COMMENTARY

Smoking in movies: a major problem and a real solution

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Smoking depicted in movies is a major and growing public-health problem. Despite a falling prevalence of smoking in the real world, the frequency of smoking in top-grossing movies in the USA has about doubled since 1990, when the US tobacco industry first promised Congress that it would stop paid product-placement in movies.[1] Indeed the frequency of smoking in movies has returned to levels not seen since 1950, well before popular understanding that smoking was a major cause of disease and death.[2] Concern over smoking in movies led WHO to make "Smoke Free Film" a theme of 2003 World No Tobacco Day.

There is already a strong case, from cross-sectional[3,4] and experimental studies,[5] that smoking in movies increases adolescent smoking. Such studies, whilst important, always suffer from the limitation that they represent a snapshot in time that might miss some important factor. Longitudinal studies, which follow up people over time and monitor changes in smoking behaviour while simultaneously measuring exposure (to movies showing smoking, in this case), provide the strongest evidence for causality that can be obtained in a population-based study.

This association between smoking in movies and increased rates of smoking by adolescents makes the report in this issue of *The Lancet* by Madeline Dalton and colleagues especially important. These investigators provide the strongest and most convincing evidence to date that smoking in movies promotes initiation of smoking in adolescents, and show that this effect is very large. After controlling for a wide variety of other effects—grade in school, sex, school, friend smoking, sibling smoking, parent smoking, receptivity to tobacco promotions, school performance, sensation-seeking propensity, rebelliousness, self esteem, parent's education, authoritative parenting, and perception of parental disapproval of smoking—52·2% of smoking initiation in the 10–14-year-olds that were studied was attributed to seeing smoking in movies.

This effect is stronger than the effect of traditional cigarette advertising and promotion, which accounts for "only" 34% of new experimentation,[6] probably because, as the tobacco industry has known for decades,[7] the subliminal effects of smoking in movies is a more powerful force than overt advertising.

Smoking in movies nearly triples the relative risk that an adolescent will start smoking. This number, however, does not tell the whole story. Like cigarette advertising and promotion,[8] the effects of smoking in movies are strongest in children whose parents are the best role models. Children of non-smoking parents who are in the top quartile of exposure to smoking in movies are 4·1 times as likely to smoke as those in the lowest exposure quartile. This effect is substantially stronger than the increase by 1·6 times between these two exposure groups in children of smoking parents.

Thus smoking in movies is having a major effect on health. In the USA, about 2050 adolescents (age 12–17) start smoking every day and about 32% of these people—660 a day—will die prematurely because of smoking.[9] Assuming that the 52·2% attributable risk observed by Dalton and colleagues applies to this whole group, smoking in movies is responsible for addicting 1080 US adolescents to tobacco every day, 340 of whom will die prematurely as a result.

The good news is that the effect of smoking in movies shows a clear dose-response relation. So, as Dalton and colleagues note, reducing the exposure to smoking in movies will reduce the effect on smoking and death. This goal could be accomplished easily by simply including smoking (or other tobacco promotions, such as appearance of cigarette billboards) as a reason for rating movies as "adult content", an "R" rating (children under 17 not admitted without a parent) in the USA.[10,11] In the sample of movies in Dalton's study, about 60% of the total exposure to smoking in movies was in youth-rated films (G, PG, and PG-13 in the USA; J Sargent, personal communication). Eliminating smoking in these movies would reduce the exposure by about 800 occurrences, more than a one-quartile drop in exposure, which would reduce the effect of smoking in movies by about half. Put another way, an R rating for smoking in movies would prevent about 330 adolescents from starting to smoke and ultimately extend 170 lives every day.

These numbers underestimate the true benefits of an R rating because in recent years (after Dalton and colleagues finished their data collection), the number and amount of smoking in youth-rated movies has increased.

An adult content or R rating for smoking in movies would not have much effect on the movies that children see because, unlike sex and violence (the primary other reasons, along with offensive language, that films are rated for adult content), smoking in movies does not sell movie tickets.[5] Studios would simply stop putting smoking in movies aimed at an adolescent market.

