Exposure to parental and sibling smoking and the risk of smoking uptake in childhood and adolescence: a systematic review and meta-analysis

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ABSTRACT

Background There is increasing evidence that contact with other smokers, particularly in the family, is a strong determinant of risk of smoking uptake. A systematic review and meta-analysis of the magnitude of these effects is reported.

Methods Studies were identified by searching four databases to March 2009 and proceedings from international conferences. Meta-analyses were performed using random effects, with results presented as pooled ORs with 95% CIs.

Results 58 studies were included in the meta-analyses. The relative odds of uptake of smoking in children were increased significantly if at least one parent smoked (OR 1.72, 95% CI 1.59 to 1.86), more so by smoking by the mother (OR 2.19, 95% CI 1.73 to 2.79) than the father (OR 1.66, 95% CI 1.42 to 1.94), and if both parents smoked (OR 2.73, 95% CI 2.28 to 3.28). Smoking by a sibling increased the odds of smoking uptake by 2.30 (95% CI 1.85 to 2.86) and smoking by any household member by 1.92 (95% CI 1.70 to 2.16). After adjusting for overestimation of RRs it is estimated that, in England and Wales, around 17 000 young people take up smoking by the age of 15 each year as a consequence of exposure to household smoking.

Conclusions Parental and sibling smoking is a strong and significant determinant of the risk of smoking uptake by children and young people and, as such, is a major and entirely avoidable health risk. Children should be protected from exposure to smoking behaviour, especially by family members.

INTRODUCTION

Approximately 1.1 billion people in the world are current smokers, and this figure is expected to rise to more than 1.6 billion by the year 2025.[1] Since regular smoking results in a 50% likelihood of premature death and an average reduction of life expectancy by 10 years,[2] smoking represents a massive public health problem. Tobacco smoke is powerfully addictive, so experimentation and uptake of smoking which, in developed countries typically occurs in late childhood or adolescence,[3] is a highly hazardous behaviour. It is crucially important therefore to prevent exposure of young people to factors that increase their risk of smoking uptake.

Various risk factors have been shown to be associated with tobacco use in adolescents,[4] ranging from individual to social and community and societal factors—including age, sex and socioeconomic status[5]; parental separation[6] or family conflict[7]; and smoking among peer groups,[8–10] in feature films[11] and, perhaps most importantly, among family members.[4] Smoking in the home is a recognised and entirely avoidable health hazard for children both before and after birth through passive exposure to smoke,[12] however the influence of family smoking on the risk of smoking uptake and consequent future health of the individual is an aspect that to date has not be systematically reviewed and quantified.

In this paper we have reviewed the available world literature on the relation between smoking by family members and uptake of smoking among children and adolescents, and combined this information in meta-analyses to provide summary estimates of the magnitude of the effects of smoking by different family members. We have also used these estimates to calculate the number of children and young people in England and Wales who take up smoking each year as a result of smoking by others in their household. The work was carried out as part of a more extensive review of the effects of passive smoking in children for the Royal College of Physicians.[13]

METHODS

Systematic review

We performed comprehensive searches of four databases (EMBASE, MEDLINE, PsycINFO and CAB Abstracts) from 2000 to March 2009 to identify all epidemiological studies assessing the association between smoking by children (2–12 years of age) and adolescents (13–19 years of age) and exposure to parental smoking (either parent, at least one parent, both parents), household smoking or sibling smoking (search strategies available by request). We also hand-searched conference proceedings from the Society for Research on Nicotine and Tobacco, the UK National Smoking Cessation Conference, the World Conference on Tobacco or Health from 2006 to 2009, as well as reference lists from identified relevant studies and previous narrative reviews. The systematic review was carried out in accordance with the Meta-analysis Of Observational Studies in Epidemiology (MOOSE) guidelines.[14]

One author (MLJ) assessed the eligibility of studies from their titles and abstracts, excluding those that were not relevant. The full texts of eligible papers were assessed independently for eligibility by two authors (MLJ and JL-B) who extracted the data using a data extraction form. Disagreements were resolved through discussion.

No language restrictions were imposed during the searches, but papers written in languages

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other than English were excluded from the meta-analyses. The Newcastle Ottawa Scale[15] was used to assess the quality of the included studies based on selection, comparability between exposure groups and ascertainment. The a priori chosen score of 7 was used to distinguish high from poorer quality studies.

Meta-analysis

Dichotomous data were extracted using raw values, crude ORs or adjusted ORs with 95% CIs. Adjusted ORs were used in preference where available. We performed meta-analyses using random effect models since the effect measures were expected to be heterogeneous. Exposure groupings (eg, any parent smoking) were defined empirically from those used in the studies. Heterogeneity between the studies was assessed using I^2.[16] Subgroup analyses based on quality (high vs poorer quality) and the definition of smoking status (current vs ever) were used to explore heterogeneity. Owing to disparate categories for the ages of the children and adolescents across the studies, subgroup analysis based on age at outcome was not feasible. Where extreme levels of heterogeneity were seen between the studies ($I^2 \geq 90\%$), we performed sensitivity analyses to assess the effect of excluding outliers. We also restricted analyses to studies which adjusted for at least three of the following known confounders: age, gender, socioeconomic status, ethnicity, educational achievement, peer smoking and family structure. Publication bias was assessed using funnel plots. Analysis was performed using Review Manager 5.0.[17] p Values <0.05 were considered statistically significant.

Population attributable fraction estimation

We estimated the proportion of children who live in a household in which at least one person smokes using data from the Health Survey for England,[13] and used the formula $p(OR - 1)/[p(OR - 1) + 1]$, in which p is the proportion of the cohort exposed to passive smoking and OR is the OR for smoking in children or adolescents where a member of the household smokes, to estimate the proportion of children whose smoking is attributable to household smoking exposure. We then used national smoking prevalence data for England and Wales to estimate the number of smokers by age 15 generated as a result of household exposure. Since ORs overestimate true RRs and this effect becomes increasingly relevant as the prevalence of the outcome increases, we also calculated the attributable risk using a lower RR based on an independent estimate that the magnitude of this likely overestimation is of the order of 15%.[18]

RESULTS

Overview of included studies

We identified 179 potentially eligible studies published between 2000 and 2009 and excluded 102 after screening the title and abstract. A further 19 studies were excluded from the meta-analyses because the data were not presented in a suitable format for meta-analysis,[3 19–29] the same cohort was used in an already included study[30 31] or they were published in a non-English language,[32–36] leaving 58 studies included in the meta-analysis (table 1, figure 1).[5 7 10 37–91] The majority of the studies were conducted in the USA or Europe and measured adolescent smoking status by self-reports, although two assessed cotinine in saliva.[40 44] The majority of the included studies reported adjusted estimates, with only three reporting crude estimates.[51 62 87] The quality of the studies ranged from 3 to 8, with a median score of 6.

