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Why has adolescent smoking declined dramatically? An investigation of changing exposure to risk factors using analysis of repeat cross-sectional data from New Zealand 2002-2015.

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ABSTRACT

Objectives: Adolescent smoking has declined in New Zealand and in many other developed countries since the late 1990s, yet the drivers of the decline are not well understood. We set out to determine i) whether changes in exposure to known risk factors account for the decline, ii) whether relationships between risk factors and smoking outcomes have changed over time.

Design: Analysis of repeat cross-sectional data from a nationally representative survey.

Setting: New Zealand

Participants: Secondary school students aged 14-15**.** N=20,443 - 31,833 per year.

Outcome measure: Regular (at least monthly) smoking

Methods: We analysed smoking prevalence and exposure to risk factors (one or more parents smoke, best friend smokes, older sibling(s) smoke, and past week exposure to smoking in the home), 2002-2015. For each risk factor we calculated annual average change in exposure, and odds ratios (OR) for each year, overall and for Māori.

Results: There were declines in adolescent smoking (absolute annual average decrease 1.2%), exposure to best friend smokes (1.5%), older sibling smokes (0.7%), daily exposure to smoking in the home (0.6%), maternal (0.5%) and paternal (0.5%) smoking. The adjusted OR for exposure to smoking in the home and best friend smokes increased markedly over the study period while the OR for parental smoking remained unchanged and was not a significant independent risk factor in most years. Trends were similar overall and for Māori.

Conclusions: Changes in parental smoking do not account for smoking decline among adolescents. Decreasing exposure to daily smoking in the home and older sibling smoking

have likely contributed. Smoking status of best friend remains the strongest risk factor, with exposure falling dramatically at the individual level, in line with population declines in adolescent smoking since 2002. Further research is needed to identify additional factors, outside our model, that appear to be driving this trend.

ARTICLE SUMMARY

Strengths and limitations of this study

- The study is original because little research has explored explanations for the decline in adolescents smoking, examined trends in exposure to risk factors for adolescent smoking, or investigated the changing relationship between risk factors and smoking outcomes over time.
- The survey has a large sample size (N=20,443 - 31,833 per year), allowing accurate population estimates of smoking prevalence and exposure to risk factors.
- Due to data limitations, the study only includes a small number of risk factors.
- However the risk factors included have consistently been found to be among the strongest and most important predictors of adolescent smoking.

INTRODUCTION

Smoking is a leading cause of preventable illness and premature death[1] and a key driver of health disparities between ethnic and socioeconomic groups.[2 3] Long-term tobacco use typically begins with experimental smoking in adolescence,[4] and, internationally, considerable research and policy attention has focused on understanding and preventing smoking uptake in this age group.

The dramatic decline in adolescent smoking observed since the late 1990s in many high income countries is good news from a public health perspective. In New Zealand (NZ), for example, regular smoking (defined as at least monthly) among 14-15 year olds declined from a peak of 29% in 1999 to 6% in 2014, with decreases across all main ethnic and socioeconomic groups, and a convergence between boys and girls over the period.[5] However, as in other countries, ethnic disparities remain pronounced with Māori (indigenous) smoking prevalence in this age group at 13.2% compared 4.2% among non-Māori in 2014.[6] In the context of such marked disparities, understanding the drivers of smoking decline among Māori adolescents is particularly important, to inform policies and programmes aimed at further reducing smoking in this priority group.

Other countries including the USA, England and Australia have also experienced a decline in teen smoking from the late 1990s, following a sharp rise in the early 1990s.[7] It is important to understand the causes of this decline in order to help ensure it is sustained, and to enable replication in other countries. Yet little research has focused on explaining this phenomenon. Public health interventions such as increases in tobacco tax or smokefree environment legislation may have played a role,[8-10] but they do not fully explain the observed trends, since declines in adolescent smoking have occurred in countries with widely differing regulatory contexts. This, and the fact that other adolescent risk behaviours (e.g. alcohol use, teen pregnancy) have also declined over a similar time period,[7] suggests broader socio-cultural changes rather than specific tobacco control policies may contribute to this international trend.

Such shifts could be generated by new technologies, for example. When the use of cell phones rose and smoking fell among adolescents in the late 1990s, a causal association was hypothesised.[11] More recently, attention has turned to other new technologies – smartphones and social media - and their potential role in driving generational change in attitudes and

behaviour.[12] There is face validity to the idea that these new technologies may have changed the way young people socialise or project their identity, displacing the role of smoking or providing less opportunity for it. However, this hypothesis is challenged by a consistent body of evidence showing a *positive* association between smoking and cell phone/ internet/social media use at the individual level.[13-22]

Other major changes since the mid-90s that could potentially impact on youth behaviour include changes in parenting;[23-25] changes in the school environment and climate; [26-28] and broad economic and labour market conditions resulting in young people leaving school and achieving independence later than previous cohorts.[29 30] But before exploring these novel explanations for smoking decline, it is important to determine the extent to which the observed trends can be explained by changing exposure to known predictors of smoking initiation.

Proximal risk factors for adolescent smoking have been studied extensively. For example parental, sibling and peer smoking have consistently been identified as key risk factors,[4 31] with the Surgeon General's 2012 review concluding that the evidence is suggestive of a causal role for peer influences, and a potential causal role for parental smoking.[4] The review found that smoking by older siblings influences smoking in adolescents more consistently than does smoking by parents.[4] Exposure to smoking in the home, although a less studied factor, has also been shown to be strongly related to the likelihood of smoking in adolescents independent of parental smoking status.[32-36] Studies suggest second hand smoke exposure may biologically predispose children to nicotine dependence [37-41] in addition to providing pro-smoking socialization.[33]

Despite extensive risk factor research, few studies have explored how exposure to risk factors has changed over time, whether the relationships between established risk factors and outcomes

have changed over time, or how such changes may be contributing to changes in adolescent smoking at the population level. Assuming that an observed individual-level relationship between a risk factor and adolescent smoking is causal, then declining exposure to that risk factor over time at the population level (while the strength of association is maintained or increased at the individual level) must contribute to a population decline in adolescent smoking.

The current study explores trends in exposure of 14-15 year olds to key risk factors – parental, sibling and peer smoking and exposure to smoking in the home - and their relationship to adolescent smoking from 2002 to 2015 in New NZ. We sought to better understand the potential drivers of adolescent smoking decline, overall and for Māori. Our study also updates regular smoking trends (overall, and by ethnicity) using 2015 data.

METHODS

We analysed repeat cross-sectional data from the ASH Year 10 Snapshot Survey series, an annual school-based survey of 14-15 year olds in NZ, which is part of the New Zealand Youth Tobacco Monitor. The questionnaire includes a set of 'core' questions that have remained consistent over time to enable trend monitoring, and additional questions which change from year to year. Years included in the current study were 2002-2015, since key variables of interest were unavailable prior to 2002. Furthermore, exposure to smoking in the home was not included in the questionnaire in 2002 or 2004-5, and therefore multivariable analysis includes only data from 2003 and 2006-15.

The ASH Year 10 Snapshot is a census-style survey, meaning all public and private schools with Year 10 students are eligible and invited to participate each year. School response rates range from 44-67% ,[42] and overall response rates from 36% to 54% (Table 1). The sample (N=20,000 – 32,000 per year) comprises approximately half the Year 10 population each year, and closely resembles the Year 10 population, albeit with modest but consistent under-

representation of Māori and low decile students. Detailed comparison of the sample and population by year is available in a supplementary file.

Table 1. Sample size and student response rate by year

Year	NZ Year 10 population	Valid responses included in study	% Year 10 population included in study
2002	58,812	28,088	50%
2003	61,028	31,377	54%
2004	62,852	30,807	46%
2005	64,619	31,833	51%
2006	63,086	31,690	52%
2007	62,012	25,109	42%
2008	61,485	29,682	50%
2009	61,355	24,755	42%
2010	61,210	31,696	54%
2011	59,562	26,028	45%
2012	59,627	30,396	43%
2013	57,929	27,014	49%
2014	59,612	29,303	47%
2015	59,528	20,443	36%
Total	**852,717**	**398,221**	**47%**

Aside from changes to non-core questions, the method for the survey has been reasonably consistent across included years. However fieldwork was undertaken earlier in the year in 2011 and subsequently, meaning respondents were slightly younger on average in 2011 and subsequent years, than in 2010 and prior years.

In participating schools, the one-page survey is completed in class time under the supervision of teaching staff. Individual students may choose not to participate. To protect the confidentiality of students' responses, identifying information is not collected, and teachers are requested not to check the completed surveys. Completed surveys are returned to ASH, which oversees data entry, cleaning and coding.

The survey was approved, as a component of the NZ Youth Tobacco Monitor, by the Ministry of Health Multiregional Health and Disability Ethics Committee in 2007. Further details on survey methodology are available elsewhere.[42]

Variables

The outcome variable, 'regular smoking' (Y/N) was defined as smoking at least monthly, based on the question 'How often do you smoke now?'

Smoking status of mother, father, older sibling(s), and best friend were based on the question 'Which of the following people smoke?' with a dichotomous variable (current smoker, yes/no) created for each. In addition, parental smoking was grouped into one variable for the purposes of multivariable analysis, coded 0 = neither parent smokes, 1 = only mother smokes, 2 = only father smokes, 3 = both parents smoke.

Past week exposure to smoking in the home was based on the question 'During the past 7 days, on how many days have people smoked around you in your home?' Response categories were 0 days, 1-2 days, 3-4 days, 5-6 days and 7 days. 'Daily exposure' was defined as 7 days, while 'any exposure' was defined as 1-7 days.

Demographic variables were age (14 or 15 years old), sex (male or female), ethnicity (prioritised Māori, Pacific, Asian, NZ European/other (NZEO); and dichotomised into Māori and non-Māori) and school decile. School decile is a school-level measure of the socio-economic position of a school's student community.[43] We grouped school decile into low (deciles 1-3: most deprived), medium (4-7), and high (8-10: least deprived). Each school also had an identification number (school ID) which was assigned to all respondents from that school.

Analysis

Following previously published ASH analyses, analysis was restricted to respondents aged 14 or 15 at the time of the survey. For consistency between univariate and multivariable (adjusted) analyses, only respondents with complete data for all variables (smoking status, parental smoking, sibling smoking, best friend smoking, age, gender, ethnicity, school decile, and school ID) were included in the analyses. In addition, only schools with at least 20 respondents were included.

