxidase (MAO) antagonizes the effects of reserpine. It was concluded that reserpine was a nonselective blocker of monoamine storage and that a deficiency, not an excess, of neurotransmitters due to increased metabolism was responsible for reserpine's effects (Baumeister & Francis, 2002).

Dopamine, first discovered in 1910 and soon forgotten about due to its weak sympathomimetic effects, was soon examined for its mediating effects on reserpine. It was soon realized that areas of the brain containing large amounts of dopamine (the corpus stratum, for example) have little norepinephrine, while areas containing large

amounts of norepinephrine (such as the medulla oblongata) have little dopamine. Large amounts of dopamine in the corpus stratum led researchers to suggest that dopamine plays a role in the extrapyramidal motor system.

One of reserpine's side effects is pseudoparkinsonism, which can be explained by the depletion of dopamine. L-dopa was found to reverse the effects of reserpine-induced pseudoparkinsonism (as well as true Parkinsonism), leading to advances in the treatment of Parkinson's disease and the realization that Parkinsonism is caused by a depletion of dopamine. Meanwhile, the induction of pseudoparkinsonism showed that reserpine caused a depletion of dopamine.

The antipsychotic effects seen in reserpine and chlorpromazine (which has similar pseudoparkinsonism-inducing effects) were so consistently present with the pseudoparkinsonism that there was assumed to be a connection. Further study showed that while reserpine depletes all the major neurotransmitters, chlorpromazine does not deplete serotonin or the catecholamines, thus limiting its actions to dopamine. Yet there was still an antipsychotic effect and the presence of extrapyramidal symptoms such as pseudoparkinsonism.

All the while, there was a great deal of research going on examining the effects of stimulants on the brain. Originally, it was hypothesized that amphetamines directly stimulate peripheral adrenergic receptors. However, Jacques van Rossum, a Dutch pharmacologist, discovered that the locomotor stimulant effects of cocaine could be blocked by reserpine, while the effects of amphetamines could not. It was later discovered that amphetamines are chemically similar in structure to dopamine and that

chlorpromazine and haloperidol (Haldol) could block their effects, while as mentioned previously, reserpine could not.

In 1966, van Rossum combined his research on amphetamines with the research on reserpine's and chlorpromazine's antipsychotic effect and hypothesized that schizophrenia was due to an excess of dopamine (Baumeister & Francis, 2002). This has since been supported by research showing the causes and effects of amphetamine psychosis (due to amphetamine's effects on dopamine transmission) and that untreated schizophrenics do not suffer from Parkinson's disease--it is generally medically caused in schizophrenics, and those who are treated with atypical antipsychotics do not experience its effects.

SCHOOLS OF THOUGHT ON STIMULANTS AND SCHIZOPHRENIA

1. Smoking and stimulant use cause schizophrenia in susceptible individuals.

Petit-Zeman (2002) hypothesizes that nicotine use is a cause of mental illness in susceptible individuals. She cites research that followed over 1,000 young adults over a five-year period and found that the 13 percent who suffered from major depression at the beginning of the study were three times more likely to progress from light smoking to daily smoking despite no evidence that depression increased the tendency to take up smoking. However, a history of daily smoking prior to the study doubled the risk of

developing major depression during that period, an instance where smoking predates mental illness.

Another study she cites followed 8,704 teenagers who were not depressed and may or may not have been smokers along with 6,947 teenagers who were highly depressed and did not smoke in the previous month. In this study, it was found that the previous smoking was more significant than the depression in determining who would become a heavy smoker, but more importantly, teenagers who were mentally fit but smoked heavily were four times more likely to develop depression than those who did not smoke.

People who smoked a pack per day at age 16 were discovered to have 16 times the likelihood of developing panic disorder, seven times the chance of developing agoraphobia, and were five times as likely to develop generalized anxiety disorder. Yet anxious adolescents were found to be no more likely to become smokers than non-anxious adolescents.

Though there are no clear studies showing the correlation of smoking prior to schizophrenia, anecdotal evidence suggests that most schizophrenics started smoking prior to their schizophrenia, and as mentioned in the introduction to this paper, almost all schizophrenics smoke. Thus, Petit-Zeman (2001) says, there is a likelihood that the smoking may have caused the schizophrenia due to some as yet unknown effect of nicotine or other chemicals on the brain.