Parental smoking

Exposure was determined by having one parent who smokes in 14 studies, of which a pooled analysis found that the risk of smoking in adolescence was increased by 62% (OR 1.62, 95% CI 1.49 to 1.76; see figure 5 in online supplement)[10 38 40 42 47 48 65 71 76 78 83–86] which was independent of quality and the definition of smoking used. The effect was stronger for smoking by the mother (OR 2.19, 95% CI 1.73 to 2.79; 24 studies, I^2=99%; figure 2)[41 48 49 51 55 57 58 60–64 66 68 69 72 79 80 82 87–91] than the father (OR 1.66, 95% CI 1.42 to 1.94; 18 studies, I^2=90%; figure 3).[48 51 55 57 58 60–64 70 72 79 80 87–89 91] Subgroup analysis based on quality found higher magnitudes of effect for higher quality studies for smoking by the mother (higher quality, OR 2.35, 95% CI 1.56 to 3.53, 13 studies); conversely, lower quality studies had a higher pooled magnitude of effect for smoking by the father (low quality, OR 1.94, 95% CI 1.52 to 2.48, 7 studies) (tests for subgroup differences, $p<0.0001$ for both comparisons). Studies which defined smoking status in the adolescent as having ever tried smoking found stronger effects (mother, OR 2.76, 95% CI 1.58 to 4.82, 11 studies; father, OR 1.88, 95% CI 1.47 to 2.40, 11 studies) than those that used current smoking (mother, OR 1.64, 95% CI 1.36 to 1.96, 11 studies; father, OR 1.45, 95% CI 1.15 to 1.83, 6 studies) (test for subgroup differences, $p<0.0001$ for both comparisons). Sensitivity analyses where studies with outlier results were excluded had marginal effects on the pooled estimates for mother and father smoking. Additionally, sensitivity analyses restricted to studies which adjusted for at least three of the specified confounders had small effects on the pooled estimates for smoking by the mother (OR 2.25, 95% CI 1.66 to 3.05, 15 studies) and father (OR 1.52, 95% CI 1.32 to 1.76, 11 studies).

Among children with at least one parent who smoked, the risk of smoking in the child was increased by 1.72 (95% CI 1.59 to 1.86; 10 studies, I^2=23%, figure 6 in online supplement)[7 39 44–46 53 54 56 74 75]; if both parents smoked, the risk was increased almost threefold (pooled OR 2.73, 95% CI 2.28 to 3.28; 15 studies, I^2=93%, figure 7 in online supplement).[10 42 44 47 48 57 65 71–73 76 78 79 85 87] Subgroup analysis showed similar estimates for quality and for the definition used for smoking status. Sensitivity analyses where studies with outlier results were excluded reduced the pooled effects for both parents smoking (OR 2.16, 95% CI 1.88 to 2.48, I^2=72%, excluded studies,[42 57] Asian and European subgroups[78 79]). Additionally, sensitivity analyses restricted to studies which adjusted for at least three of the specified confounders had little effect on the pooled estimate for both parents smoking (OR 2.76, 95% CI 2.20 to 3.47, 11 studies).

Sibling smoking

A pooled analysis of 23 studies found that the effect of smoking by a sibling (usually defined within the included studies as an older sibling, with the remaining studies defining as any sibling) more than doubled the risk of an adolescent smoking (OR 2.30, 95% CI 1.85 to 2.86; 23 studies, I^2=95%; figure 4).[10 37 39 40 46 55 57–62 64 65 73 74 76 77 79 80 87–89] Subgroup analyses showed higher magnitudes of effect for poorer quality (OR 2.95, 95% CI 2.09 to 4.18) than for high quality studies (OR 1.87, 95% CI 1.62 to 2.17), and for studies assessing ever tried smoking (OR 2.60, 95% CI 1.95 to 3.46, 10 studies) (test for subgroup differences, $p<0.0001$ for both comparisons). Sensitivity analyses where studies with outlier results were excluded had marginal effects on the pooled estimate (OR 2.04, 95% CI 1.81 to 2.30, I^2=77%, excluded studies[57 59 64]). Additionally, sensitivity analyses restricted to studies which adjusted for at least three of the

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Table 1 Characteristics of the 58 included studies

Study year (reference)	Adolescents (n)	Age of adolescents (years)	Source of sample	NOS score for quality	Study design	Confounders allowed for*
Asbridge *et al*[37]	3400	13–19	School-based (30 metropolitan schools), Toronto Youth Crime and Victimisation Survey, Canada	7	Cross-sectional	A, B, C, E
Bauman *et al*[38]	8273	13–17	School-based, National Longitudinal Study of Adolescent Health, USA	7	Longitudinal	A
Bergamaschi *et al*[39]	2691	16	School-based (2nd year high school students), Romagna, Italy	6	Cross-sectional	A
Bricker *et al*[40]	5520	8	School-based (40 schools),Washington State, USA	6	Longitudinal	A
Cornelius *et al*[41]	567	14	Family-based survey (from prenatal clinic), Magee Women's Hospital, Pittsburgh, USA	8	Longitudinal	A, J
Den Exter Blockland *et al*[42]	2206	10–14	School-based (Dutch youth), The Netherlands	7	Cross-sectional	A, B, C, E
Elder *et al*[43]	660	11–16	School-based, Hispanic adolescents, San Diego, California, USA	6	Cross-sectional	A, B, J
Fidler *et al*[44]	650	11–16	School-based, (36 schools), HABITS Study, South London, UK	8	Longitudinal	A, B, C, D
Fisher *et al*[45]	10 593	12–18	Family study, Growing Up Today Study (GUTS), NHS 2 Study, USA	6	Cross-sectional	A, B, J
Forrester *et al*[46]	3641	12–17	Community study (rural area), Oregon, USA	7	Longitudinal	A, B, E, J
Foster *et al*[47]	2153	14	Family study, Dartmouth, North England	6	Longitudinal	A, C, J, S
Gilman *et al*[48]	559	12–17	Family study, New England, USA	7	Longitudinal	A, B, D
Griesbach *et al*[7]	10 500	15	WHO cross-national study of health behaviours, health and its social context in children and adolescents, Europe and North America	7	Cross-sectional	A, B, C, S
Griesler *et al*[49]	1165	14–21	National Longitudinal Survey of Youth (NLSY), African, Hispanic and white mothers and children	7	Longitudinal	A, C, D, E, J, S
Gritz *et al*[50]	1004	10–18	School-based (6 schools), Houston-Galveston, Texas, USA	6	Longitudinal	C, D, J
Hesketh *et al*[51]	6674	13–18	School-based, Zhejiang Province, Eastern China	3	Cross-sectional	–
Hollis *et al*[52]	2526	14–17	Clinic-based, adolescent primary care patients in medical office waiting rooms, Oregon, USA	6	Cross-sectional	A
Jackson[53]	1220	11–14	School-based (4 middle schools), Central Carolina, USA	6	Cross-sectional	A, B, D
Jackson and Dickinson[54]	594	17.1 (mean)	School-based (1 element school out of 12), Child-to-Adolescent Panel Study (CAPS), North Carolina, USA	6	Longitudinal	A, B, D
Johnson *et al*[55]	3654	8–14	School-based (24 public schools), The Child and Adolescent Trial for Cardiovascular Health (CATCH), from 4 geographical regions, USA	7	Longitudinal	A, B, D
Johnson *et al*[56]	4763	14–15	School-based (22 schools), Acadiana Coalition of Teens against Tobacco (ACTT), 6 Louisiana parishes, USA	7	Cross-sectional	A
Kalesan *et al*[57]	37 224	11–18	School-based (315 schools), Maryland, USA	6	Cross-sectional	B
Kelishadi *et al*[58]	427	12–20	Isfanah, Iran	7	Case-Control	B, C, E, S
Komro *et al*[59]	1282	13–16	Community study, Minnesota, Tobacco Free Future Project (TFFP), USA	6	Cross-sectional	A
Kristajanson *et al*[60]	7430	14–16	School-based, youth in Icelandic secondary schools, Iceland	6	Cross-sectional	C, E, J, S
Malcon *et al*[61]	1187	10–19	School-based (urban area), Pellet, Southern Brazil	6	Cross-sectional	A, B
Martini and Sulistyowati[62]	1630	13–18	School-based, two regions and two cities, East Java Province, Indonesia	4	Cross-sectional	–
Menezes *et al*[63]	4452	10–12	Community-based, a cohort of children born in 1993 and followed and later sought 2004–5	7	Longitudinal	A, B, C, D, E
Milton *et al*[64]	247	9–11	School-based (6 primary schools), Liverpool, UK	7	Longitudinal	A, B, D, J
Molyneux *et al*[65]	6522	11–16	School-based (10 secondary schools, 50 state secondary schools), Nottinghamshire, UK	6	Cross-sectional	A, B, C, J
Molyneux *et al*[10]	2109	13–16	School-based (10 secondary schools), from 50 state public secondary schools, Nottingham, UK	5	Cross-sectional	B, J
Moore *et al*[66]	1246	15–16	School-based (55 secondary schools), Wales, UK	6	Cross-sectional	A, B, E, J
Nichols *et al*[67]	858	12–15	School-based, (30 schools), New York, USA	6	Longitudinal	A, D
O'Byrne *et al*[68]	816	15.1 (mean)	School-based (2 high schools and 2 junior high schools), Kansas City, Metropolitan area, USA	7	Cross-sectional	A, B, D
O'Callaghan *et al*[69]	4541	5–14	Mater-University, Queensland Study of pregnancy (MUSP): a cohort of women at first antenatal visit	6	Longitudinal	A, C, S
Ogwell *et al*[70]	1130	12–17	School-based (10 schools from 209 primary schools), Nairobi Province, Kenya	7	Cross-sectional	A, B, E
Otten *et al*[71]	5495	11–16	School-based (33 schools), The Netherlands	7	Longitudinal	A, B, E