We used SPSS (IBM Corp. Released 2016. IBM SPSS Statistics for Windows, Version 24.0. Armonk, NY: IBM Corp.) to tabulate prevalence of regular smoking and prevalence of exposure to risk factors (overall and by sex, ethnicity and school decile) for each year. We then quantified the mean annual absolute change in proportion of respondents exposed to each risk factor using weighted linear regression with year as the independent variable. The weights were $1/SD^2$ of the proportions.

Logistic regression (PROC Glimmix) using SAS/STAT software, (Version 9.4 of the SAS system for Windows. Copyright © 2002-2012, SAS Institute Inc., Cary, NC, USA.) was used to calculate univariate odds ratios (OR) to determine the strength of the relationship between each risk factor and regular smoking for each year, overall and for Māori. To adjust for potential clustering at the school level, school ID was entered as a random effect in the estimation of odds ratios. Multivariable logistic regression analysis (again with a random effect of school) was then conducted to assess the independent influence of each risk factor on regular smoking for each year, overall and for Māori, adjusting for sex, age, school decile and the other risk factors in the model.

RESULTS

Prevalence of regular smoking

We found that the long-term decline in prevalence of regular smoking among 14-15 year olds continued in 2015, to 5.4% overall (Fig 1a), and 11.1% for Māori (Fig 1b). Based on weighted linear regression, the overall regular smoking rate reduced by an average of 1.2 % per year (in absolute terms) from 2002-2015.

Changes in exposure to risk factors over time

Trends in exposure to risk factors are shown in Figure 1. Parental smoking (Fig 1a and 1b) declined only modestly over the study period, compared with the marked decline in regular smoking in 14-15 year olds; maternal and paternal smoking both declined by an average rate of 0.5% per annum. As shown in Figures 1b and 1c, smoking among older siblings declined slightly more, at an average rate of 0.7% per annum, but only 'best friend smokes' declined at a similar rate to regular smoking in 14-15 year olds (1.5% per annum). The change in exposure to smoking in the home (1-7 days) over the study period was not statistically significant, but daily (7 day) exposure fell from 22% to 13% overall (an average decrease of 0.6% per annum), and 38% to 25% for Māori. For Māori, trends in exposure to risk factors follow a similar pattern to overall trends but at markedly higher levels of exposure, as shown in Figures 1b and 1d.

Changes in the relationship between risk factors and regular smoking over time

As shown in Table 2, univariate analysis confirmed that smoking status of best friend, older sibling(s), and parents were all significant risk factors for smoking in 14-15 year olds in 2003 and 2015 at the individual level (overall and for Māori), as was exposure to smoking in the home. After adjusting for age, sex, school decile and risk factors in the model, strong and statistically significant associations with smoking status of best friend and older sibling(s) and exposure to smoking in the home persisted, but parental smoking had only a weak or non-significant association with the likelihood of regular smoking.

Table 2: Odds ratios for regular smoking among adolescents, 2003 and 2015, overall and for Māori.

	2003			2015		
	OVERALL					
	N=31,377			N=20,443		
	Prevalence of exposure (%)	Odds ratio (unadjusted)	Adjusted Odds Ratio	Prevalence of exposure (%)	Odds Ratio (unadjusted)	Adjusted Odds Ratio
No exposure to smoking in the home (0 days)	70.2	1.0	1.0	71.2	1.0	1.0
Daily exposure to smoking in the home	21.6	3.8 (3.6-4.1)	1.8 (1.7-2.0)	12.9	9.0 (7.7-10.5)	3.3 (2.7-4.1)
Any (1-7 days) exposure to smoking in the home#	29.8	3.3 (3.1-3.5)	1.7 (1.5-1.8)	28.8	6.3 (5.5-7.2)	2.6 (2.1-3.1)
Neither parent smokes	59.6	1.0	1.0	67.1	1.0	1.0
Only mother smokes	11.7	2.7 (2.4-2.9)	1.3 (1.2-1.5)	9.8	3.9 (3.2-4.7)	1.2 (1.0-1.5)*
Only father smokes	14.4	1.9 (1.7-2.0)	1.3 (1.2-1.4)	11.9	2.5 (2.1-3.1)	1.2 (0.9-1.5)*
Both parents smoke	14.3	4.2 (3.9-4.6)	1.3 (1.2-1.5)	11.2	6.2 (5.3-7.3)	1.2 (1.0-1.5)*
Older sibling smokes	24.9	3.6 (3.4-3.8)	2.1 (1.9-2.2)	16.6	4.7 (4.1-5.3)	1.7 (1.5-2.0)
Best friend smokes	28.5	11.4 (10.7-12.1)	8.4 (7.9 -9.0)	9.3	19.6 (17.0-22.5)	11.8 (10.1-13.6)
	MĀORI					
	N=5,425			N=4,215		
No exposure to smoking in the home (0 days)	52	1.0	1.0	50.6	1.0	1.0
Daily exposure to smoking in the home	38.0	2.8 (2.4-3.1)	1.9 (1.6-2.2)	25.3	6.9 (5.3-9.0)	4.2 (3.0-5.9)
Any (1-7 days) exposure to smoking in the home#	47.6	2.6 (2.3-2.9)	1.8 (1.6-2.1)	49.4	5.2 (4.1-6.6)	3.4 (2.5-4.5)
Neither parent smokes	36.0	1.0	1.0	43.6	1.0	1.0
Only mother smokes	20.1	2.0 (1.7-2.3)	1.3 (1.1-1.6)	17.5	2.2 (1.6-2.9)	0.8 (0.5-1.1)*
Only father smokes	13.8	1.9 (1.6-2.3)	1.5 (1.2-1.8)	14.0	1.8 (1.3-2.5)	0.9 (0.6-1.3)*
Both parents smoke	30.2	2.7 (2.3-3.1)	1.2 (1.0-1.4)*	24.8	3.7 (2.9-4.7)	0.9 (0.7-1.3)*

Older sibling smokes	42.5	2.4 (2.1-2.7)	1.6 (1.4-1.9)	32.6	2.9 (2.4-3.5)	1.5 (1.2-1.9)
Best friend smokes	46.0	6.8 (6.0-7.8)	5.5 (4.8-6.3)	18.5	11.2 (9.0-13.4)	8.2 (6.5-10.2)

Notes: model includes age, sex, ethnicity, decile (low, medium, high), smoking status of parents (none, mother, father, both), older sibling, best friend, and days exposed to smoking in the home (0, 1-2, 3-4, 5-6,7) except:

model uses exposure to smoking in the home (any/none) instead of days exposed to smoking in the home (0, 1-2, 3-4, 5-6,7)

Models for Māori students only do not include ethnicity

All Odds Ratios are statistically significant ($p<.001$) except those marked with * which did not reach statistical significance at the 95% level.

Confidence intervals for prevalence are uniformly narrow, and have been omitted to aid the legibility of the table.

We found that some risk factors had an increasing strength of association with smoking over time. As shown in Table 2 and Figure 2, the adjusted odds ratio for daily exposure to smoking in the home (OR 1.8 (1.7-2.0) in 2003, OR 3.3 (2.7-4.1) in 2015) and best friend smoking (8.4 (7.9 -9.0) in 2003; 11.8 (10.1-13.6) in 2015) increased markedly over the study period. In contrast, there was little change in adjusted ORs for parental and sibling smoking between 2003 and 2015. A similar pattern was seen in both the sample overall, and in Māori (not shown).

DISCUSSION

We found that the longstanding decline in 14-15 year old smoking in NZ continued in 2015. Best friend smoking declined at a similar rate to 14-15 year old smoking during the 2002-2015 period and exposure to other risk factors decreased more modestly. There was no change in past week exposure to smoking in the home at the 1-7 days level, but daily exposure fell significantly. With the exception of parental smoking, all the risk factors examined were significant independent risk factors for adolescent smoking in all years. The strength of association (adjusted OR) between adolescent smoking and certain risk factors - exposure to smoking in the home and best friend smoking - increased markedly over the study period, while other relationships remained unchanged. Trends were similar in Māori and the population overall, although Māori adolescents were exposed to all risk factors at markedly higher rates than the general population, which accounts for higher adolescent smoking prevalence in this group.

The aim of the study was to determine whether changing exposure to known risk factors explained the dramatic decline in adolescent smoking seen recently in NZ. Based on our results, we can conclude that declines in daily exposure to smoking inside the home, older sibling smoking and best friend smoking were all likely contributors, as each was

independently associated with regular smoking (with previous research suggestive of a causal relationship) and prevalence of exposure declined over the study period. Our findings do not support the idea that reduced smoking prevalence among parents was a significant driver of adolescent smoking decline. In most years parental smoking was not a statistically significant independent predictor of adolescent smoking, so we can conclude that (modest) declines in exposure to parental smoking have made little if any contribution. Our findings, consistent with previous research,[33-36] suggest it is exposure to smoking in the home, rather than parental smoking per se, which influences smoking uptake in this age group. Our findings add to a growing body of research suggesting that exposure to second hand smoke may predispose young people to tobacco use and nicotine dependency via biological, psychological and/or social mechanisms.[44] Greater research and policy attention to smokeefree homes (and cars) as part of a comprehensive smoking prevention approach is therefore warranted.

The smoking status of respondents' best friend was by far the strongest predictor of regular smoking in 14-15 year olds, with the association strengthening over the study period. It is important to note that this association may be partly due to smokers seeking out other smokers as friends, as well as via a probable causal influence.[4] However, at the population level, it would be a circular to suggest that declining best friend smoking was driving the decline in prevalence of adolescent smoking, since survey respondents and their best friends belong to the same cohort. The question remains: if decreases in best friend smoking are resulting in reduced risk of adolescent smoking at the individual level, what is driving the decline in best friend smoking? One possibility is that, since younger adolescents are strongly influenced by adolescents slightly older then themselves (including siblings), a virtuous cycle may have developed whereby a decline in smoking in one year group has led to lower smoking uptake in the subsequent year group, and so on. Further research, perhaps drawing

on communicable disease methodology, could test this hypothesis and explore how the 'social transmission' of smoking (and other health risk behaviours) influences population prevalence over time. Should this hypothesis prove to be correct, the trigger for the sudden change from rapidly rising to rapidly falling adolescent tobacco use from the late 1990s to the early 2000s still remains to be identified.