Nicotine has been shown to increase dopamine levels, though levels in rats sufficient to cause significant anxiety or other mental illness are attainable only by the heaviest of chain smokers (Petit-Zeman, 2001). Still, in people predisposed to

schizophrenia (that is, who may already have high levels of dopamine), it may not take a great increase for one to move into schizophrenia.

Lyons, et al (2002) noted that twins of schizophrenics who were not themselves schizophrenic still had significantly higher rates of smoking than the general population, as well as a greater difficulty quitting than the general population, suggesting that there may be a connection between nicotine use and a family vulnerability to schizophrenia that may actually predate schizophrenia.

Amphetamine psychosis resembles schizophrenia in many ways, and consequently, it is quite difficult to differentiate between amphetamine psychosis and a schizophrenic who abuses amphetamines. This, combined with the knowledge and belief of permanent damage from drug abuse leads to the possibility that schizophrenia could be caused in some from abuse of amphetamines.

Considering that, again, as many as 60% of schizophrenics use illicit drugs (Pencer & Addington, 2003), and that most of them use amphetamines (Wheatley, 1998), and that amphetamines may cause psychotic reactions, there should be no surprise that such a conclusion could be made. Amphetamine psychosis was the link that led to the development of the dopamine hypothesis of schizophrenia (Angrist, et al, 2001; Baumeister & Francis, 2002), as amphetamine psychosis looks very similar to paranoid schizophrenia (Harris & Batki, 2000).

It was also discovered that haloperidol was effective in treating both schizophrenia and amphetamine psychosis. In addition, bilateral injection of dopamine into the striatum could cause amphetamine-like psychosis and that neuroleptics (like haloperidol) could then reverse those symptoms (Angrist, et al, 2001).

As researchers questioned what was due to the effects of the drug and what was due to the disease, many research projects were undertaken attempting to induce psychosis in non-mentally ill drug abusers, giving schizophrenics small doses of amphetamines to see if they would induce psychosis at a lower level than normal (yes, they would, in 40% of patients, Yui et al, 1999), and using various neuroleptics to counteract all these effects (Angrist, et al, 2001).

One crucial difference between amphetamine psychosis and schizophrenia was discovered, however. Amphetamine psychosis mimics primarily the positive symptoms of schizophrenia, that is, the thought disturbances, hallucinations, and paranoia, while the negative symptoms (flat affect, motor retardation, etc.) seen in schizophrenia are not seen in amphetamine psychosis (Angrist, et al, 2001; Yui, et al, 1999; Harris & Batki, 2000).

Yui, et al (1999) note that symptoms similar to amphetamine psychosis (flashbacks) can recur in response to stress in some individuals who have previously abused amphetamines and induced psychosis. They consider this a result of changes in dopaminergic systems resulting in a sensitization to stress, and note that such a reaction does not appear to occur in people who have not abused amphetamines.

Yui, et al (1999) also notes that cocaine psychosis is similar to amphetamine psychosis, except with the added sensation of extreme sensitization and appetite for more cocaine (a normal effect of cocaine usage), while phencyclidine (PCP) psychosis tends to include both positive and negative symptoms (i.e., flat affect and motor retardation not found in amphetamine psychosis). Harris & Batki (2000) describe cocaine psychosis as being distinguishable from schizophrenia by the absence of flat affect, self-limited nature, presence of psychomotor agitation, and insistent search for communication rather than an

avoidance of contact. In addition, cocaine psychosis usually lasts less than 24 hours and generally occurs first when the person changes ingestion routes from nasal to smoking or intravenous. Tizabi, et al (1999) describes phencyclidine psychosis as one of the best pharmacological models of schizophrenia.

Shortly after World War II, large amounts of methamphetamine that was kept by the military was stolen and found its way onto the Japanese black market, leading to a major methamphetamine epidemic. Many of the users developed psychosis, and in many cases, the psychosis did not stop with cessation of use; some took years to recover. In these cases, some of the users developed negative as well as positive symptoms, and in fact, there were no clinical factors that one could use to distinguish between amphetamine-induced psychosis and paranoid schizophrenia (Yui, et al, 1999).