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Table 1 Continued

Study year (reference)	Adolescents (n)	Age of adolescents (years)	Source of sample	NOS score for quality	Study design	Confounders allowed for*
Ozawa *et al*[72]	2012	15–18	Schools-based, senior high school, Fukuoka City, Japan	7	Cross-sectional	A, E
Pärna *et al*[73]	4049	13–18	School-based, Tallinn, Moscow	7	Cross-sectional	A, C, S
Pust *et al*[5]	1298	12–17	Study-Drug Affinity of Young People in Federal Republic of Germany	7	Cross-sectional	A, E
Rajan *et al*[74]	3962	8–9	School-based (20 schools), Washington State, USA	7	Longitudinal	A
Rozi *et al*[75]	772	14.8 (mean)	School-based, government and private schools, Karachi, Pakistan	6	Cross-sectional	A, D
Sargent and Dalton[76]	372	8–17	School-based (3 rural Vermont), Dartmouth, USA	7	Longitudinal	A, C, E
Sasco *et al*[77]	3650	11–17	School-based, public urban schools, France	6	Longitudinal	A, B, S
Scragg *et al*[78]	29 271	14–15	School-based (4th form), New Zealand	5	Cross-sectional	A, B, C
Scragg and Laugesen[79]	28 689	14–15	School-based (year 10 students), National Surveys of Year 10 Students, New Zealand	7	Cross-sectional	A, B, D
Shamsuddin and Haris[80]	244	15–16	School-based (6 Secondary Schools), Kota Bharu, Kelantan, Malaysia	7	Cross-sectional	A, E, J
Simons-Morton *et al*[81]	3451	11–14	School-based (7 middle schools), Maryland School district, Washington DC, USA	6	Cross-sectional	A, B, D
Siziya *et al*[84]	1838	13–15	School-based, Zambia Global Youth Tobacco Survey (GYTS) 2002 data, Chongwe district, Zambia	6	Cross-sectional	A, J
Siziya *et al*[83]	2323	6–16	School-based, Global Youth Tobacco Survey (GYTS) 2003 data, Kilimanjaro region, Northern Tanzania	6	Cross-sectional	A, C, J
Siziya, *et al*[82]	1790	13–15	School-based (public and private junior high school), data from East Timor Global Youth Tobacco Survey (GYTS)	6	Cross-sectional	A, B, C, J
Szabo *et al*[85]	4125	12–17	School-based (68 schools), Victoria, Australia	7	Cross-sectional	A, B, E
Tyc *et al*[86]	237	12–18	School-based (1 large junior school and 2 senior high schools), Memphis, USA	7	Cross-sectional	A, B, C
Vink *et al*[87]	3598	12–15	Netherlands Twin Register (NTR)	5	Longitudinal	–
Wen *et al*[88]	3957	11–17	School-based (6 secondary schools out of 18 in cluster sampling), Huangpu district, Guangzhou, South China	7	Cross-sectional	A, B
Withers *et al*[89]	2150	14–16	Family study: Family Health Services Authority (FHSA) and the Office of Population Consensus Surveys (OPC)	5	Cross-sectional	A, S
Yorulmaz *et al*[90]	883	11–19	School-based (28 middle and high school), Edirne, Turkey	6	Cross-sectional	A, B, C
Zhang *et al*[91]	2763	13.6 (mean)	School-based (4 junior high schools), Henan Province, China	7	Cross-sectional	A, B, C

*Potentially important confounders allowed for: A, age; B, gender; C, measure of socioeconomic status; D, ethnicity/race; E, educational achievement; J, peer smoking; S, measure of family structure; –, performed no adjustment.
NOS, Newcastle–Ottawa Scale.

specified confounder effect had similar marginal effects on the pooled estimate (OR 1.94, 95% CI 1.67 to 2.24, 12 studies).

Household smoking

Any household smoking (usually defined as either living with a smoker or that an adult smokes in the home) increased the risk by 1.92 (95% CI 1.70 to 2.16, 12 studies; I^2=80%; figure 8 in online supplement).[5 7 43 50 52 59 64 67 78–81] No significant differences in effect size were seen between the subgroup analyses for quality or definition for smoking status (test for subgroup differences, p=0.86 and p=0.17, respectively). Sensitivity analyses restricted to studies which adjusted for at least three of the specified confounders slightly reduced the pooled estimate (OR 1.69, 95% CI 1.54 to 1.87, 8 studies), but the effect remained highly statistically significant.

Publication bias

No evidence of publication bias was seen in the funnel plots for the relation between adolescent smoking and either parent smoking, both parents smoking, mother smoking, father smoking, and at least one parent smoking. However, there was some evidence of asymmetry in the funnel plot for sibling smoking and household smoking, where smaller sized studies showing protective effects on the risk of adolescent smoking seemed to be missing from the funnel plots (see figure 9 in online supplement).