It is possible that changes in the social meaning of smoking[45 46] and the policy context[10 47] may have played a role. For example, policy responses to rising adolescent smoking in the 1990s may have influence teen smoking in NZ, as they appear to have done in Australia[9 10] and the US.[8] Smoking in NZ 14-15 year olds peaked in 1999, two years after the legal age of tobacco purchase was raised from 16 to 18 years of age, and a year after a 1998 tax increase which raised the price of a packet of 20 cigarettes by 13%.[48] The turning point in adolescent smoking also closely followed the 'Why start?' mass media campaign targeting youth which ran from 1996-1998. It is plausible that, collectively, these measures contributed to the denormalisation of smoking, which, rather than any specific intervention, may have been the trigger for adolescent smoking decline both in NZ and other jurisdictions. As Simon Chapman has pointed out, denormalisation involves an 'interplay of continuous, uncontrolled, unmeasured, and sometimes unmeasurable variables intended to influence [tobacco] consumption', and cannot be reduced to the sum of its parts.[49] However if tobacco denormalisation, along with the other factors discussed above, explains the decline in adolescent smoking, is it simply a coincidence that teen alcohol use, teen pregnancy and juvenile crime have also declined over the same period, or does this suggest other over-arching influences that are impacting on a range of youth risk-taking behaviours?

As far as we are aware, this is the first study to explore trends in exposure to known risk factors with the purpose of better understanding the drivers of adolescent smoking decline. Strengths of the study include the large sample size, and demographic similarity between the

sample and the Year 10 population, suggesting response bias was not a substantial issue. Systematic under- and over-representation were found to be relatively consistent over time and therefore unlikely to affect trend analysis, which was the focus of our study. The methods for the ASH survey were broadly consistent between years, with minor changes (e.g. a change in fieldwork timing from 2011) unlikely to contribute significantly to the trends observed. Since there is strong similarity between NZ and other high income Western countries at a late stage in the tobacco epidemic in terms of trends in adolescent smoking and known risk factors, it is likely that our conclusions may be generalisable to similar countries, but this remains to be confirmed through further research.

Given the complex array of factors at various levels that are known to influence smoking uptake, one of the limitations of our study was the limited number of risk factors for which consistent data was available. Having said that, we found that less than 1% of students unexposed to any of the included risk factors were regular smokers in 2015. So while we have included only a small number of risk factors, collectively they have strong predictive power. The study was based on self-report data, with its inherent limitations; however recent biomarker testing of a sub-sample of ASH Year 10 participants indicated that the survey provides an accurate population estimate of smoking prevalence.[50] We used school decile as a proxy for socioeconomic status (SES), since more direct measures were unavailable. Because school communities are heterogeneous, it is an imperfect measure at the individual level, and residual confounding by socioeconomic status is possible in our adjusted associations. Finally, it is not possible to draw causal inferences based on cross-sectional data, and, much remains unknown about the mechanisms by which the risk factors examined might influence adolescent behaviour.

Conclusions

In summary, our findings suggest that declining exposure to daily smoking in the home and older sibling smoking have contributed to the decline in adolescent smoking in New Zealand since 2002, but reduced exposure to these risk factors only partially explains the pattern observed. Changes in parental smoking prevalence appear to have had little or no effect. We hypothesise that adolescent smoking decline may have been (to some degree) self-perpetuating via a virtuous cycle of peer influence, however further research is needed to confirm this. It is likely that factors other than those in our model are at play, with changes in the social meaning of smoking, the policy context and broader socio-cultural changes all potential contributors. Further research is needed to identify other contributing factors and determine their relative importance.

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Contributorship statement

All authors contributed to the conception and design of the study. The analysis was executed by DS and JB, with oversight by RE. JB drafted the manuscript with input from all authors.

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Competing interests

None declared.

Data sharing statement

The data is owned by ASH New Zealand, and cannot be shared by the authors.

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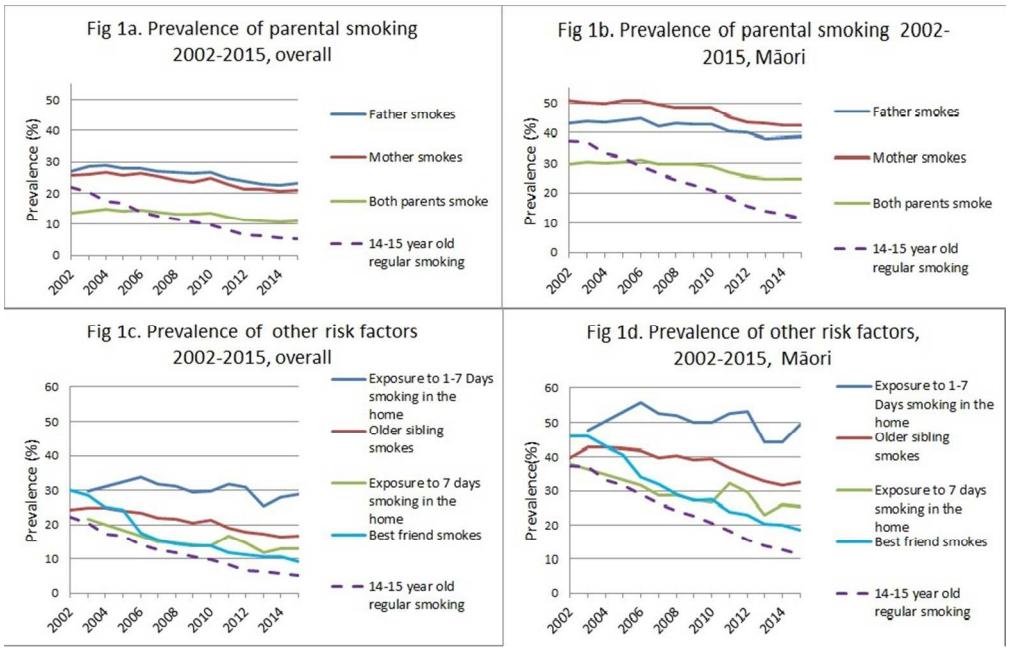
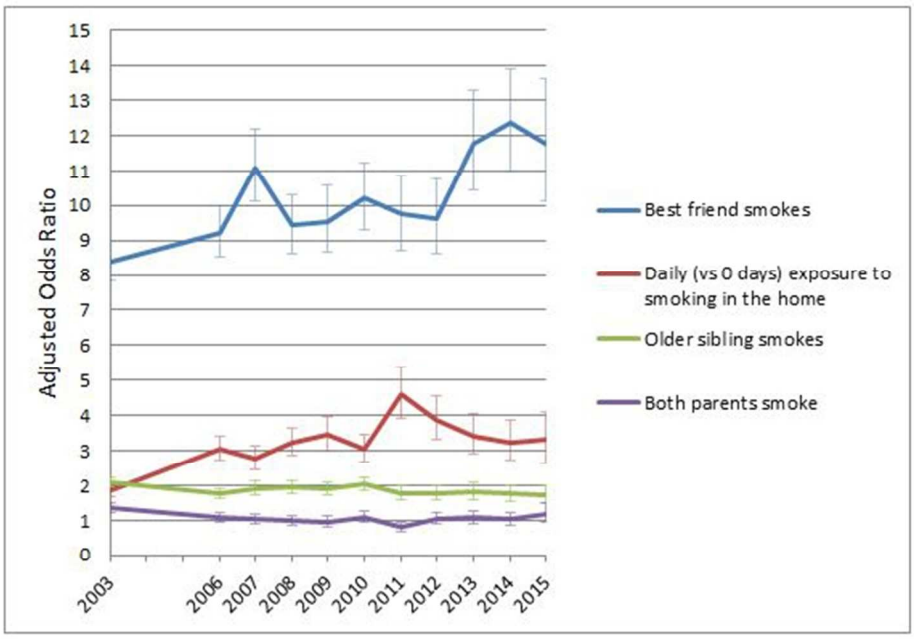


Prevalence of regular smoking and risk factors, 2002-2015, overall and Māori.

265x170mm (96 x 96 DPI)



Adjusted odds ratios for key risk factors, 2003-2015.

165x110mm (96 x 96 DPI)

Supplementary table: Sample characteristics and comparison with Year 10 population (% absolute difference)

			Age		Sex		School Decile			Ethnicity			
		Total	14 years	15 years	Male	Female	Low	Medium	High	NZEO	Maori	Pacific	Asian
2002	**N**	28088	13161	14927	13911	14177	4505	13007	10576	19395	4670	1833	2190
	%		47%	53%	50%	50%	16%	46%	38%	69%	17%	7%	8%
	dif from pop				*-1%*	*1%*	*-4%*	*2%*	*4%*				
2003	**N**	31377	14585	16792	15492	15885	5272	14274	11831	20716	5425	2174	3062
	%		46%	54%	49%	51%	17%	45%	38%	66%	17%	7%	10%
	dif from pop				*-2%*	*2%*	*-3%*	*3%*	*5%*				
2004	**N**	30807	18668	12139	14996	15811	5889	13373	11545	19888	5694	2244	2981
	%		61%	39%	49%	51%	19%	43%	37%	65%	18%	7%	10%
	dif from pop				*-2%*	*2%*	*-1%*	*1%*	*4%*				
2005	**N**	31833	15901	15932	15211	16622	4719	15374	11740	21176	5435	2172	3050
	%		50%	50%	48%	52%	15%	48%	37%	67%	17%	7%	10%
	dif from pop				*-3%*	*3%*	*-3%*	*2%*	*5%*				
2006	**N**	31690	19996	11694	15642	16048	4416	15935	11339	20161	6237	2252	3040
	%		63%	37%	49%	51%	14%	50%	36%	64%	20%	7%	10%
	dif from pop				*-2%*	*2%*	*-4%*	*4%*	*4%*	*2%*	*-2%*	*-1%*	*1%*
2007	**N**	25109	16213	8896	11988	13121	3558	11062	10489	16187	4653	1804	2465
	%		65%	35%	48%	52%	14%	44%	42%	64%	19%	7%	10%
	dif from pop				*-4%*	*4%*	*-4%*	*-2%*	*10%*	*5%*	*-3%*	*-2%*	*0%*
2008	**N**	29682	18395	11287	14462	15220	4732	12875	12075	17913	5552	2863	3354

