EDITORIAL

Tobacco Use in Childhood and Adolescence

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Teenagers start smoking as part of the process of experimentation with the adult environment, accompanied by curiosity over the effect smoking has. Repeating the experiment, badly tolerated at first, leads to the unconscious learning of the association between certain situations and the pharmacological effects of nicotine that make the situations easier, leading to nicotine use for emotional regulation and to the initiation of dependence. Both the initiation and the continuation of tobacco use, as with other addictions, derive from the individual's social environment and are reinforced by other biological or psychological characteristics that determine the extent to which the dependency develops. This is the biopsychosocial model of addictive behavior, which brings together biological aspects (a natural tendency to be overweight, sad, or depressed), psychological components (involvement with stressful environments, peer groups, and leaders), and social components (the influence of publicity, and the easy access to tobacco at an affordable price). As with all behavior, classical and operant conditioning processes influence smoking behavior, which is not engaged in for pleasure but in response to particular antecedent or trigger stimuli (coffee, meals, alcohol, social relations, etc). Once the network of determinants and associations is established, addiction follows.

We studied 459 smokers (mean [SD] age of 46 [12.4] years) who had undergone treatment to give up smoking at our unit and found that 93.2% were smoking by the age of 20. This finding is consistent with other studies that demonstrate the low probability of becoming a smoker once adulthood is reached and consequently the importance of preventing tobacco dependence in childhood and adolescence in order to delay initiation and subsequently reduce the prevalence of smoking in the population.

Studies have shown that in the United States of America, 26% of 15-year-olds questioned declared they had smoked at least 1 cigarette over the previous 30 days[1]; 30% said the same in Europe, including 38% in Eastern Europe[2]; and 29% said so in Spain,[3] where there was a higher proportion of girls smoking but where boys smoked more cigarettes and the age of onset of smoking decreased from 13.6 to 13.1 years between 1994 and 1996.

These data, amply supported, demonstrate that tobacco dependency is common among adolescents and is clearly increasing. Current knowledge, summarized at the beginning of this editorial, indicates that social associations of smoking flourish in the media and tobacco marketing has a negative influence, encouraging an increase in the future prevalence of smoking. But there are other factors that influence this tendency: the media inform us daily of the increase in the school failure rate, the formation of anti-social groups, and signs of maladjustment to school and society among the young. And it is precisely the students who do not perform well at school, who are least satisfied with the school environment, who have low expectations of the future, who drop out of school, and who have a low self esteem who are more likely to become smokers.[4] The tendency to become dependent on tobacco and the future consequences of addiction are thus directed towards the less favored sectors of society, as has been demonstrated for years in developed countries.

Current understanding of strictly biological factors that influence the development of an individual's behavior as a future smoker is very limited. Many of these factors have been considered psychological components, overestimating this aspect of the smoking behavior model. For example, many studies have found that tobacco dependency in parents is associated with a greater prevalence of smoking among their children, a relation which is attributed to learning by imitation. But is that all there is or is there a biological conditioning component?

Currently, 40% of children are exposed to environmental tobacco smoke at home,[5] the well-documented, detrimental effects[6] of which include an increase in respiratory and middle ear infections, greater risk of bronchospasm and atopy, and up to three times greater risk of sudden infant death associated independently with passive smoking during both breast-feeding[7] and pregnancy.[8] The reason for this association is clear considering that the components of tobacco

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Manuscript received May 26, 2003. Accepted for publication June 3, 2003.

smoke absorbed during pregnancy are capable of interfering with the neuroregulation of respiration, contributing to the appearance of apneic events in newborn babies.[9] As the influence of environmental tobacco smoke has been amply demonstrated, proven in fact, to modify the neural regulation of functions as fundamental as respiration, might not the tobacco smoke that reaches the fetus or child manage to alter the complex mechanism of the central nervous system, known to be affected by nicotine in the establishment of the addiction? If the answer is "yes", then repeated intrauterine exposure or even postnatal exposure could make the future adolescent hunger for the substance, making them more susceptible to dependence. We are still a long way from finding evidence to confirm or reject this conjecture although certain findings may point in this direction: exposure of cell cultures to nicotine encourages the genetic expression of tyrosine hydroxylase, the enzyme that catalyzes the conversion of tyrosine into L-dopa, precursor of dopamine.[10]