Population attributable fraction

Health survey for England data indicate that, in 2007, around 22% of children aged up to 15 lived in a household in which someone smokes.[13] Using the OR for household smoking (1.92) as the estimated RR of smoking in these children, the proportion of smoking uptake among children up to the age of 15 likely to be attributable to exposure to smoking in the home is estimated at 17%. In 2008 there were about 675 000 15-year-old adolescents in England and Wales,[92] of whom 20% smoke[93]; a 17% attributable fraction translates into approximately 23 000 new smokers by age 15 arising from exposure to smoking in the home. Allowing for overestimation of the true RR from estimating pooled ORs,[18] the adjusted estimate of RR is then

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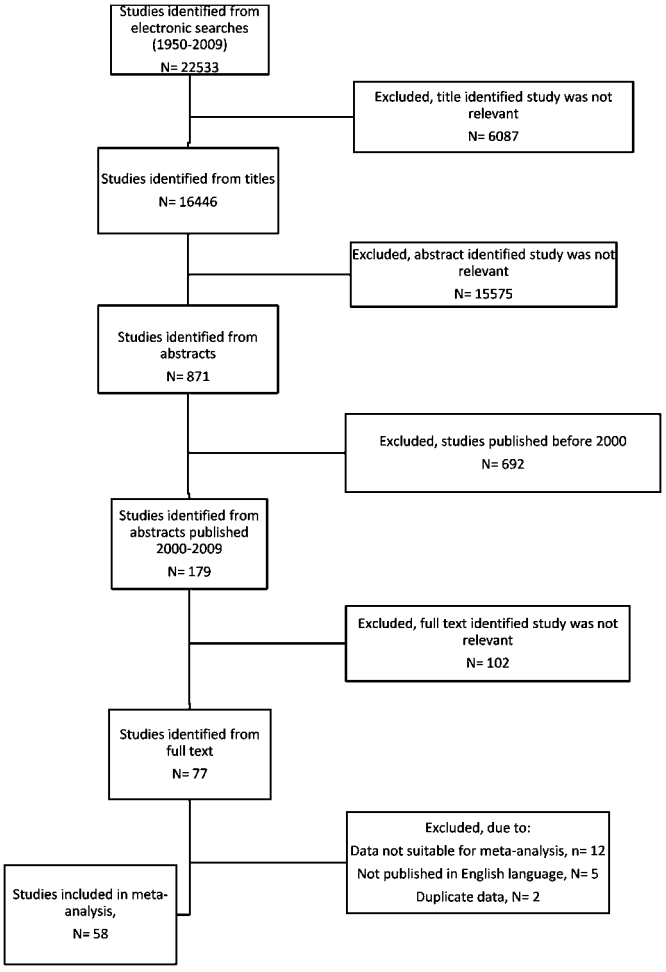


Figure 1 Flow chart of included studies.

approximately 1.67, the corrected population attributable fraction 13% and the estimated number of new smokers attributable to household exposure 17 000.

DISCUSSION

This study uses systematic review and meta-analysis methods to provide summary estimates of the effect of living with a family member who smokes on the risk that a child or young person takes up smoking. The analyses confirm that smoking by siblings, parents or other household members—and particularly by parents—is a strong and significant influence on smoking uptake. The effect of individual family member smoking is strongest for smoking by the mother, but is stronger still when both parents smoke, with a near threefold increase in risk. We estimate that around 23 000 adolescents who are smoking regularly at the age of 15 in England and Wales are doing so as a consequence of exposure to household smoking. The consequences of smoking uptake in these individuals are likely to include significant morbidity and premature mortality, and also the financial poverty arising from a regular habit which, at 20 cigarettes per day at current UK prices, costs around £2000 per year. For those who commence smoking at younger age, these impacts are likely to be all the greater since early uptake of smoking is also associated with higher levels of tobacco dependence[94] and heavier smoking.[5]

The great majority of the studies we assessed relied on self-reported smoking status which has been shown to be a good indicator of true smoking status in general, but may underestimate smoking in adolescents.[95] This may result in underestimation of the observed effects and also contribute to some of the observed heterogeneity between studies. A limitation of this meta-analysis was that high levels of heterogeneity were observed for some comparisons, which suggests that there are unexplained reasons for variation in the findings between studies over and above chance which may result in potentially misleading summary estimates. We investigated the reasons for heterogeneity by performing subgroup analyses and sensitivity analyses; however, these analyses revealed relatively consistent findings. Generally, the pooled results did not differ appreciably between studies of different methodological quality or in relation to the definition of smoking status of the adolescent. ORs were higher, however, for studies that measured ever having tried smoking, consistent with the fact that a substantial proportion of children and young people experiment with smoking but do not become regular addicted smokers. We assessed the effects on smoking status in adolescence rather than attempting to assess the effect on the stage transitions of tobacco use—where adolescents can be labelled as 'triers', 'experimenters', 'regular users' and 'dependent users'[96]—due to an insufficient number of studies being identified which categorised the stages of smoking. It is therefore difficult to determine at which stages the effects of parental and sibling smoking have more impact. Although inconsistent findings for a dose response association were seen in previous studies,[97] we found strong evidence of this, where adolescents were at a greater risk of smoking when both parents smoked than when only one parent smoked. We also found that the magnitude of the effect was stronger when the mother smoked than when the father smoked. This finding appears to be more pronounced in recent years,[97] and there was some evidence of a sex-specific effect where girls were influenced more by maternal than paternal smoking while the opposite applied to boys (data not shown). Further reasons for heterogeneity between the studies could be differences in approaches to adjustment for confounding; however, our sensitivity analyses restricted to studies which adjusted for multiple known confounding factors generally found the pooled results were very similar to the results from the unrestricted analyses. However, the pooled estimate for exposure to any household smoking was reduced from a pooled OR of 1.92 to 1.69, but the results remained highly statistically significant.

A limitation of our systematic review was that we excluded studies at the full text stage which were written in a non-English language, so the findings of the meta-analyses are biased towards English speaking countries. However, this exclusion criterion only resulted in five papers being excluded (conducted in France, the Netherlands, Slovakia, China and Austria), so the exclusion of these papers is unlikely to have a substantial impact on the conclusions of this study.

In the meta-analysis we estimated pooled ORs since 56 of the 58 included studies reported this measure of effect. Since smoking in adolescents is not rare, our estimated pooled ORs will overestimate the true RRs although, at an estimated 15%,[18] the magnitude of this effect is small. Thus, allowing for overestimation of the true pooled estimate has marginal effects on the estimated strength of the association. Allowing for the overestimation in the calculation for the population attributable fraction results in an estimated 17 000 new smokers by age 15 arising from exposure to smoking in the home.

The importance of this study is that, irrespective of the true magnitude of these effects, it identifies a further and to date

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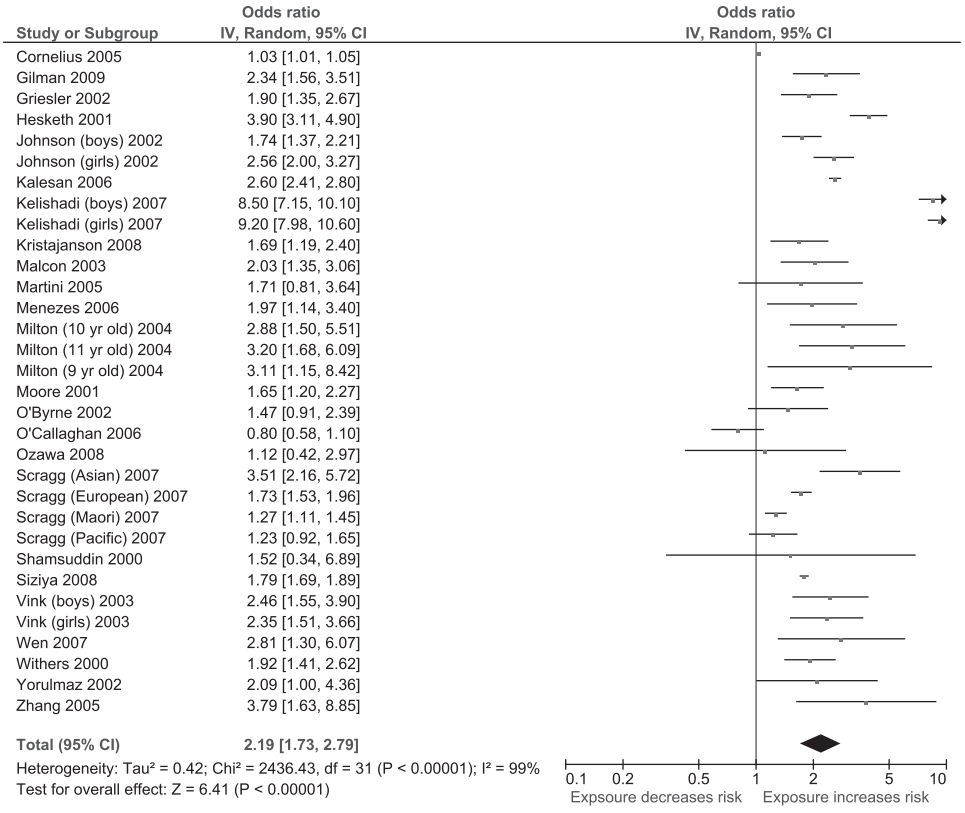