	%		62%	38%	49%	51%	16%	43%	41%	60%	19%	10%	11%
	dif from pop				*-3%*	*3%*	*-2%*	*0%*	*3%*	*1%*	*-3%*	*1%*	*1%*
2009	**N**	24755	15977	8778	11607	13148	3235	11029	10491	15410	4304	2211	2830
	%		65%	35%	47%	53%	13%	45%	42%	62%	17%	9%	11%
	dif from pop				*-4%*	*4%*	*-5%*	*1%*	*4%*	*4%*	*-5%*	*0%*	*2%*
2010	**N**	31696	19725	11971	16636	15060	5111	14049	12536	18806	6315	3041	3534
	%		62%	38%	52%	48%	16%	44%	40%	59%	20%	10%	11%
	dif from pop				*1%*	*-1%*	*-2%*	*1%*	*2%*	*2%*	*-3%*	*0%*	*1%*
2011	**N**	26028	21390	4638	12462	13566	3720	10843	11465	15669	4995	2453	2911
	%		82%	18%	48%	52%	14%	42%	44%	60%	19%	9%	11%
	dif from pop				*-3%*	*3%*	*-4%*	*-1%*	*6%*	*3%*	*-4%*	*0%*	*0%*
2012	**N**	30396	25098	5298	14918	15478	4982	12461	12953	17878	5800	3048	3670
	%		83%	17%	49%	51%	16%	41%	43%	59%	19%	10%	12%
	dif from pop				*-2%*	*2%*	*-1%*	*-2%*	*4%*	*4%*	*-3%*	*1%*	*1%*
2013	**N**	27014	22126	4888	13546	13468	4049	10875	12090	16146	5111	2628	3129
	%		82%	18%	50%	50%	15%	40%	45%	60%	19%	10%	12%
	dif from pop				*-1%*	*1%*	*-3%*	*-2%*	*5%*	*3%*	*-4%*	*0%*	*1%*
2014	**N**	29303	24206	5097	14164	15139	4359	12836	12108	16847	5779	2952	3725
	%		83%	17%	48%	52%	15%	44%	41%	57%	20%	10%	13%
	dif from pop				*-3%*	*3%*	*-3%*	*2%*	*2%*	*2%*	*-4%*	*1%*	*1%*
2015	**N**	20443	16630	3813	10155	10288	3540	10024	6879	12123	4215	1884	2221
	%		81%	19%	50%	50%	17%	49%	34%	59%	21%	9%	11%
	dif from				*-2%*	*2%*	*-2%*	*5%*	*-2%*	*4%*	*-3%*	*0%*	*-1%*

Notes: Population data is based on Ministry of Education (MoE) records. Ethnicity data is not available from MoE prior to 2006, so ethnic comparison of population and sample is not possible 2002-2005.

Comparison between sample and population on age of students (14 or 15 years) is not shown since MoE data is measured at the midpoint in the year, whereas fieldwork dates differ from year to year, and as a result meaningful comparison is not possible.

STROBE Statement—Checklist of items that should be included in reports of ***cross-sectional studies***

	Item No	Recommendation	Page no.
Title and abstract	1	(*a*) Indicate the study's design with a commonly used term in the title or the abstract	1
		(*b*) Provide in the abstract an informative and balanced summary of what was done and what was found	2
Introduction			
Background/rationale	2	Explain the scientific background and rationale for the investigation being reported	3-4
Objectives	3	State specific objectives, including any prespecified hypotheses	6
Methods			
Study design	4	Present key elements of study design early in the paper	6-7
Setting	5	Describe the setting, locations, and relevant dates, including periods of recruitment, exposure, follow-up, and data collection	6
Participants	6	(*a*) Give the eligibility criteria, and the sources and methods of selection of participants	6
Variables	7	Clearly define all outcomes, exposures, predictors, potential confounders, and effect modifiers. Give diagnostic criteria, if applicable	8
Data sources/ measurement	8*	For each variable of interest, give sources of data and details of methods of assessment (measurement). Describe comparability of assessment methods if there is more than one group	*8*
Bias	9	Describe any efforts to address potential sources of bias	NA
Study size	10	Explain how the study size was arrived at	6
Quantitative variables	11	Explain how quantitative variables were handled in the analyses. If applicable, describe which groupings were chosen and why	9
Statistical methods	12	(*a*) Describe all statistical methods, including those used to control for confounding	9
		(*b*) Describe any methods used to examine subgroups and interactions	9
		(*c*) Explain how missing data were addressed	9
		(*d*) If applicable, describe analytical methods taking account of sampling strategy	9
		(*e*) Describe any sensitivity analyses	NA
Results			
Participants	13*	(a) Report numbers of individuals at each stage of study—eg numbers potentially eligible, examined for eligibility, confirmed eligible, included in the study, completing follow-up, and analysed	7
		(b) Give reasons for non-participation at each stage	NA
		(c) Consider use of a flow diagram	NA
Descriptive data	14*	(a) Give characteristics of study participants (eg demographic, clinical, social) and information on exposures and potential confounders	7 +Supl
		(b) Indicate number of participants with missing data for each variable of interest	NA
Outcome data	15*	Report numbers of outcome events or summary measures	9
Main results	16	(*a*) Give unadjusted estimates and, if applicable, confounder-adjusted estimates and their precision (eg, 95% confidence interval). Make clear	10-12

		which confounders were adjusted for and why they were included	
		(*b*) Report category boundaries when continuous variables were categorized	9
		(*c*) If relevant, consider translating estimates of relative risk into absolute risk for a meaningful time period	NA
Other analyses	17	Report other analyses done—eg analyses of subgroups and interactions, and sensitivity analyses	9-13
Discussion			
Key results	18	Summarise key results with reference to study objectives	13-14
Limitations	19	Discuss limitations of the study, taking into account sources of potential bias or imprecision. Discuss both direction and magnitude of any potential bias	16
Interpretation	20	Give a cautious overall interpretation of results considering objectives, limitations, multiplicity of analyses, results from similar studies, and other relevant evidence	27
Generalisability	21	Discuss the generalisability (external validity) of the study results	16
Other information			
Funding	22	Give the source of funding and the role of the funders for the present study and, if applicable, for the original study on which the present article is based	18

*Give information separately for exposed and unexposed groups.

Note: An Explanation and Elaboration article discusses each checklist item and gives methodological background and published examples of transparent reporting. The STROBE checklist is best used in conjunction with this article (freely available on the Web sites of PLoS Medicine at http://www.plosmedicine.org/, Annals of Internal Medicine at http://www.annals.org/, and Epidemiology at http://www.epidem.com/). Information on the STROBE Initiative is available at www.strobe-statement.org.

BMJ Open

Why has adolescent smoking declined dramatically? An investigation of changing exposure to risk factors using analysis of repeat cross-sectional data from New Zealand 2002-2015.

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Why has adolescent smoking declined dramatically? An investigation of changing exposure to risk factors using analysis of repeat cross-sectional data from New Zealand 2002-2015.

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ABSTRACT

Objectives: Adolescent smoking has declined in New Zealand and in many other countries since the late 1990s, yet the drivers of the decline are not well understood. We set out to determine i) whether changes in exposure to known predictors account for the decline, ii) whether relationships between known predictors and smoking outcomes have changed over time.

Design: Analysis of repeat cross-sectional data from a nationally representative survey.

Setting: New Zealand

Participants: Secondary school students aged 14-15. N=20,443 - 31,833 per year.

Outcome measure: Regular (at least monthly) smoking

Methods: We analysed smoking prevalence and exposure to risk factors (parental smoking, best friend smoking, older sibling smoking, and past week exposure to smoking in the home) based annual survey data 2002-2015. For each risk factor we calculated annual average change in exposure, and strength of association with adolescent smoking, expressed as odds ratios, (OR) for each year.

Results: There were declines in exposure to best friend smoking (absolute annual average decrease 1.5%), older sibling smoking (0.7%), daily exposure to smoking in the home (0.6%), maternal (0.5%) and paternal (0.5%) smoking. Adjusted ORs for exposure to smoking in the home and best friend smoking increased markedly over the study period while ORs for other risk factors remain unchanged.

Conclusions: Decreasing exposure to daily smoking in the home and older sibling smoking have likely contributed to declining adolescent smoking, but do not fully account for observed trends. Smoking status of best friend remains the strongest risk factor, with exposure falling dramatically, in line with population declines in adolescent smoking since 2002. Further research is need to determine whether a 'virtuous cycle' (with declines in adolescent smoking begetting further declines) has contributed to observed trends, and to assess the contribution of factors outside our model.

ARTICLE SUMMARY

Strengths and limitations of this study

- The survey has a large sample size (N=20,443 - 31,833 per year), allowing accurate population estimates of smoking prevalence and exposure to risk factors (and estimates of the strength of relationships between risk factors and smoking outcomes) based on individual-level data.
- Due to data limitations, the study only includes a small number of risk factors, however the risk factors included have consistently been found to be among the strongest and most important predictors of adolescent smoking.
- Our study design (using repeat cross sectional data) does not enable causal inferences to be drawn, however our study builds on existing knowledge about the predictors of adolescent smoking initiation and, in the absence of certainty about causality, accounts for change over time in statistical terms.

INTRODUCTION

Smoking is a leading cause of preventable illness and premature death[1] and a key driver of health disparities between ethnic and socioeconomic groups.[2 3] Long-term tobacco use

typically begins with experimental smoking in adolescence,[4] and, internationally, considerable research and policy attention has focused on understanding and preventing smoking uptake in this age group.

The dramatic decline in adolescent smoking observed since the late 1990s in many high income countries is good news from a public health perspective. In New Zealand (NZ), for example, regular smoking (defined as at least monthly) among 14-15 year olds declined from a peak of 29% in 1999 to 6% in 2014, with decreases across all main ethnic and socioeconomic groups, and a convergence between boys and girls over the period.[5] Over the same period, the proportion who had never smoked (i.e. not even a few puffs) rose from 32% to 78%.[5] However, as in other countries,[6] ethnic and socioeconomic disparities remain pronounced, for example Māori (indigenous) smoking prevalence in this age group was 13.2% in 2014 compared to 4.2% among non-Māori.[7] In the context of such marked disparities, understanding the risk factor profile of priority groups is particularly important, to inform policies and programmes aimed at further reducing smoking uptake.

Other countries including the USA, England and Australia have also experienced a decline in teen smoking from the late 1990s, following a sharp rise in the early 1990s.[8] It is important to understand the causes of this decline in order to help ensure it is sustained, and to enable replication in other countries. Yet little research has focused on explaining this phenomenon. Public health interventions such as increases in tobacco tax or smokefree environment legislation may have played a role,[9-11] but they do not fully explain the observed trends, since declines in adolescent smoking have occurred in countries with widely differing regulatory contexts. This, and the fact that other adolescent risk behaviours (e.g. alcohol use, teen

pregnancy) have also declined over a similar time period,[8] suggests broader socio-cultural changes rather than specific tobacco control policies may contribute to this international trend.