Who can doubt that the higher incidence of smoking among children whose parents smoke might arise not from learning or imitation alone but also from a genetic component? And why not consider the possibility that this genetic basis is a consequence of adaptation to environmental tobacco smoke encountered when cells are still undergoing continuous mitosis in the uterus of a smoking mother? Findings from studies on twins indicate a substantial genetic component to smoking that for some authors could be the cause of between 50% and 80% of tobacco dependence.[11]

The first studies investigating the pathogenesis of these components were directed at the origins of the nicotinic receptor and its polymorphisms, but those found in the *CHRNB2* gene, related to the β_2 chains of the nicotinic receptor, were not more prevalent in smokers than in nonsmokers, according to 1 group of investigators. Nevertheless, they found that mice who lack this gene did not develop dependence on nicotine.[12,13] Analyzing other points in the complex fabric of neurobiological interactions that influence tobacco addiction, the same research group suggested the presence of a haplotype that protects against smoking initiation in one of the 4 polymorphisms in the type 5 dopamine receptor gene, DRD5.[14] Recently Batra et al[15] wrote an excellent review on the current state of our understanding of genetic influences on tobacco dependence, which can be summarized as follows:

– Genetic conditioning leading to more rapid nicotine metabolism through genetic variations in the cytochrome P, in its CYP2A6 and CYP2D6 forms, would encourage dependence. People in whom the activity of that enzyme system is defective, such as those with the genetic variants 2 and 3 of CYP2D6, have reduced nicotine metabolism and, at the same time, a lower incidence of dependence compared to control groups. Similar effects have been found for alcoholism.

– Genetic variations in dopamine receptors, their transport, and their metabolism may also affect the development of nicotine addiction and explain differences in use, maintenance of dependence, and difficulty in controlling it.

– Although the analysis of serotonin transporter gene polymorphisms, which are closely involved in withdrawal syndromes, has produced contradictory results with respect to allele frequencies in smokers and nonsmokers, they could account for tobacco dependence in certain persons with anxiety personality traits.

The risk of initiating smoking, then, has both a genetic and environmental component, but unless one were to argue the extreme evolutionary theory that the environment modifies genes, the genetic influence would only come to bear by increasing "susceptibility" to becoming a smoker. It is social conditioning, above all, that will lead a child to initiate tobacco use. Then, once again, a genetic predisposition will influence the maintenance of the smoking habit and the nature of use, such as degree of smoking, difficulty in quitting and maintaining abstinence, or the mechanisms involved in resuming the habit.

Even bearing in mind that genes might have some influence in the decision to give up smoking and maintain abstinence, once again it is the social environment that is predominant in the decision. The main motive for giving up smoking is the realization of its detrimental effects on health, either through personal experience, through other people known to the smoker, or through convincing information. Children or adolescents, however, do not think in the same way as adults and other approaches have to be taken in order to create an environment that is hostile to the initiation of smoking. These include increasing the cost of cigarettes, controlling tobacco marketing, encouraging aesthetic or ecological beliefs (the wood of a whole tree is needed to dry the tobacco of 300 cigarettes), reinforcing social and family pressure. And why not encourage children to learn and develop coping strategies for facing problematic situations, given that children with greater social difficulties (low expectations and self-esteem) are more likely to become smokers.

In the first longitudinal study carried out in Spain on this subject, Ariza and Nebot[16] analyzed the factors capable of predicting the initiation of smoking in 1460 adolescents between 12 and 19 years of age who were followed up for 12 months. They found that girls started smoking younger and more girls started smoking during the study period but that boys smoked more. Factors capable of predicting the onset of smoking included having friends who smoked, positive attitudes towards smoking, the intention to smoke in the future, and the drinking of alcohol. Tobacco use to counter stress and to control weight have been mentioned by other authors as factors inducing tobacco dependence,[17,18] and are therefore factors to be controlled and adjusted if we

want to modify the social context and, through it, influence the social and psychological parts of the model that lead to smoking. These are the only factors we can influence at present until we know more about the biological factors.