Figure 2 Mother smoking and the risk of adolescent smoking. Squares represent the estimate of effect and horizontal lines the 95% CIs from the individual studies. The centre of the diamond represents the pooled OR and the corner of the diamond the 95% CIs. ORs >1 indicate that exposure to mother smoking increases the risk of adolescent smoking.

neglected third-party impact of smoking behaviour. The health risks to children and adults arising from passive smoke exposure are now well recognised and extensively documented,[12 13] but arise directly and only from exposure to tobacco smoke. In this instance we identify an impact with the potential for substantial short- and long-term harm to health arising from the availability of cigarettes and/or the role model of smoking behaviour in the family. The number of children potentially affected is substantial, with around 2 million children in the UK living in a household that includes a smoker. This is a massive public health problem in its own right.

Effective measures to prevent smoking are well defined and promoted by the World Health Organization[98] and include a range of policies that will help to reduce the prevalence of smoking in younger adults—and hence the typical parents of younger children—and thus reduce exposure to smoking behaviour. However, they do not yet address the need to ensure that children grow up in a home that is not only smoke-free—to protect them from the direct health consequences of passive smoking—but also free of smokers—to protect them from the adverse effects of smoking role models since observation of parental smoking behaviour can have profound influences on the

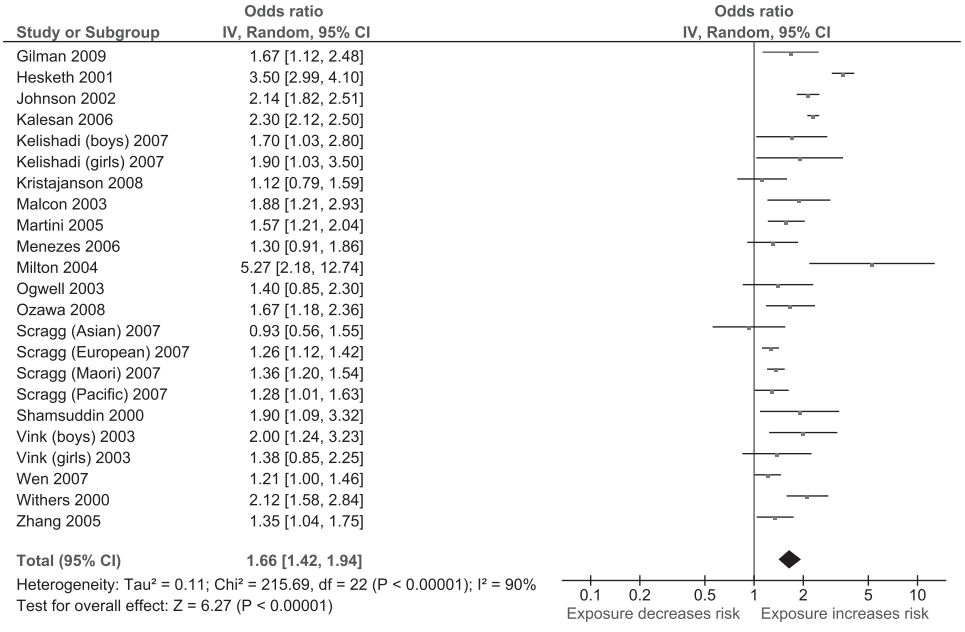


Figure 3 Father smoking and the risk of adolescent smoking. Squares represent the estimate of effect and horizontal lines the 95% CIs from the individual studies. The centre of the diamond represents the pooled OR and the corner of the diamond the 95% CIs. ORs >1 indicate that exposure to father smoking increases the risk of adolescent smoking.

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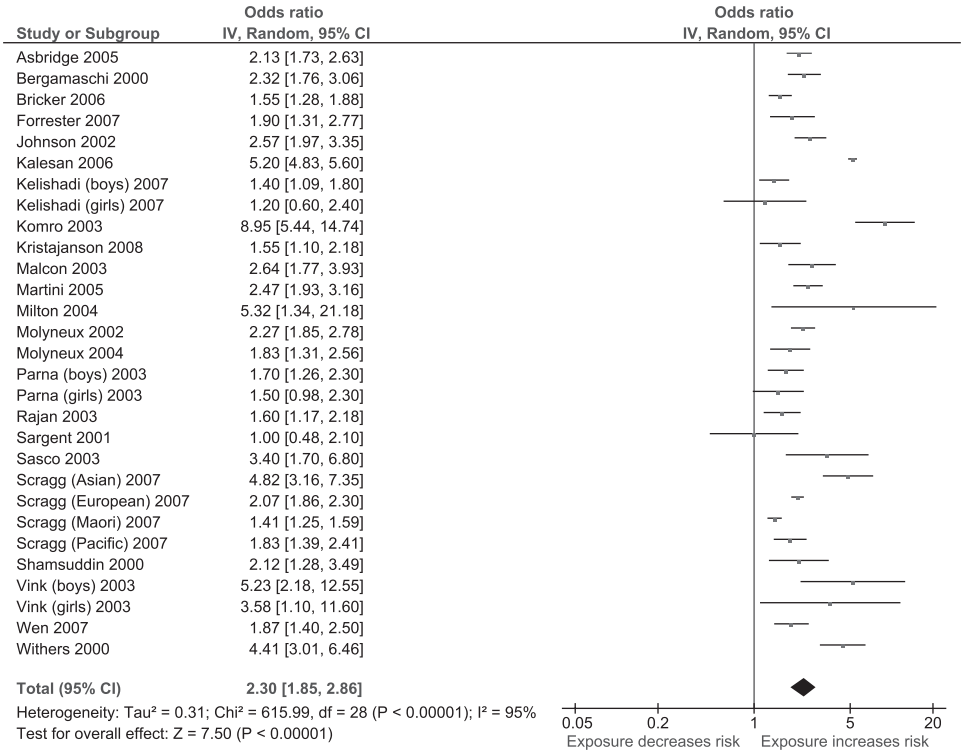


Figure 4 Sibling smoking and the risk of adolescent smoking. Squares represent the estimate of effect and horizontal lines the 95% CIs from the individual studies. The centre of the diamond represents the pooled OR and the corner of the diamond the 95% CIs. ORs >1 indicate that exposure to sibling smoking increases the risk of adolescent smoking.

perspectives of preschool children who view this behaviour as appropriate or normative in social situations.[99 100] Preventing this latter effect will demand further radical changes in public policy and behaviour, and in the acceptability of smoking in places where children are present. Protection of children from this major cause of harm needs to become a high public health priority.