Research has attempted to replicate this situation, and has found that, if taken repeatedly over long periods of time, amphetamines can produce a schizophrenic syndrome that will get progressively more severe as drug use continues and will continue long after drug cessation. This appears to be due to effects in both the presynaptic and postsynaptic dopamine mechanisms, as increased release of dopamine and increased behavioral response to dopamine agonists have been demonstrated, and greater sensitization occurs over time (Yui et al, 1999).

2. Stimulant abuse and smoking relieve monotony.

The life of a schizophrenic is likely rather boring, especially while one is under treatment. A typical schizophrenic does not hold a job, so there is little to do during the

daytime. Friends and family fear psychotic relapses, so social contacts are minimized (Tidey et al, 1999). Medications used to combat schizophrenia (especially typical neuroleptics) also cloud the mind and retard motor functioning, making it all the more difficult to think and move normally, both making social interaction and any other sort of activity difficult to impossible.

In the life of a schizophrenic, there is no social constraint against smoking (Tidey et al, 1999). People in the schizophrenic's life (assuming there are any on a consistent basis) do not complain about smoking or drug use, and there is no reason to quit to pass a pre-employment drug test or so that one does not get nicotine fits while on the job, as there is no job to worry about.

In a way, the only person the schizophrenic is responsible to is himself or herself. If smoking and stimulant abuse is not perceived as a problem to himself or herself, then there is no reason for that person to quit. Sure, there may be a psychiatrist involved, but then again, schizophrenics tend to be somewhat suspicious towards them anyway.

Smoking may be the one of the few pleasures one has left in life. Some smokers have made that assertion, and for a schizophrenic, that may be true (Meadows, et al, 2001). A typical schizophrenic, whether out of boredom or because of some perceived need for more nicotine, tends to smoke far more cigarettes than the average person does. While much of that may be related to the average person's constraints on smoking (must take breaks from work in order to smoke with the number of breaks regulated by management, for example), some of that may also be simply because there is little else for a schizophrenic to do all day.

McChargue, et al (2002) see few motivators in a schizophrenic's life to quit smoking. In addition, there is little in the way of social support for the schizophrenic in that area, and few mental health providers give any access to effective smoking cessation programs or any positive reinforcement around that issue. Indeed, some even fear allowing schizophrenics to undergo the stress of quitting. Cognitive deficits related to neuroleptic medication may also make it difficult for the schizophrenic to comprehend social training around quitting smoking and comply with a prescribed program.

Stimulant use may be the same. Stimulants like amphetamines and cocaine give artificial pleasure in non-pleasurable activities. They relieve boredom, give the user a sudden sense of energy, and make the user not only feel like doing something, but like they are doing something. Such an effect may be especially significant to a schizophrenic who spends the day wasting away and feels life passing him or her by.

There is also a forced interaction around stimulant use. The schizophrenic must obtain these substances, and accordingly, must develop contacts with which he can purchase these drugs. These contacts become social interactions, even if for a short time and with a specific stated purpose. For the schizophrenic, the meetings with the drug dealer may be the most significant social interactions in his or her life, and those interactions may be as important as the actual use of the drugs.

Tidey, et al (1999) discovered that matching an alternative task with a monetary reinforcer (in essence, simulating a workplace environment) and limiting access to cigarettes during that time (as would be expected in a normal workplace environment) greatly reduced the amount of smoking in schizophrenics. In fact, the subjects in their study even decreased the amount of other drugs consumed outside of the study, with only

occasional positive tests for marijuana and no positives for amphetamines during the study's experimental phase. They cite previous studies that show that access to non-drug reinforcers can reduce drug self-administration in both mentally ill and mentally healthy individuals.

3. Stimulant use and smoking counteract the unpleasant effects of neuroleptic medication.

This is perhaps the most commonly cited reason for the high incidence of smoking among schizophrenics, and among the most highly researched. Neuroleptic medicines, especially the older typical neuroleptics like haloperidol and chlorpromazine, cause numerous side effects from cognitive dysfunction to extrapyramidal side effects. Since these medications in general antagonize the excessive release of dopamine, and stimulants (including nicotine) agonize dopamine, there is in effect, a lessening of the effects of the medication, including the side effects (Watkins, et al, 2000; Forchuk, et al, 2002; McChargue, et al, 2002).