Such shifts could be generated by new technologies, for example. When the use of cell phones rose and smoking fell among adolescents in the late 1990s, a causal association was hypothesised.[12] More recently, attention has turned to other new technologies – smartphones and social media - and their potential role in driving generational change in attitudes and behaviour.[13] There is face validity to the idea that these new technologies may have changed the way young people socialise or project their identity, displacing the role of smoking or providing less opportunity for it. However, this hypothesis is challenged by a consistent body of evidence showing a *positive* association between smoking and cell phone/ internet/social media use at the individual level.[14-23]

Other major changes since the mid-90s that could potentially impact on youth behaviour include changes in parenting;[24-26] changes in the school environment and ethos; [27-29] and broad economic and labour market conditions resulting in young people leaving school and achieving independence later than previous cohorts.[30 31] But before exploring these novel explanations for smoking decline, it is important to determine the extent to which the observed trends can be explained by changing exposure to known predictors of smoking initiation.

Proximal risk factors for adolescent smoking have been studied extensively. For example parental, sibling and peer smoking have consistently been identified as key risk factors,[4 32] with the Surgeon General's 2012 review concluding that the evidence is suggestive of a causal role for peer influences, and a potential causal role for parental smoking.[4] The review found that smoking by older siblings influences smoking in adolescents more consistently than does smoking by parents.[4] Exposure to smoking in the home, although a less studied factor, has also been shown to be strongly related to the likelihood of smoking in adolescents independent of

parental smoking status.[33-37] Studies suggest second hand smoke exposure may biologically predispose children to nicotine dependence [38-42] in addition to providing pro-smoking socialization.[34]

Despite extensive risk factor research, few studies have explored how exposure to risk factors has changed over time, whether the relationships between established risk factors and outcomes have changed over time, or how such changes may be contributing to changes in adolescent smoking at the population level. Assuming that an observed individual-level relationship between a risk factor and adolescent smoking is causal, then declining exposure to that risk factor over time at the population level (while the strength of association is maintained or increased at the individual level) must contribute to a population decline in adolescent smoking. Even in the absence of certainty about causality, exploration of changes in level of exposure and strength of association over time allows us to 'explain' or account for changes in outcomes over time in statistical terms.

The current study explores trends in exposure of 14-15 year olds to known predictors of adolescent smoking (parental, sibling and peer smoking and exposure to smoking in the home) and the strength of the relationships between these risk factors and adolescent smoking from 2002 to 2015 in NZ. We sought to better understand the potential drivers of adolescent smoking decline to inform the continuation and replication of this positive trend. Our study also updates regular smoking trends (overall, and by ethnicity, gender and socioeconomic position) using 2015 data.

METHODS

We analysed repeat cross-sectional data from the ASH Year 10 Snapshot Survey series, an annual school-based survey of 14-15 year olds in NZ, which is administered by Action on Smoking and Health NZ (ASH) and is part of the New Zealand Youth Tobacco Monitor. The

questionnaire includes a set of 'core' questions that have remained consistent over time to enable trend monitoring, and additional questions which change from year to year. Years included in the current study were 2002-2015, since key variables of interest were unavailable prior to 2002. Furthermore, exposure to smoking in the home was not included in the questionnaire in 2002 or 2004-5, and therefore multivariable analysis includes only data from 2003 and 2006-15.

All public and private schools with Year 10 students were invited to participate in the ASH Year 10 Snapshot each year. School response rates range from 44-67% [43] (with a lower school response rate in 2015 due to limited resources for liaising with schools that year). Table 1 shows the sample size and student response rate (as a proportion of the total Year 10 population) by year.

Following previously published ASH analyses, analysis was restricted to respondents aged 14 or 15 at the time of the survey. For consistency between bivariate (unadjusted) and multivariable (adjusted) analyses, only respondents with complete data for all variables (smoking status, parental smoking, sibling smoking, best friend smoking, age, gender, ethnicity, school decile, and school ID, and for 2003 and 2006-15 exposure to smoking in the home) were included in the analyses. In addition, only schools with at least 20 respondents were included. Table 1 shows the number of valid survey responses received (i.e. those with complete data for age (14/15 years), sex, ethnicity and smoking status), and the number included in our study (after exclusions above), by year, and shows that after application of our exclusion criteria 96% of valid responses were included.

Table 1. Sample size and student response rate by year

Year	NZ Year 10	Valid	Responses	% Year 10

	population	survey responses	included in study	population included in study
2002	58,812	29,173	28,088	50%
2003	61,028	32,705	31,377	54%
2004	62,852	31,630	30,807	46%
2005	64,619	32,561	31,833	51%
2006	63,086	32,844	31,690	52%
2007	62,012	25,978	25,109	42%
2008	61,485	30,903	29,682	50%
2009	61,355	25,757	24,755	42%
2010	61,210	32,832	31,696	54%
2011	59,562	26,856	26,028	45%
2012	59,627	31,983	30,396	43%
2013	57,929	28,340	27,014	49%
2014	59,612	31,125	29,303	47%
2015	59,528	21,567	20,443	36%
Total	**852,717**	**414,254**	**398,221**	**47%**

The sample (N=20,443 to 31,833 per year) comprised approximately half the Year 10 population each year. Detailed comparison of the included sample and population, by year, is provided in a Supplementary Table S1. This comparison shows that the sample closely resembles the population albeit with modest but consistent under-representation of Māori and students from low decile schools. (School decile is a school-level measure of the socio-economic position of a school's student community, explained further below.[44])

Fieldwork was undertaken earlier in the year in 2011 and subsequently, meaning respondents were 2-3 months younger on average in 2011 and subsequent years, than in 2010 and prior years. Excluding the timing of fieldwork and changes to non-core questions, there has been consistency in survey instruments, administration and data management across included years.

In participating schools, the one-page survey is completed in class time under the supervision of teaching staff. Individual students may choose not to participate. To protect the confidentiality of students' responses, identifying information is not collected, and teachers

are requested not to check the completed surveys. Completed surveys are returned to ASH, which oversees data entry, cleaning and coding.

The survey was approved, as a component of the NZ Youth Tobacco Monitor, by the Ministry of Health Multiregional Health and Disability Ethics Committee in 2007. Further details on survey methodology are available elsewhere.[43]

Variables

The outcome variable, 'regular smoking' (Y/N) was defined as smoking at least monthly, based on the question 'How often do you smoke now?' The answer categories were: 'I have never smoked/I am not a smoker now'; 'At least once a day'; 'At least once a week'; 'At least once a month'; and 'Less often than once a month'.

Smoking status of mother, father, older sibling(s), and best friend were based on the question 'Which of the following people smoke?' with a dichotomous variable (current smoker, yes/no) created for each. Previous research suggests that maternal smoking is more strongly associated with adolescent smoking initiation than paternal smoking, therefore we examined exposure to maternal and paternal smoking separately. For the purposes of multivariable analysis, parental smoking was grouped into one variable, coded 0 = neither parent smokes, 1 = only mother smokes, 2 = only father smokes, 3 = both parents smoke.

Past week exposure to smoking in the home was based on the question 'During the past 7 days, on how many days have people smoked around you in your home?' Response categories were 0 days, 1-2 days, 3-4 days, 5-6 days and 7 days. We recoded the responses into three categories: 'Daily exposure' (7 days) 'less than daily exposure' (2-7 days) and 'no exposure' (0 days).

Demographic variables were age (14 or 15 years old), sex (male or female), ethnicity (prioritised Māori, Pacific, Asian, NZ European/other (NZEO); and dichotomised into Māori and non-Māori) and school decile. School decile is calculated by the Ministry of Education for purposes of funding allocation, and is a school-level measure of the socioeconomic position of a school's student community. Details of how school decile is calculated are available from the Ministry of Education.[44] We grouped school decile into low (deciles 1-3: most deprived), medium (4-7), and high (8-10: least deprived). Each school also had an identification number (school ID) which was assigned to all respondents from that school.

Analysis

We used SPSS (IBM Corp. Released 2016. IBM SPSS Statistics for Windows, Version 24.0. Armonk, NY: IBM Corp.) to tabulate prevalence of regular smoking and prevalence of exposure to risk factors (overall and by sex, ethnicity and school decile) for each year. We then quantified the mean annual absolute change in proportion of respondents exposed to each risk factor using weighted linear regression (to adjust for differing variance by year by giving more weight to more accurate estimates of prevalence) with year as the independent variable. The weights were $1/SD^2$ of the proportions.

Logistic regression (PROC Glimmix) using SAS/STAT software, (Version 9.4 of the SAS system for Windows. Copyright © 2002-2012, SAS Institute Inc., Cary, NC, USA.) was used to calculate bivariate odds ratios (OR) to determine the strength of the relationship between each risk factor and regular smoking for each year (Model 1, unadjusted). To adjust for potential clustering at the school level, school ID was entered as a random effect in the estimation of odds ratios. Multivariable logistic regression analysis (again with a random effect of school) was then conducted for each year using three models. Model 2 assessed the association between each risk factor and regular smoking adjusting for demographic variables

(sex, age, ethnicity, and school decile) only. Model 3 adjusted for demographic factors (as in Model 2) plus parental, sibling and best friend smoking, to determine the independent relationship of each with adolescent smoking. Model four adjusted for all of the variables in Model 3, plus past week exposure to smoking in the home. This approach allows us to present adjusted estimates for parental and sibling smoking including and excluding smoking in the home, a factor which is likely to be on the causal pathway between parental and sibling smoking and adolescent smoking.

Because Māori adolescents have high smoking rates and are therefore a key priority group for preventive interventions, we wanted to test whether the relationships between risk factors and outcomes identified in the main analyses were also applicable to Māori. We conducted supplementary analyses for Māori, using logistic regression methods described above for Model 1 (unadjusted) and Model 4 (fully adjusted) models, except multivariate models did not include ethnicity.

Patient and Public Involvement

Patients were not involved in the design or conduct of this study, and nor were members of the general public.

RESULTS

Prevalence of regular smoking

We found that the long-term decline in prevalence of regular smoking among 14-15 year olds continued in 2015, to 5.4% (Figure 1). Based on weighted linear regression, the overall regular smoking rate reduced by an average of 1.2 % per year (in absolute terms) from 2002-2015.