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REFERENCES

1. **World Bank.** *Curbing the Epidemic Governments and the Economics of Tobacco Control*. Washington, DC: World Bank, 1999.
2. **Doll R,** Peto R, Boreham J, *et al*. Mortality in relation to smoking: 50 years' observations on male British doctors. *BMJ* 2004;**328**:1519—33.
3. **Hill KG,** Hawkins JD, Catalano RF, *et al*. Family influences on the risk of daily smoking initiation. *J Adolesc Health* 2005;**37**:202—10.
4. **Geckova A,** van Dijk JP, van Ittersum-Gritter T, *et al*. Determinants of adolescents' smoking behaviour: a literature review. *Cent Eur J Public Health* 2002;**10**:79—87.
5. **Pust S,** Mohnen SM, Schneider S. Individual and social environment influences on smoking in children and adolescents. *Public Health* 2008;**122**:1324—30.
6. **Kestila L,** Koskinen S, Martelin T, *et al*. Influence of parental education, childhood adversities, and current living conditions on daily smoking in early childhood. *Eur J Public Health* 2006;**16**:617—26.
7. **Griesbach D,** Amos A, Currie C. Adolescent smoking and family structure in Europe. *Soc Sci Med* 2003;**56**:41—52.
8. **de Vries H,** Engels R, Kremers S, *et al*. Parents' and friends' smoking status as predictors of smoking onset: findings from six European countries. *Health Educ Res* 2003;**18**:627—36.
9. **Kobus K.** Peers and adolescent smoking. *Addiction* 2003;**98**:37—55.
10. **Molyneux A,** Lewis S, Antoniak M, *et al*. Prospective study of the effect of exposure to other smokers in high school tutor groups on the risk of incident smoking in adolescence. *Am J Epidemiol* 2004;**159**:127—32.
11. **Wellman RJ,** Sugarman DB, DiFranza JR, *et al*. The extent to which tobacco marketing and tobacco use in films contribute to children's use of tobacco. A meta-analysis. *Arch Pediatr Adolesc Med* 2006;**160**:1285—96.
12. **Tobacco Advisory Group.** *Going Smoke-free. The Medical Case for Clean Air in the Home, at Work and in Public Places. Report on Passive Smoking by the Tobacco Advisory Group of the Royal College of Physicians.* London: Royal College of Physicians, 2005.
13. **Tobacco Advisory Group.** *Passive smoking and children. Report by the Tobacco Advisory Group*. London: Royal College of Physicians, 2010.
14. **Stroup DF,** Berlin JA, Morton SC, *et al*. Meta-analysis of observational studies in epidemiology: a proposal for reporting. Meta-analysis Of Observational Studies in Epidemiology (MOOSE) Group. *J Am Med Assoc* 2000;**283**:2008—12.
15. **Wells G,** Shea B, O'Connell D, *et al*. *Newcastle-Ottawa Scale (NOS) for Assessing the Quality of Non-randomised Studies in Meta-analysis*. http://www.ohri.ca/programs/clinical_epidemiology/oxford.htm (accessed Mar 2009).
16. **Higgins JP,** Thompson SG, Deeks JJ, *et al*. Measuring inconsistency in meta-analyses. *BMJ* 2003;**327**:557—60.
17. **The Nordic Cochrane Centre.** *Review Manager (RevMan)*. Copenhagen: The Cochrane Collaboration, 2008.
18. **Davies HT,** Crombie IK, Tavakoli M. When can odds ratios mislead? *BMJ* 1998;**316**:989.
19. **Andersen MR,** Leroux BG, Marek PM, *et al*. Mothers' attitudes and concerns about their children smoking: do they influence kids? *Prev Med* 2002;**34**:198—206.
20. **Bard DE,** Rodgers JL. Sibling influence on smoking behavior: a within-family look at explanations for a birth-order effect. *J Appl Soc Psychol* 2003;**33**:1773—95.
21. **Bricker JB,** Peterson AV, Andersen MR, *et al*. Parents' and older siblings' smoking during childhood: changing influences on smoking acquisition and escalation over the course of adolescence. *Nicotine Tob Res* 2007;**9**:915—26.
22. **den Exter Blockland EA,** Hale WW 3rd, Meeus W, *et al*. Parental support and control and early adolescent smoking: a longitudinal study. *Subst Use Misuse* 2007;**42**:2223—32.
23. **Effuso L,** Barra D, Del Castello E, *et al*. Factors influencing the age at which adolescents start smoking. A comparison between a big and a small city. *Ital Heart J* 2002;**3**:64—8.
24. **Faucher MA.** Why do girls smoke? A path analysis of mother-daughter connectedness, self-concept, race/ethnicity and smoking behavior of mothers and

Thorax: first published as 10.1136/thx.2010.153379 on 15 February 2011. Downloaded from http://thorax.bmj.com/ on March 29, 2024 by guest. Protected by copyright.