That the cognitive dysfunction caused by neuroleptics is relieved by smoking is not simply a subjective report. It has been shown through research. Tracy, et al (2000) found that subjects did significantly better on a battery of neuropsychological intelligence and memory tests when provided with cigarettes than when not smoking.

Another effect of smoking is that nicotine increases the metabolism of most neuroleptics, in effect reducing their dosage. While this does not bode well for the schizophrenic (in that the risk of relapse is greater), the immediate effect is that he or she

feels better. The primary cause of relapse in a schizophrenic is noncompliance with medication due to an inability to handle side effects, so a case could be made for an effectively reduced dosage being superior to no dosage at all.

When questioned about their smoking, schizophrenics do frequently mention the reduction of side effects, along with the usual factors of habit, enjoying smoking, relaxation, increased sociability, and addiction (Forchuk, et al, 2002). Many report wanting to quit, but they have a very low success rate in doing so, far lower than the general population (Forchuk, et al, 2002; McChargue, et al, 2002).

For one, not only do they have the usual unpleasantness of nicotine withdrawal to deal with, they also eliminate the benefit of smoking on their medication side effects. In addition, one of the primary triggers for relapse is stress, and the effects of nicotine withdrawal create stress. Schizophrenics tend to have a lower stress threshold than most people, and consequently, given the choice between a cigarette and a psychotic break, most would accept the cigarette (McChargue, et al, 2002).

Few question the benefits of quitting smoking, in that the health benefits in doing so have been well documented. What is questioned is whether the risks outweigh the benefits, the best methods for aiding a schizophrenic in quitting smoking, and if there is an alternative way to achieve the benefits gained by smoking.

One interesting possibility is clozapine (Clozaril), an atypical antipsychotic. In addition to having far fewer movement-related side effects (including little risk of tardive dyskinesia or pseudoparkinsonism), it elevates mood and appears actually to reduce smoking in schizophrenics. Lyons, et al (2002) state that other atypical antipsychotics don't appear to have this benefit. One major disadvantage to this is that clozapine has a

major risk of fatal agranulocytosis (Julien, 2002), thus mandating regular blood testing. Given the poor compliance risk of schizophrenics, few doctors are willing to prescribe clozapine when there are other atypical antipsychotics available that have no risk for agranulocytosis, few extrapyramidal or cognitive side effects, and no reduction effect on smoking.

George, et al (2000) disagree with Lyons, et al (2002), stating that in fact, schizophrenics who use risperidone (Risperdal) or olanzepine (Zyprexa) are more likely to quit smoking than those on typical neuroleptics.

Another option being studied is nicotine replacement therapy, that is, providing the schizophrenic with a form of nicotine that carries fewer of the health effects, such as Nicorette gum, Nicotrol inhalers, or Nicoderm transdermal patches (Dalack & Meador-Woodruff, 1999). Though they may be quite helpful for the schizophrenic who smokes, in that they still can reduce the side effects of the neuroleptic medication, they tend to be cost-prohibitive for most patients who live on limited incomes. In addition, they demand compliance, in that he or she must remember to use the gum, patch, or inhaler as recommended, again a risk with a patient population that tends to be low in compliance. Results have been rather inconclusive, but in general seem to reduce smoking without actually stopping it (McChargue, et al, 2002).

Perhaps the most promising advance may be the atypical neuroleptics. The newer medications such as risperidone (Risperdal) and olanzepine (Zyprexa), safer descendents of clozapine, do not have most of the negative side effects of the conventional neuroleptics haloperidol or chlorpromazine (Julien, 2001). As such, if they are the first medication given, it is possible that the schizophrenic may not turn to stimulant abuse or

smoking to counter side effects. In addition, patients on these medications appear better able to function in society with them, and it is possible that they may thus be able to hold down some degree of employment with economic, social, and anti-smoking benefits resulting. The medications are too new to know how beneficial they will be in the long run, but at this point it appears promising.

A NEW APPROACH FOR DRUG TREATMENT IS NEEDED

One of the issues that schizophrenics face is that drug treatment and addiction support groups both tend to be very intolerant of relapse, and in addition, the support groups tend to be very intolerant of medication.