Supplementary Figures S1, S2 and S3 show trends in prevalence of regular smoking stratified by ethnicity, school decile and gender, and indicate that smoking has declined in all demographic groups. Furthermore ethnic, socioeconomic position (SEP), and gender disparities have narrowed over time in absolute terms. However, as shown in Figure 2, smoking remains strongly patterned by both ethnicity and school decile, and is increasingly concentrated in Maori, Pacific and low decile groups (in relative terms) as adolescent smoking in other groups becomes increasingly rare.

Changes in exposure to risk factors over time

Trends in exposure to risk factors are shown in Figure 1. Parental smoking (Fig 1a) declined only modestly over the study period with maternal and paternal smoking both declining by an average rate of 0.5% per annum. Smoking among older siblings declined slightly more (Fig 1b), at an average rate of 0.7% per annum, and 'best friend smokes' had the highest rate of decline at 1.5% per annum. Prevalence of daily exposure to smoking in the home fell from 22% to 13% overall (an average decrease of 0.6% per annum), however less than daily exposure increased over the study period.

Supplementary Figures S4 to S10 show exposure to risk factors over time by ethnicity and school decile. They show that trends in exposure to risk factors followed a similar pattern in all ethnic and SEP subgroups, but disparities in levels of exposure were marked in all years.

Changes in the relationship between risk factors and regular smoking over time

As shown in Table 2, bivariate analysis (Model 1, unadjusted) confirmed that smoking status of best friend, older sibling(s), and parents were all strongly associated with smoking in 14-15 year olds in 2003 and 2015 at the individual level, as was exposure to smoking in the home. After adjusting for age, sex, and school decile (Model 2) the associations were slightly

attenuated but remained strong. Model 3 added parental smoking, sibling smoking and best friend smoking to the model (but excluded exposure to smoking in the home), and after adjustment for these and the demographic factors, associations were weakened but remained statistically significant in both 2003 and 2012. The fully adjusted model (Model 4) included exposure to smoking in the home as well as all the other risk and demographic factors. In Model 4, strong and statistically significant associations with smoking status of best friend and older sibling(s) and exposure to smoking in the home persisted, but parental smoking had only a weak (2003) or non-significant (2015) association with the likelihood of regular smoking.

Table 2: Associations between risk factors and regular smoking among adolescents, 2003 and 2015expressed as odds ratios and 95% confidence intervals.

	2003 (N=31,377)				
	Prevalence of exposure (%)	Model 1: unadjusted odds ratio	Model 2: Odds ratio, adjusted for demographic factors	Model 3: Odds ratio adjusted for demographics, parental, sibling and best friend smoking	Model 4: Odds ratio fully adjusted for all demographics and risk factors
No exposure to smoking in the home (0 days)	70.2	1.0	1.0	-	1.0
Daily exposure to smoking in the home (7 days)	21.6	3.8 (3.6, 4.0)	3.4 (3.2, 3.6)	-	1.8 (1.7, 2.0)
Less than daily exposure to smoking in the home (1-6 days)	8.1	2.2 (2.0, 2.4)	2.1 (1.9. 2.3)	-	1.4 (1.3, 1.6)
Neither parent smokes	59.6	1.0	1.0	1.0	1.0
Only mother smokes	11.7	2.7 (2.4, 2.9)	2.3 (2.1, 2.5)	1.8 (1.6, 2.0)	1.3 (1.2, 1.5)
Only father smokes	14.4	1.9 (1.7, 2.0)	1.8 (1.7, 2.0)	1.5 (1.4, 1.7)	1.3 (1.2, 1.4)
Both parents smoke	14.3	4.2 (3.9, 4.6)	3.6 (3.3, 3.9)	2.0 (1.8, 2.1)	1.4 (1.2,1.5)
Older sibling smokes	24.9	3.6 (3.4, 3.8)	3.1 (3.0, 3.3)	2.2 (2.0, 2.3)	2.1 (1.9, 2.2)
Best friend smokes	28.5	11.4 (10.7, 12.1)	10.2 (9.5, 10.8)	8.6 (8.1, 9.2)	8.4 (7.9, 9.0)
	2015 (N=20,443)				
	Prevalence of exposure (%)	Model 1: unadjusted odds ratio	Model 2: Odds ratio, adjusted for demographic factors	Model 3: Odds ratio adjusted for demographics, parental, sibling and best friend smoking	Model 4: Odds ratio fully adjusted for all demographics and risk factors
No exposure to smoking	71.2	1.0	1.0	-	1.0

in the home (0 days)					
Daily exposure to smoking in the home	12.9	9.0 (7.7, 10.5)	7.3 (6.2, 8.5)	-	3.3 (2.7, 4.1)
Less than daily exposure to smoking in the home	28.8	6.3 (5.5, 7.2)	3.7 (3.1, 4.4)	-	2.6 (2.1, 3.1)
Neither parent smokes	67.1	1.0	1.0	1.0	1.0
Only mother smokes	9.8	3.9 (3.2, 4.7)	3.2 (2.6, 3.8)	2.1 (1.7, 2.6)	1.2 (1.0, 1.5)*
Only father smokes	11.9	2.5 (2.1-3.1)	2.3 (1.9, 2.8)	1.8 (1.4, 2.2)	1.2 (0.9, 1.5)*
Both parents smoke	11.2	6.2 (5.3, 7.3)	4.8 (4.0, 5.6)	2.3 (1.9, 2.8)	1.2 (1.0, 1.5)*
Older sibling smokes	16.6	4.7 (4.1, 5.3)	3.8 (3.3, 4.3)	2.0 (1.7, 2.3)	1.7 (1.5, 2.0)
Best friend smokes	9.3	19.6 (17.0, 22.5)	17.1 (14.9, 19.7)	13.0 (11.2, 15.0)	11.8 (10.1, 13.6)

*All Odds Ratios are statistically significant (p<.001) except those marked with * which did not reach statistical significance at the 95% level.*

Confidence intervals for prevalence are uniformly narrow, and have been omitted to aid the legibility of the table.

We found that some risk factors had an increasing strength of association with smoking over time. As shown in Table 2, the fully-adjusted odds ratio (Model 4) for daily exposure to smoking in the home (OR 1.8 (1.7, 2.0) in 2003, OR 3.3 (2.7, 4.1) in 2015) and best friend smoking (8.4 (7.9, 9.0) in 2003; 11.8 (10.1, 13.6) in 2015) increased markedly over the study period. The statistical significance ($p<0.0001$) of these changes in OR was confirmed by modelling the interaction effect between risk factors and year (Supplementary Table S2). In contrast, there was no significant change in fully-adjusted ORs for parental and sibling smoking between 2003 and 2015. A similar pattern was seen in both the sample overall, and in Māori (Supplementary Table S3). Fully adjusted ORs for key risk factors for all available years are presented in Supplementary Figure S11, showing considerable year to year variation (and overall upward trend) in ORs for best friend smoking and exposure to smoking in the home, while ORs for parental and sibling smoking remained relatively constant over time.

DISCUSSION

We found that the longstanding decline in 14-15 year old smoking in NZ continued in 2015. Exposure to best friend smoking declined strongly during the 2002-2015 period and exposure to other risk factors decreased more modestly. There was no change in past week exposure to smoking in the home overall (i.e.1-7 days), but daily exposure fell significantly, whilst less than daily exposure increased. With the exception of parental smoking, all the risk factors examined were significant independent risk factors for adolescent smoking in the fully adjusted models in all years. The strength of association (adjusted OR) between adolescent smoking and certain risk factors - exposure to smoking in the home and best friend smoking - increased markedly over the study period, while other relationships remained unchanged.

The aim of the study was to determine whether changing exposure to known risk factors explained (in statistical terms) the dramatic decline in adolescent smoking seen recently in NZ. Based on our results, we can conclude that declines in daily exposure to smoking in the home, older sibling smoking and best friend smoking were all likely contributors, as each was independently associated with regular smoking (with previous research suggestive of a causal relationship) and prevalence of exposure declined over the study period.

The marked differences between unadjusted and adjusted ORs suggest that the risk factors in our study were strongly related to one another. This is consistent with research that shows that smoking clusters within occupations and neighbourhoods and is becoming increasingly concentrated in low-income communities.[45 46] As a result, adolescents tend experience life within smoking or non-smoking social circles, and therefore the smoking status of family members and friends are strongly correlated.

Our findings with regards to parental smoking are complex to interpret. Parental smoking was strongly associated with adolescent smoking in the unadjusted model and (somewhat less so) in adjusted models that excluded smoking in the home. However, in the fully adjusted model, the association became weak or non-significant. This, together with the strong association between parental smoking and SHS exposure in the home (data not shown), suggests that exposure to smoking in the home mediates or confounds (or both) the relationship between parental smoking and adolescent smoking. Our data and study design do not allow us to determine whether confounding or mediation is the dominant phenomenon. However, our findings suggest that declines in parental smoking have probably influenced the decline in adolescent smoking not directly (since parental smoking was not an independent risk factor in most years) but via declining exposure to other risk factors, particularly daily exposure to smoking in the home. This interpretation is consistent with previous research,[34-37] suggesting parental smoking may influence adolescent smoking primarily via exposure to smoking in the

home. Our findings add to a growing body of research suggesting that exposure to second hand smoke may predispose young people to tobacco use and nicotine dependency via biological, psychological and/or social mechanisms.[47] Greater research and policy attention to smokefree homes (and cars) as part of a comprehensive smoking prevention approach is therefore warranted.

The smoking status of respondents' best friend was by far the strongest risk factor for regular smoking in 14-15 year olds, with the association strengthening over the study period. It is important to note that this association may be partly due to smokers seeking out other smokers as friends, as well as via a probable causal influence.[4] However, at the population level, it would be a circular to suggest that declining best friend smoking was driving the decline in prevalence of adolescent smoking, since survey respondents and their best friends belong to the same cohort. The question remains: if decreases in best friend smoking are resulting in reduced risk of adolescent smoking at the individual level, what is driving the decline in best friend smoking? One possibility is that, since younger adolescents are strongly influenced by peers and adolescents slightly older then themselves (including siblings), a virtuous cycle may have developed whereby a decline in adolescent smoking at time 1 has led to a subsequent decline in adolescent smoking at time 2 and so on. Further research, perhaps drawing on communicable disease methodology, could test this hypothesis and explore how the 'social transmission' of smoking (and other health risk behaviours) influences population prevalence over time. Should this hypothesis prove to be correct, the trigger for the sudden change from rapidly rising to rapidly falling adolescent tobacco use from the late 1990s to the early 2000s still remains to be identified.