These data highlight the important role schools have in preventing tobacco dependency, but, unfortunately, what seems good in theory has not proved so in practice as results have been disappointing. Bruvold[19] performed a meta-analysis of 94 studies to investigate the effect school programs had on preventing tobacco dependence. He found that programs had a significant informative impact, including studies that were not aimed at instructing, as all school programs, independently of their objectives, have a high component of information. However, results showed little change in attitudes when compared with controls; only programs that tackled social influences and alternatives to tobacco use (basically dealing in coping strategies) had positive results, although these were small, over the year. Fernandez et al[20] studied several meta-analyses performed on school programs for preventing tobacco, alcohol, and marijuana use, and drew up a list of prerequisites for the design and application of programs of this type. The recommendations included the need to train and advise personnel to run the program, periodic reinforcement throughout schooling, concentration of information on short-term effects of smoking (aesthetics and capacity for exercise, among others) and the influence, through this information, on the various conditioning factors that affect conduct. However, these authors recommended that the programs should recognize and consider substituting the message of abstinence for one of responsible use, an alternative more utopian than realistic and one I could not disagree with more. An individual's capacity to develop an addiction very probably varies from person to person according to as yet unknown genetic factors, but one thing we do know is that it is impossible for a person who has become dependent on tobacco to maintain "responsible use" of the substance he is addicted to. Conversely, compulsiveness forms a substantial part of the concept and definition of dependence. To suggest, then, that a young person use an addictive substance, in this case tobacco, with moderation and responsibility within a program aimed at preventing its use is quite unrealistic, even more so considering that, at the ages involved, concepts like responsibility are still being formed and developed. Presenting this alternative within a drug prevention program could be equivalent to encouraging the children to experiment with drugs, justified with the same reasoning. Moreover, what is the line that separates responsible use from irresponsible use? Do we have a generally accepted notion that there is one? Obviously, to someone who smokes 40 cigarettes a day, the fact that his son smokes 10 a day might seem to be responsible use but a nonsmoking parent will find the same amount excessive.

The argument that the child is not addicted yet as he has not been smoking for long cannot be used in favor of proposing responsible use. The little we know about the addiction process includes reports that the percentage (confidence interval) of children 10 to 17 years old who had smoked in the previous 30 days and who considered that smoking "relaxes or calms" was 67.9% (6) and that 56% (6.3) believed it was "hard to quit." Those percentages were not significantly different from those found for young adults 18 to 22 years of age (75.5% [3.6] and 61.6% [4.2], respectively). Similar results and indications of addiction in smoking children have been found in other studies: 63.1% of girls between the ages of 12 and 17 who had smoked in the previous 30 days showed one or more addiction indicators, and 51.6% felt dependent on cigarettes, with the percentages increasing in direct relation with use.[22]

In the future, the development of genetic research will very probably give us other explanations of the association between tobacco dependent parents and the incidence of tobacco use among their children. Maybe then gene therapy may help us prevent the onset of smoking and maybe anti-smoking programs will become unnecessary. At present, though, and very probably then too, making smoking unattractive to teenagers and cigarettes difficult to obtain remain the most effective, the cheapest, and consequently the most efficient remedy against smoking. Why, with so much concern over the cost of medication, are those strategies not used?