The tobacco-control movement has spent many years and millions of dollars attempting to reduce youth smoking by working to implement policies that restrict youth access to cigarettes—with no effect on youth-smoking prevalence.[12,13] By contrast, the work by Dalton and colleagues, together with the earlier research in this area, strongly indicates that pushing for policy changes to reduce youth exposure to smoking in movies will have a rapid and substantial effect on youth smoking—and the subsequent disease and death smoking causes. It is time for health advocates worldwide to join with WHO, the American Medical Association, the American Legacy Foundation, and the Los Angeles Department of Health[10] in insisting that the authorities who rate movies give movies that depict smoking an adult content or R rating.

Every day of delay means more unnecessary addiction and death because of Hollywood's love affair with the tobacco industry.

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Human genetic variation and disease

Genetic differences between people and populations affect susceptibility to disease, resistance to infection, and response to drug treatment. The near completion of the human genome sequence and the discovery of common sequence variations now make it possible to examine the link between these variations and disease.

Single-nucleotide polymorphisms (SNPs) are the most common known variation in the human genome sequence. SNPs alter the composition of a single nucleotide in an otherwise conserved region of DNA. David Reich and colleagues[1] recently validated the data on the frequency of SNPs in the human genome by repeating the sequencing of candidate SNPs in at least 40 individuals. These investigators found that the vast majority of SNPs in existing databases are truly polymorphic. On average, SNPs are found roughly every 1200 base-pairs, or eight times in every 10 000 base-pairs.[2] Thus any two human beings are likely to be roughly 99·92% identical at the nucleotide-sequence level.

Base changes in both protein-coding and protein-non-coding regions can significantly affect the chance of disease. Most importantly and most obviously, single-base changes underlie most common dominant and recessive diseases with single-gene Mendelian inheritance. SNP markers have also been useful for mapping human disease genes. For example, the use of SNP markers for linkage analysis has already made possible the more rapid positional cloning of genes associated with disease susceptibility, such as the *NOD2* locus associated with Crohn's disease.[3]

Whilst most common diseases, such as hypertension, adult-onset diabetes, and atherosclerosis, can be linked in some cases to single-gene disorders, most individuals with these syndromes do not carry the major predisposing mutations. The "common disease, common variant" hypothesis predicts that most of the variation in susceptibility to highly prevalent diseases is caused by variants that occur at high frequency in human populations.[4] The use of SNP markers makes it possible to search for such variants systematically.[5] In a recent example, variations in common SNPs in a single gene, *ADAM33*, have been proposed as risk factors for asthma.[6]

One of the most touted applications for SNP analysis in medical practice has been the concept of pharmacogenomics. The principle is that genetic variation between people may explain differences in drug metabolism as well as side-effects. Variations in both the coding sequence[7] and the promoters[8] of different cytochrome P450 genes have been found to affect drug metabolism and drug response. Such variations may also occur in drug targets.

In principle, the identification of such variations could lead to the practice of "personalised medicine". In such a scheme, each patient would receive individualised medical treatment based on genetically determined drug response. Similarly, patients could be selected for chemopreventive regimens based on genetic risk. However, the vision of personalised medicine is far from being realised in the clinic.

In the meantime, several other applications of SNPs are likely to enter clinical use more quickly. These include the use of SNPs as markers to identify regions of chromosomal loss of heterozygosity in cancer[9] as well as identity analysis for forensic examination and parentage testing.

In passing, it is worth noting that other uncharacterised sequence variations in the human genome may be more common than SNPs. Comparison of the human and chimpanzee genomes shows that the two species are roughly 98·8% identical at the nucleotide-sequence level.[10] However, nucleotide insertions and deletions show differences in about 3·5% of the genome between the two species.[11] Such differences are caused in large part by mobile genetic elements such as retrotransposons, which move through the genome by producing RNA intermediates and then insert reverse-transcribed cDNA into a new site. Human beings, too, are likely to show significant numbers of insertion and deletion changes that may account for many of the subtle variations to be found between them.

Over the next decades, the discovery of human genetic variation is likely to transform our understanding of medical biology and the practice of clinical medicine. However, the era of genetic understanding for common disorders is still in its infancy.

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