Whilst we found strong associations with established risk factors in our analyses, there are likely to be other factors influencing the decline in adolescent smoking that this study did not address. For example, it is possible that changes in the social meaning of smoking[48 49] and the

policy context[11 50] – factors that were not included in our analyses - may have played a role in triggering adolescent smoking decline. For example, policy responses to rising adolescent smoking in the 1990s may have influence teen smoking in NZ, as they appear to have done in Australia[10 11] and the US.[9] Smoking in NZ 14-15 year olds peaked in 1999, two years after the legal age of tobacco purchase was raised from 16 to 18 years of age, and a year after a 1998 tax increase which raised the price of a packet of 20 cigarettes by 13%.[51] The turning point in adolescent smoking also closely followed the 'Why start?' mass media campaign targeting youth which ran from 1996-1998. It is plausible that, collectively, these measures contributed to the denormalisation of smoking, which, rather than any specific intervention, may have been the trigger for adolescent smoking decline both in NZ and other jurisdictions. Mass media campaigns from 2000 focusing on second hand smoke and a 2004 ban on smoking in pubs and all other indoor workplaces likely contributed to the ongoing denomalisation of smoking (in particular indoor smoking),[52] and may underpin the observed decline in daily exposure to smoking in the home. As Simon Chapman has pointed out, denormalisation involves an 'interplay of continuous, uncontrolled, unmeasured, and sometimes unmeasurable variables intended to influence [tobacco] consumption', and cannot be reduced to the sum of its parts.[53]

However if tobacco denormalisation, along with the other factors discussed above, explains the decline in adolescent smoking, is it simply a coincidence that teen alcohol use, teen pregnancy and juvenile crime have also declined over the same period, or does this suggest other over-arching influences that are impacting on a range of youth risk-taking behaviours?

As far as we are aware, this is the first study to explore trends in exposure to known risk factors with the purpose of better understanding the drivers of adolescent smoking decline. Strengths of the study include the large sample size, and the demographic similarity between the sample and the Year 10 population, suggesting response bias was not a substantial issue.

Systematic under- and over-representation were found to be relatively consistent over time and therefore unlikely to affect trend analysis, which was the focus of our study. The methods for the ASH survey were broadly consistent between years, with minor changes (e.g. a change in fieldwork timing from 2011) unlikely to contribute significantly to the trends observed. Since there is strong similarity between NZ and other countries at a late stage in the tobacco epidemic in terms of trends in adolescent smoking and known risk factors, it is likely that our conclusions may be generalisable to similar countries, but this remains to be confirmed through further research.

Given the complex array of factors at various levels that are known to influence smoking uptake, one of the limitations of our study was the limited number of risk factors for which consistent data was available. Having said that, we found that less than 1% of students unexposed to any of the included risk factors were regular smokers in 2015. So while we have included only a small number of risk factors, collectively they have strong predictive power. The study was based on self-report questionnaire data, with its inherent limitations (e.g. potential for social desirability bias, and misinterpretation of questions resulting in misclassification); however recent biomarker testing of a sub-sample of ASH Year 10 participants indicated that the survey provides an accurate population estimate of smoking prevalence.[54] We used school decile as a proxy for socioeconomic position (SEP), since more direct measures were unavailable. Because school communities are heterogeneous, it is an imperfect measure at the individual level, and residual confounding by socioeconomic status is possible in our adjusted associations. Finally, it is not possible to draw causal inferences based on cross-sectional data, and, much remains unknown about the mechanisms by which the risk factors examined might influence adolescent behaviour.

Conclusions

In summary, our findings suggest that declining exposure to daily smoking in the home and older sibling smoking have contributed to the decline in adolescent smoking in New Zealand since 2002, but reduced exposure to these risk factors only partially explains the pattern observed. We hypothesise that adolescent smoking decline may have been (to some degree) self-perpetuating via a virtuous cycle of peer influence, however further research is needed to confirm this. It is likely that factors other than those in our model are at play, with changes in the social meaning of smoking, the policy context and broader socio-cultural changes all potential contributors. Further research is needed to identify other contributing factors and determine their relative importance.

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Contributorship statement

All authors contributed to the conception and design of the study. The analysis was executed by DS and JB, with oversight by RE. JB drafted the manuscript with input from all authors.

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Competing interests

None declared.

Data sharing statement

The data is owned by ASH New Zealand, and cannot be shared by the authors.

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Fig 1a. Trends in adolescent and parental smoking prevalence, 2002-2015

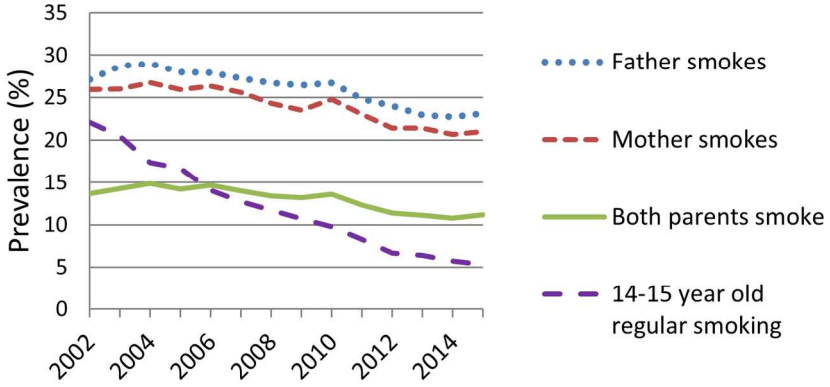


Fig 1b. Trends in exposure to other risk factors, 2002-2015

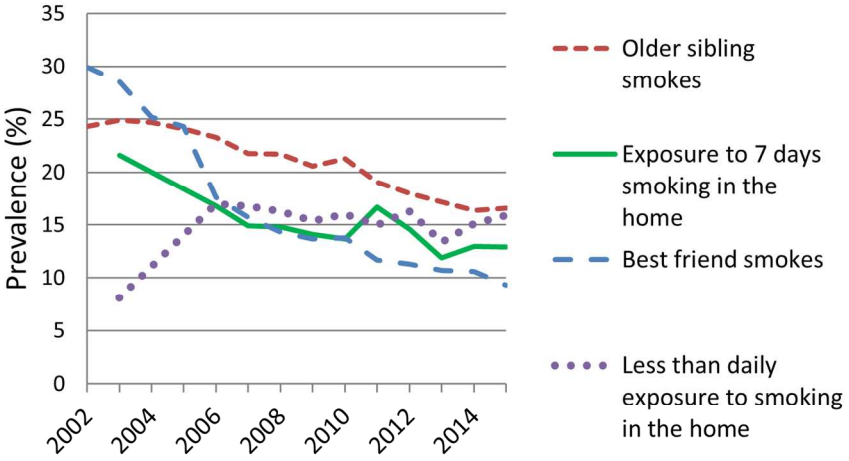


Fig 1: Prevalence of regular smoking and risk factors in 14-15 year olds, 2002 to 2015.

137x178mm (300 x 300 DPI)

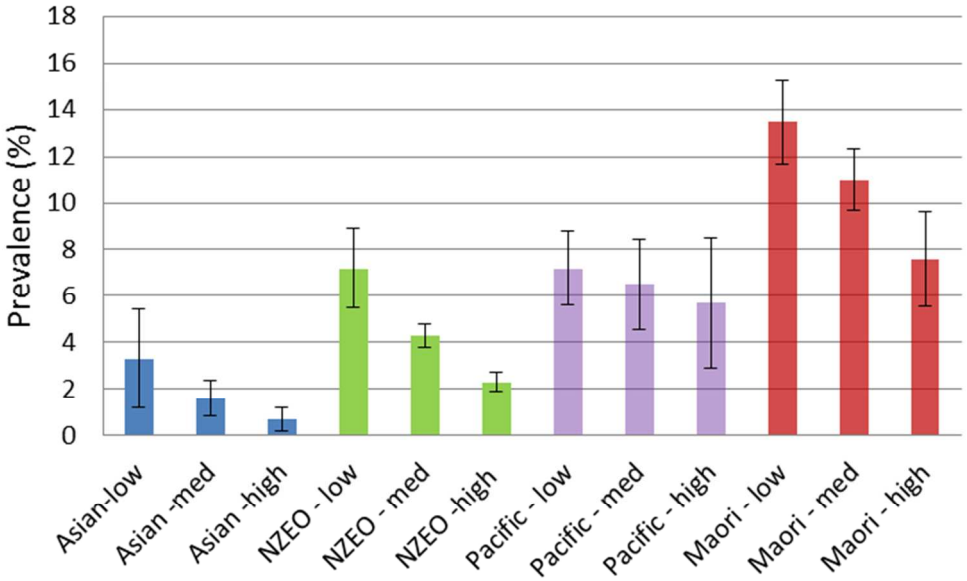


Fig 2: Regular smoking in 14-15 year olds, stratified by ethnicity and school decile, 2015.