REFERENCES

1. Johnston LD, O'Malley PM, Bachman JG. 1996 secondary school students in: National Survey Results on Drug Use from the Monitoring the Future Study, 1975-1995. Vol I. Rockville: National Institute on Drug Abuse, NHI Pub no 97-4139.
2. Hibell B, Anderson B, Ahlström S, Balakireva O, Bjarnason T, Kokkevi A, et al. The 1999 ESPAD Report. Alcohol and other drug use among students in 30 European countries. Stockholm: Swedish Council for Information on Alcohol and Other Drug. The Pompidou Group at the Council of Europe, 2000.
3. Encuesta sobre drogas a la población escolar, 1996. Disponible en: http://www.mir.es/pnd/observa/html/principa.htm
4. González Ferreras C. Factores biopsicosociales que inciden en el consumo de tabaco en población infanto-juvenil. Bases para una educación preventiva. Sevilla: Consejería de Asuntos Sociales. Comisionado para la Droga. Junta de Andalucía, 1999.
5. Mackay J, Eriksen M. The tobacco atlas. Geneva: World Health Organization, 2002.
6. E.P.A. Respiratory health effects of passive smoking. Washington DC: Environmental Protection Agency 600/6-90/006F, December 1992.
7. Schoendorf KC, Kiely JL. Relationship of sudden infant death syndrome to maternal smoking during and after pregnancy. Pediatrics 1992;90:905-8.
8. DiFranza JR, Lew RA. Effect of maternal cigarette smoking on pregnancy complications and sudden infant death syndrome. J Fam Pract 1995;40:385-94.
9. Avery ME, Frantz ID III. To breathe or not to breathe –what have we learned about apneic spells and sudden infant death? N Engl J Med 1983;309:107-8.
10. Hiremagalur B, Nankova B, Nitahara J, et al. Nicotine increases expression of tyrosine hydroxylase gene: involvement of protein kinase A-mediated pathway. J Biol Chem 1993; 268:23704-11.

11. Worsnop C. Smoking. Not for anyone. Chest 2003;123:1338-40.
12. Straub RE, Sullivan PF, Ma Y, Myakishev MV, Harris-Kerr C, Wormley B, et al. Susceptibility genes for nicotine dependence: a genome scan and follow-up in an independent sample suggest that regions on chromosomes 2, 4, 10, 16, 17 and 18 merit further study. Mol Psychiatry 1999;4:129-44.
13. Silverman MA, Neale MC, Sullivan PF, Harris-Kerr C, Wormley B, Sadek H, et al. Haplotypes of four novel single nucleotide polymorphisms in the nicotinic acetylcholine receptor β2-subunit (CHRNB2) gene show no association with smoking initiation or nicotine dependence. Am J Med Genet (Neuropsychiatr Genet) 2000;96:646-53.
14. Sullivan PF, Neale MC, Silverman MA, Harris-Kerr C, Myakishev MV, Wormley B, et al. An association study of DRD5 with smoking initiation and progression to nicotine dependence. Am J Med Genet 2001;105:259-65.
15. Batra V, Patkar AA, Berrettini WH, Weinstein SP, Leone FT. The genetic determinants of smoking. Chest 2003;123:1730-9.
16. Ariza C, Nebot M. Predictores de la iniciación al consumo de tabaco en escolares de enseñanza secundaria en Barcelona y Lleida. Rev Esp Salud Pública 2002;76:227-38.
17. Waldron I, Lye D. Relationships of teenage smoking to educational aspirations and parent's education. J Subst Abuse 1990;2:201-15.
18. Tomeo CA, Field AE, Berkey CS, Golditz GE, Frazier AL et al. Weight concerns, weight control behaviors, and smoking initiation. Pediatrics 1999;104:918-24.
19. Bruvold WH. A meta-analysis of adolescent smoking prevention programs. Am J Public Health 1993;83:872-80.
20. Fernández S, Nebot M, Jané M. Evaluación de la efectividad de los programas escolares de prevención del consumo de tabaco, alcohol y cannabis: ¿qué nos dicen los metaanálisis? Rev Esp Salud Pública 2002;76:175-87.
21. Office on Smoking and Health. Teenage Attitudes and Practices Survey II. Centers for Disease Control and Prevention, 1993.
22. Alcohol, Drug Abuse and Mental Health Administration, public use data tape, 1992.