friends as contributors to smoking behavior in teenage girls. *Diss Abstr Int B Sci Eng* 2002; September.
25. **Harakeh Z,** Engles RCME, Vermulst AA, *et al*. The influence of best friends and siblings on adolescent smoking: a longitudinal study. *Psychol Health* 2007;**22**:269—89.
26. **Leiner M,** Medina I, Tondapu SR, *et al*. Smoking status of adolescents in two countries and the impact of the smoking status of mother, father, grandparents, and siblings. *J Sch Health* 2008;**78**:129—30.
27. **Ma GX,** Shive S, Legos P, *et al*. Ethnic differences in adolescent smoking behaviors, source of tobacco, knowledge and attitudes towards restriction policies. *Addict Behav* 2003;**28**:249—68.
28. **Madarasová Gecková A,** Stewart R, van Dijk JP, *et al*. Influence of socio-economic status, parents and peers on smoking behavior of adolescents. *Eur Addict Res* 2005;**11**:204—9.
29. **Otten R,** Engles RCME, van den Eijnden RJJM. General parenting, anti-smoking socialization and smoking onset. *Health Educ Res* 2008;**23**:859—69.
30. **Menezes AMB,** Hallal PC, Horta BL. Early determinants of smoking in adolescence: a prospective birth cohort study. *Cad Saúde Pública* 2007;**23**:347—54.
31. **Zhang L,** Wang W, Zhao Q, *et al*. Psychosocial predictors of smoking among secondary school students in Henan, China. *Health Educ Res* 2000;**15**:415—22.
32. **Courtois R,** Caudrelier N, Legay E, *et al*. Influence parentale (dépendance tabagique et styles éducatifs) sur la consommation et la dépendance tabagique de leur adolescent. *Médicale* 2007;**36**:1341—9.
33. **Den Exter Blokland E,** Engels RCME, Hale WW III, *et al*. Parental smoking cessation and smoking onset among early adolescents. *Gedrag Gezondheid: Tijdschr Psychologie Gezondheid* 2002;**30**:313—26.
34. **Geckova A,** Pudelsky M, van Dijk JP. Parental influence on smoking, alcohol consumption and sports activities of adolescents. *Ceska Slov Psychiatr* 2000;**96**:406—10.
35. **Xiaoyi F,** Danhua L. Prevention and intervention of adolescents smoking behavior. *Acta Psychologica Sinica* 2003;**35**:379—86.
36. **Zidek T,** Haidinger G, Zacharasiewicz A, *et al*. [Prevalence of smoking habits of Upper Austria students of the 7th and 8th grade and effect of smoking habits of family and peers]. *Soz Praventivmed* 2000;**45**:174—81.
37. **Asbridge M,** Tanner J, Wortley S. Ethno-specific patterns of adolescent tobacco use and the mediating role of acculturation, peer smoking, and sibling smoking. *Addiction* 2005;**100**:1340—51.
38. **Bauman KE,** Carver K, Gleiter K. Trends in parent and friend influence during adolescence. The case of adolescent cigarette smoking. *Addict Behav* 2001;**26**:349—61.
39. **Bergamaschi A,** Gambi A, Gentilini F, *et al*. Tobacco smoking among high school students in Romagna (Italy) and evaluation of a prevention campaign. *Subst Use Misuse* 2000;**35**:1277—95.
40. **Bricker JB,** Peterson AV, Leroux BG, *et al*. Prospective prediction of children's smoking transitions: role of parents' and older siblings' smoking. *Addiction* 2006;**101**:128—36.
41. **Cornelius MD,** Leech SL, Goldschmidt L, *et al*. Is prenatal tobacco exposure a risk factor for early adolescent smoking? A follow-up study. *Neurotoxicol Teratol* 2005;**27**:667—76.
42. **den Exter Blockland EA,** Engels RC, Hale WW 3rd, *et al*. Lifetime parental smoking history and cessation and early adolescent smoking behavior. *Prev Med* 2004;**38**:359—68.
43. **Elder JP,** Campbell NR, Litrownik AJ, *et al*. Predictors of cigarette and alcohol susceptibility and use among Hispanic migrant adolescents. *Prev Med* 2000;**31**:115—23.
44. **Fidler JA,** West R, van Jaarsveld CHM, *et al*. Smoking status of step-parents as a risk factor for smoking in adolescence. *Addiction* 2008;**103**:496—501.
45. **Fisher LB,** Winickoff JP, Camargo CA, *et al*. Household smoking restrictions and adolescent smoking. *Am J Health Promot* 2007;**22**:15—21.
46. **Forrester K,** Biglan A, Severson HH, *et al*. Predictors of smoking onset over two years. *Nicotine Tob Res* 2007;**9**:1259—67.
47. **Foster SE,** Jones DJ, Olson AL, *et al*. Family socialization of adolescent's self-reported cigarette use: the role of parents' history of regular smoking and parenting style. *J Pediatr Psychol* 2007;**32**:481—93.
48. **Gilman SE,** Rende R, Boergers J, *et al*. Parental smoking and adolescent smoking initiation: an intergenerational perspective on tobacco control. *Pediatrics* 2009;**123**: e274—81.
49. **Griesler PC,** Kandel DB, Davies M. Ethnic differences in predictors of initiation and persistence of adolescent cigarette smoking in the National Longitudinal Survey of Youth. *Nicotine Tob Res* 2002;**4**:79—93.
50. **Gritz ER,** Prokhorov AV, Hudmon KS, *et al*. Predictors of susceptibility to smoking and ever smoking: a longitudinal study in a triethnic sample of adolescents. *Nicotine Tob Res* 2003;**5**:493—506.
51. **Hesketh T,** Ding QJ, Tomkins A. Smoking among youths in China. *Am J Public Health* 2001;**91**:1653—5.
52. **Hollis JF,** Polen MR, Lichtenstein E, *et al*. Tobacco use patterns and attitudes among teens being seen for routine primary care. *Am J Health Promot* 2003;**17**:231—9.
53. **Jackson C.** Perceived legitimacy of parental authority and tobacco and alcohol use during early adolescence. *J Adolesc Health* 2002;**31**:425—32.
54. **Jackson C,** Dickinson D. Cigarette consumption during childhood and persistence of smoking through adolescence. *Arch Pediatr Adolesc Med* 2004;**158**:1050—6.
55. **Johnson CC,** Li D, Perry CL, *et al*. Fifth through eighth grade longitudinal predictors of tobacco use among a racially diverse cohort: CATCH. *J Sch Health* 2002;**72**:58—64.
56. **Johnson CC,** Myers L, Webber LS, *et al*. Profiles of the adolescent smoker: models of tobacco use among 9th grade high school students: Acadiana Coalition of Teens against Tobacco (ACTT). *Prev Med* 2004;**39**:551—8.
57. **Kalesan B,** Stine J, Alberg AJ. The joint influence of parental modeling and positive parental concern on cigarette smoking in middle and high school students. *J Sch Health* 2006;**76**:402—7; quiz 438—9.
58. **Kelishadi R,** Mokhtari MR, Tavasoli AA, *et al*. Determinants of tobacco use among youths in Isfahan, Iran. *Int J Public Health* 2007;**52**:173—9.
59. **Komro KA,** McCarty MC, Forster JL, *et al*. Parental, family, and home characteristics associated with cigarette smoking among adolescents. *Am J Health Promot* 2003;**17**:291—9.
60. **Kristjansson AL,** Sigfusdottir ID, Allegrante JP, *et al*. Social correlates of cigarette smoking among Icelandic adolescents: a population-based cross-sectional study. *BMC Public Health* 2008;**8**:86.
61. **Malcon MC,** Menezes AM, Maia Mde F, *et al*. [Prevalence of and risk factors for cigarette smoking among adolescents in South America: a systematic literature review]. *Pan Am J Public Health* 2003;**13**:222—8.
62. **Martini S,** Sulistyowati M. *The determinants of smoking behavior among teenagers in East Java Province, Indonesia. In: Health, Nutrition and Population (HNP) Economics of Tobacco Control Discussion Paper no 32*, 2005.
63. **Menezes AMB,** Goncalves H, Anselmi L, *et al*. Smoking in early adolescence: evidence from the 1993 Pelotas (Brazil) Birth Cohort Study. *J Adolesc Health* 2006;**39**:669—77.
64. **Milton B,** Cook PA, Dugdill L, *et al*. Why do primary school children smoke? A longitudinal analysis of predictors of smoking uptake during pre-adolescence. *Public Health* 2004;**118**:247—55.
65. **Molyneux A,** Lewis S, Antoniak M, *et al*. Is smoking a communicable disease? Effect of exposure to ever smokers in school tutor groups on the risk of incident smoking in the first year of secondary school. *Tob Control* 2002;**11**:241—5.
66. **Moore L,** Roberts C, Tudor-Smith C. School smoking policies and smoking prevalence among adolescents: multilevel analysis of cross-sectional data from Wales. *Tob Control* 2001;**10**:117—23.
67. **Nichols TR,** Birnbaum AS, Birnel S, *et al*. Perceived smoking environment and smoking initiation among multi-ethnic urban girls. *J Adolesc Health* 2006;**38**:369—75.
68. **O'Byrne KK,** Haddock CK, Poston WSC, *et al*. Parenting style and adolescent smoking. *J Adolesc Health* 2002;**30**:418—25.
69. **O'Callaghan FV,** O'Callaghan M, Najman JM, *et al*. Prediction of adolescent smoking from family and social risk factors at 5 years, and maternal smoking in pregnancy and at 5 and 14 years. *Addiction* 2006;**101**:282—90.
70. **Ogwell AE,** Astrom AN, Haugejorden O. Socio-demographic factors of pupils who use tobacco in randomly-selected primary schools in Nairobi province, Kenya. *East Afr Med J* 2003;**80**:235—41.
71. **Otten R,** Engels RC, van de Ven MO, *et al*. Parental smoking and adolescent smoking stages: the role of parents' current and former smoking, and family structure. *J Behav Med* 2007;**30**:143—54.
72. **Ozawa M,** Washio M, Kiyohara C. Factors related to starting and continuing smoking among senior high school boys in Fukuoka, Japan. *Asian Pac J Cancer Prev* 2008;**9**:239—45.
73. **Pärna K,** Rahu K, Fischer K, *et al*. Smoking and associated factors among adolescents in Tallinn, Helsinki and Moscow: a multilevel analysis. *Scand J Public Health* 2003;**31**:350—8.
74. **Rajan KB,** Leroux BG, Peterson AV, *et al*. Nine-year prospective association between older siblings' smoking and children's daily smoking. *J Adolesc Health* 2003;**33**:25—30.
75. **Rozi S,** Akhtar S, Ali S, *et al*. Prevalence and factors associated with current smoking among high school adolescents in Karachi, Pakistan. *Southeast Asian J Trop Med Public Health* 2005;**36**:498—504.
76. **Sargent JD,** Dalton M. Does parental disapproval of smoking prevent adolescents from becoming established smokers? *Pediatrics* 2001;**108**:1256—62.
77. **Sasco AJ,** Merrill RM, Benhaim-Luzon V, *et al*. Trends in tobacco smoking among adolescents in Lyon, France. *Eur J Cancer* 2003;**39**:496—504.
78. **Scragg R,** Laugesen M, Robinson E. Parental smoking and related behaviours influence adolescent tobacco smoking: results from the 2001 New Zealand national survey of 4th form students (see comment). *NZ Med J* 2003;**116**:U707.
79. **Scragg R,** Laugesen M. Influence of smoking by family and best friend on adolescent tobacco smoking: results from the 2002 New Zealand national survey of year 10 students. *Aust NZ J Public Health* 2007;**31**:217—23.
80. **Shamsuddin K,** Haris MA. Family influence on current smoking habits among secondary school children in Kota Bharu, Kelantan. *Singapore Med J* 2000;**41**:167—71.
81. **Simons-Morton B,** Haynie DL, Crump AD, *et al*. Peer and parent influences on smoking and drinking among early adolescents. *Health Educ Behav* 2001;**28**:95—107.
82. **Siziya S,** Muula AS, Rudatsikira E. Prevalence and correlates of current cigarette smoking among adolescents in East Timor-Leste. *Indian Pediatr* 2008;**45**:963—8.
83. **Siziya S,** Ntata PR, Rudatsikira E, *et al*. Sex differences in prevalence rates and predictors of cigarette smoking among in-school adolescents in Kilimanjaro, Tanzania. *Tanzan Health Res Bull* 2007;**9**:190—5.