Narcotics Anonymous and Alcoholics Anonymous both prescribe abstinence from mind-altering substances, and most members take that appropriately to include narcotic analgesics and benzodiazepines, which are addictive and should not be prescribed to those with addiction histories. However, they also unfortunately include this to include antipsychotic medication. Many a schizophrenic has had a psychotic relapse due to the insistence of a well-meaning AA or NA sponsor insisting they get off of psychiatric medications because they are "mind-altering" substances. Note, by the way, that this does not agree with what is actually printed in their literature:

Now about health: A body badly burned by alcohol does not often recover overnight nor do twisted thinking and depression vanish in a twinkling. We are convinced that a spiritual mode of living is a most powerful health restorative.

We, who have recovered from serious drinking, are miracles of mental health. But we have seen remarkable transformations in our bodies. Hardly one of our crowd now shows any dissipation.

But this does not mean that we disregard human health measures. God has abundantly supplied this world with fine doctors, psychologists, and practitioners of various kinds. Do not hesitate to take your health problems to such persons. Most of them give freely of themselves, that their fellows may enjoy sound minds and bodies. Try to remember that though God has wrought miracles among us, we should never belittle a good doctor or psychiatrist. Their services are often indispensable in treating a newcomer and in following his case afterward.

(Alcoholics Anonymous, 1939).

Many drug treatment centers are staffed and managed by people who are well schooled in the 12-Step AA/NA philosophy, but not in mental health issues. While this may work well for the majority of patients, for dually diagnosed patients, such as schizophrenic stimulant addicts, this lack of training does a great disservice. Treating a relapse as a reason for discharge or movement into a more intrusive level of care does not consider that relapse rates for schizophrenics may be higher than for most alcoholics or addicts. Medication management in these agencies may also be insufficient, and full detoxification, for example, may lead into a psychotic relapse.

One unfortunate result of this is that frequently insurance carriers may refuse to place a schizophrenic into an appropriate agency (i.e., one with medical and psychiatric management) because of a higher cost and lower success rate. It is all too common to see a schizophrenic placed into a minimally intensive agency with the expectation that he or she will recover. Copyright 2003, Brian Waterman, www.bedrugfree.net. This paper may not be reprinted or reposted without the author's express written consent.

she will soon relapse, fail to comply, be discharged, and cost the insurance company a small amount of money.

Schizophrenics need a higher level of intervention than most. Drug treatment will have to include full medication management and supervision above all else and will likely have to be residential in nature (so that all aspects of schizophrenic life, including social training and job skills, can be covered and medication compliance be assured). Smoking cessation would be an appropriate part of this, including nicotine replacement therapy and/or bupropion (Zyban) therapy, which has been shown some effectiveness in reduction of smoking in the schizophrenic (Eden Evins, et al, 2001).

After treatment, support groups should be aimed not at the addiction, but at living as a clean and sober schizophrenic. These groups could be set up in a medical facility or through an advocacy group such as the National Alliance for the Mentally Ill (NAMI). These groups could have a focus similar to a 12-step group, or could use other therapeutic methods, but either way, they would need to be open to addressing the unique needs of the schizophrenic, which are frequently ignored or mistreated by typical AA and NA groups. It would be advisable to have someone with some training in mental illness, either a psychiatrist/psychologist, counselor, or case manager leading the group, or at least in attendance so that situations requiring professional knowledge could be dealt with and psychiatric emergencies could be handled appropriately.

Meadows, et al (2001) has had some success in working with schizophrenics who want to quit smoking through a specialized group aimed specifically at those with psychotic disorders. Specially trained general practitioners (physicians trained in the specific physical and emotional effects of smoking cessation both experienced in the

general population and specific to the psychotic) worked with psychiatrists and educated their patients on all aspects of smoking cessation in the psychotic, from expected symptoms to social support to not drinking acidic drinks when using nicotine gum and how to hold it in the mouth. They reported that in their small pilot group, cigarette smoking dropped from an average of 39.5 cigarettes per day to 7.4 cigarettes daily, and many of the participants quit successfully.

Advances in the handling of schizophrenia have been occurring far more in the last 20 years with the marketing of atypical antipsychotics and the mainstreaming of the mentally ill into society, but there is still a great deal of progress that needs to be made. However, with the ever-expanding amount of research specifically into the needs of the mentally ill, there is a great deal of potential for the years ahead.

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