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84. **Siziya S,** Rudatsikira E, Muula AS, *et al*. Predictors of cigarette smoking among adolescents in rural Zambia: results from a cross sectional study from Chongwe district. *Rural Remote Health* 2007;**7**:728.
85. **Szabo E,** White V, Hayman J. Can home smoking restrictions influence adolescents' smoking behaviors if their parents and friends smoke? *Addict Behav* 2006;**31**:2298–303.
86. **Tyc VL,** Hadley W, Allen D, *et al*. Predictors of smoking intentions and smoking status among nonsmoking and smoking adolescents. *Addict Behav* 2004;**29**:1143–7.
87. **Vink JM,** Willemsen G, Boomsma DI. The association of current smoking behavior with the smoking behavior of parents, siblings, friends and spouses. *Addiction* 2003;**98**:923–31.
88. **Wen X,** Chen W, Muscat JE, *et al*. Modifiable family and school environmental factors associated with smoking status among adolescents in Guangzhou, China. *Prev Med* 2007;**45**:189–97.
89. **Withers NJ,** Low JL, Holgate ST, *et al*. Smoking habits in a cohort of UK adolescents. *Respir Med* 2000;**94**:391–6.
90. **Yorulmaz F,** Akturk Z, Dagdeviren N, *et al*. Smoking among adolescents: relation to school success, socioeconomic status nutrition and self-esteem. *Swiss Med Wkly* 2002;**132**:449–54.
91. **Zhang L,** Wang WF, Zhou G. A cross-sectional study of smoking risk factors in junior high school students in Henan, China. *Southeast Asian J Trop Med Public Health* 2005;**36**:1580–4.
92. **Office for National Statistics.** *Mortality Statistics—Deaths Registered in 2008*. London: Office for Public Sector Information. 2009. http://www.statistics.gov.uk/statbase/Product.asp?vlnk=15096.
93. **Health and Social Care Information Centre.** *Drug Use, Smoking and Drinking among Young People in England in 2008*. London: Health and Social Care Information Centre, 2009.
94. **Huang HL,** Hsu CC, Magnus JH, *et al*. Perceived smoking prevalence at school-level and children's smoking initiation: a multi-level analysis of a cross-sectional data in Taiwan. *Health Policy* 2008;**86**:213–21.
95. **Newell S,** Girgis A, Sanson-Fisher R. The accuracy of self-reported health behaviors and risk factors relating to cancer and cardiovascular disease in the general population: a critical review. *Am J Prev Med* 1999;**17**:211–29.
96. **Mayhew KP,** Flay BR, Mott JA. Stages in the development of adolescent smoking. *Drug Alcohol Depend* 2000;**59**:S61–81.
97. **Avenevoli S,** Merikangas KR. Familial influences on adolescent smoking. *Addiction* 2003;**98**:1–20.
98. **World Health Organization.** *WHO Framework Convention on Tobacco Control*. Geneva: World Health Organization, 2003.
99. **Dalton MA,** Bernhardt AM, Gibson JJ, *et al*. Use of cigarettes and alcohol by preschoolers while role-playing as adults. *Arch Pediatr Adolesc Med* 2005;**159**:854–9.
100. **de Leeuw RNH,** Engles RCME, Scholte RHJ. Parental smoking and pretend smoking in young children. *Tob Control* 2010;**19**:200–5.

Journal club

Genetic susceptibility and resistance to tuberculosis

This study investigated possible biomarkers of susceptibility and resistance to *Mycobacterium tuberculosis*, identifying gene expression profiles associated with active tuberculosis (TB). Among a South African cohort, genome-wide transcription profiles of whole blood were obtained from 33 TB patients, 34 healthy donors latently infected with *M tuberculosis* (LTBI) and 9 healthy non-infected donors (NIDs). Cluster analysis of genes demonstrated pronounced differences among TB patients compared with the LBTI and NID groups, with no significant difference in clustering between the LBTI and NID groups.

Reverse transcriptase PCR identified Fc γ receptor 1B (FCGR1B) as the most strongly differentially expressed gene in the TB group compared to LBTI. This gene, combined with four other most prominently differentiated genes—CD64, Lactoferrin, guanylate binding protein 5 and Granzyme A—allowed discrimination between TB and LTBI groups with a sensitivity of 94% (30/32 patients) and specificity of 97% (33/34 patients). Functional annotation clustering demonstrated distinct differences between TB and LBTI groups with increased expression of macrophage-associated genes and reduced expression of natural killer-associated genes as well as reduced apoptosis in TB patients compared to the LBTI group.

This research demonstrates the importance of genetic control over the innate immune system in the development of active TB. Defining a genetic biosignature of resistance or susceptibility to *M tuberculosis* identifies targets for future drugs and vaccines, an approach critically important in reducing the significant burden of TB disease worldwide.

► **Maertzdorf J**, Repsilber D, Parida SK, *et al*. Human gene expression profiles of susceptibility and resistance in tuberculosis. *Genes Immun* 2011;**12**:15–22.

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