242x153mm (300 x 300 DPI)

SUPPLMENTARY MATERIAL

Characteristics of the sample

Supplementary Table S1: Sample characteristics and comparison with Year 10 population (% absolute difference)

			Age		Sex		School Decile			Ethnicity			
		Total	14 years	15 years	Male	Female	Low	Medium	High	NZEO	Maori	Pacific	Asian
2002	**N**	28088	13161	14927	13911	14177	4505	13007	10576	19395	4670	1833	2190
	%		47%	53%	50%	50%	16%	46%	38%	69%	17%	7%	8%
	dif from pop				*-1%*	*1%*	*-4%*	*2%*	*4%*				
2003	**N**	31377	14585	16792	15492	15885	5272	14274	11831	20716	5425	2174	3062
	%		46%	54%	49%	51%	17%	45%	38%	66%	17%	7%	10%
	dif from pop				*-2%*	*2%*	*-3%*	*3%*	*5%*				
2004	**N**	30807	18668	12139	14996	15811	5889	13373	11545	19888	5694	2244	2981
	%		61%	39%	49%	51%	19%	43%	37%	65%	18%	7%	10%
	dif from pop				*-2%*	*2%*	*-1%*	*1%*	*4%*				
2005	**N**	31833	15901	15932	15211	16622	4719	15374	11740	21176	5435	2172	3050
	%		50%	50%	48%	52%	15%	48%	37%	67%	17%	7%	10%
	dif from pop				*-3%*	*3%*	*-3%*	*2%*	*5%*				
2006	**N**	31690	19996	11694	15642	16048	4416	15935	11339	20161	6237	2252	3040
	%		63%	37%	49%	51%	14%	50%	36%	64%	20%	7%	10%
	dif from				*-2%*	*2%*	*-4%*	*4%*	*4%*	*2%*	*-2%*	*-1%*	*1%*

	pop												
2007	**N**	25109	16213	8896	11988	13121	3558	11062	10489	16187	4653	1804	2465
	%		65%	35%	48%	52%	14%	44%	42%	64%	19%	7%	10%
	dif from pop				*-4%*	*4%*	*-4%*	*-2%*	*10%*	*5%*	*-3%*	*-2%*	*0%*
2008	**N**	29682	18395	11287	14462	15220	4732	12875	12075	17913	5552	2863	3354
	%		62%	38%	49%	51%	16%	43%	41%	60%	19%	10%	11%
	dif from pop				*-3%*	*3%*	*-2%*	*0%*	*3%*	*1%*	*-3%*	*1%*	*1%*
2009	**N**	24755	15977	8778	11607	13148	3235	11029	10491	15410	4304	2211	2830
	%		65%	35%	47%	53%	13%	45%	42%	62%	17%	9%	11%
	dif from pop				*-4%*	*4%*	*-5%*	*1%*	*4%*	*4%*	*-5%*	*0%*	*2%*
2010	**N**	31696	19725	11971	16636	15060	5111	14049	12536	18806	6315	3041	3534
	%		62%	38%	52%	48%	16%	44%	40%	59%	20%	10%	11%
	dif from pop				*1%*	*-1%*	*-2%*	*1%*	*2%*	*2%*	*-3%*	*0%*	*1%*
2011	**N**	26028	21390	4638	12462	13566	3720	10843	11465	15669	4995	2453	2911
	%		82%	18%	48%	52%	14%	42%	44%	60%	19%	9%	11%
	dif from pop				*-3%*	*3%*	*-4%*	*-1%*	*6%*	*3%*	*-4%*	*0%*	*0%*
2012	**N**	30396	25098	5298	14918	15478	4982	12461	12953	17878	5800	3048	3670
	%		83%	17%	49%	51%	16%	41%	43%	59%	19%	10%	12%
	dif from pop				*-2%*	*2%*	*-1%*	*-2%*	*4%*	*4%*	*-3%*	*1%*	*1%*
2013	**N**	27014	22126	4888	13546	13468	4049	10875	12090	16146	5111	2628	3129
	%		82%	18%	50%	50%	15%	40%	45%	60%	19%	10%	12%
	dif from pop				*-1%*	*1%*	*-3%*	*-2%*	*5%*	*3%*	*-4%*	*0%*	*1%*
2014	**N**	29303	24206	5097	14164	15139	4359	12836	12108	16847	5779	2952	3725

	%		83%	17%	48%	52%	15%	44%	41%	57%	20%	10%	13%
	dif from pop				*-3%*	*3%*	*-3%*	*2%*	*2%*	*2%*	*-4%*	*1%*	*1%*
2015	**N**	20443	16630	3813	10155	10288	3540	10024	6879	12123	4215	1884	2221
	%		81%	19%	50%	50%	17%	49%	34%	59%	21%	9%	11%
	dif from pop				*-2%*	*2%*	*-2%*	*5%*	*-2%*	*4%*	*-3%*	*0%*	*-1%*

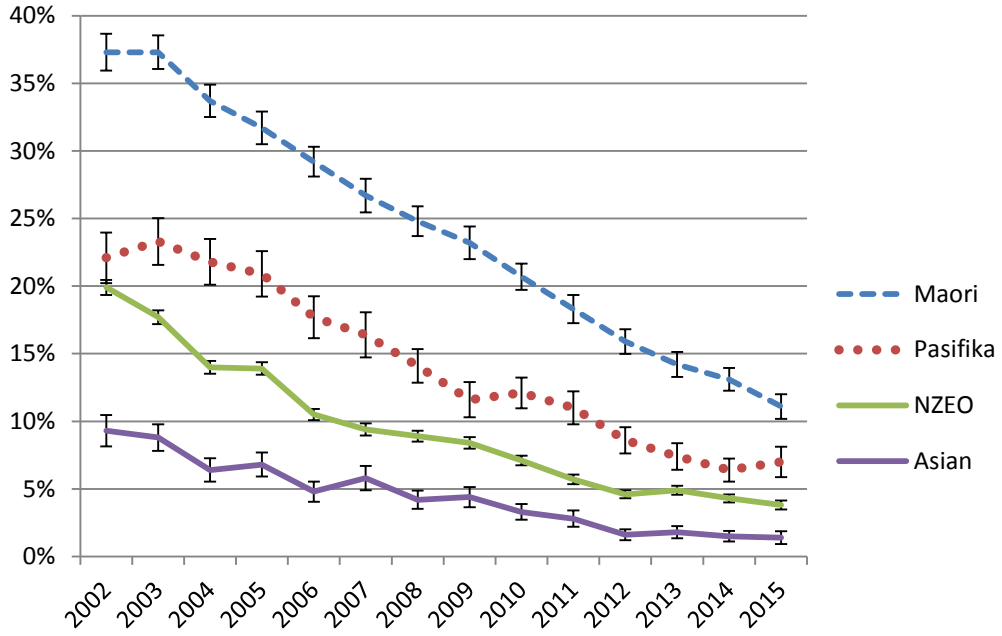
Notes: Population data is based on Ministry of Education (MoE) records which are available online at: https://www.educationcounts.govt.nz/statistics/schooling/student-numbers/6028.

Ethnicity data is not available from MoE prior to 2006, so ethnic comparison of population and sample is not possible 2002-2005.

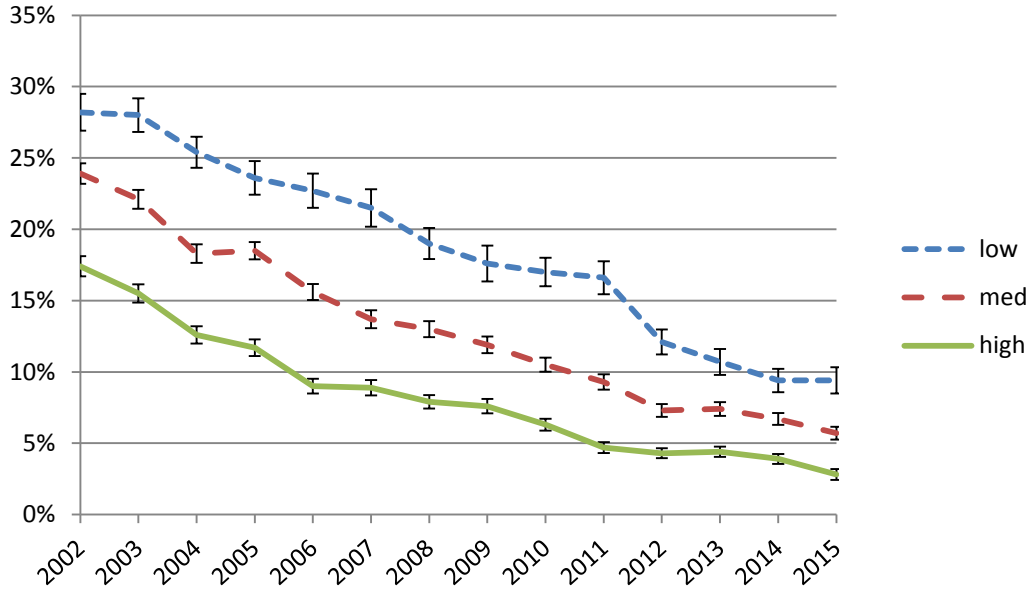
Comparison between sample and population on age of students (14 or 15 years) is not shown since MoE data is measured at the midpoint in the year, whereas survey fieldwork dates differ slightly from year to year, and as a result meaningful comparison is not possible.

Smoking prevalence by ethnicity, school decile and gender

Supplementary Figure S1: Prevalence of regular smoking in 14-15 year olds by prioritised ethnicity, 2002 to 2015



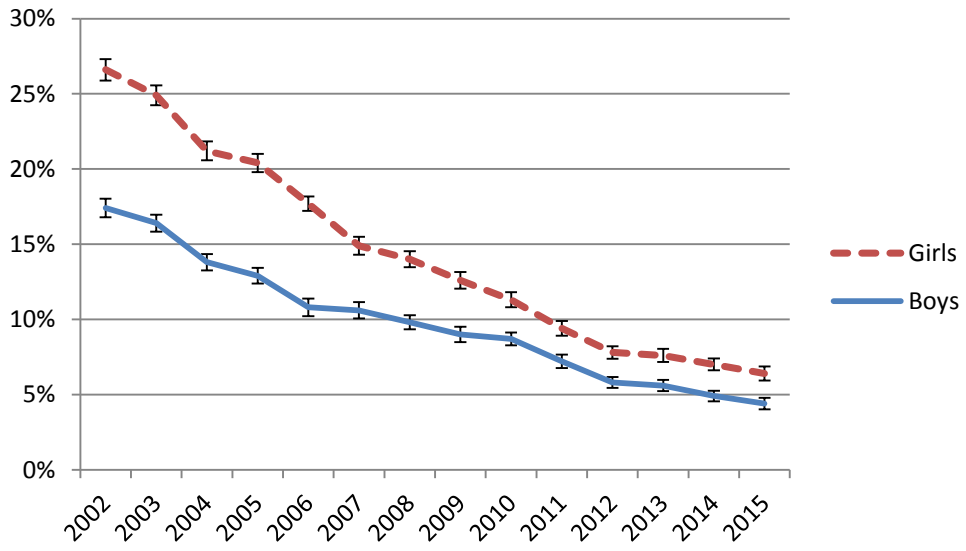
Supplementary Figure S2: Prevalence of regular smoking in 14-15 year olds by school decile, 2002 to 2015



Notes: Low decile = most deprived. High decile = least deprived.

School decile is calculated by the Ministry of Education for purposes of funding allocation, and is a school-level measure of the socioeconomic position of a school's student community. Details of how school decile is calculated are available on the Ministry of Education website: https://www.education.govt.nz/school/running-a-school/resourcing/operational-funding/school-decile-ratings/

Supplementary Figure S3: Prevalence of regular smoking in 14-15 year olds by gender, 2002 to 2015



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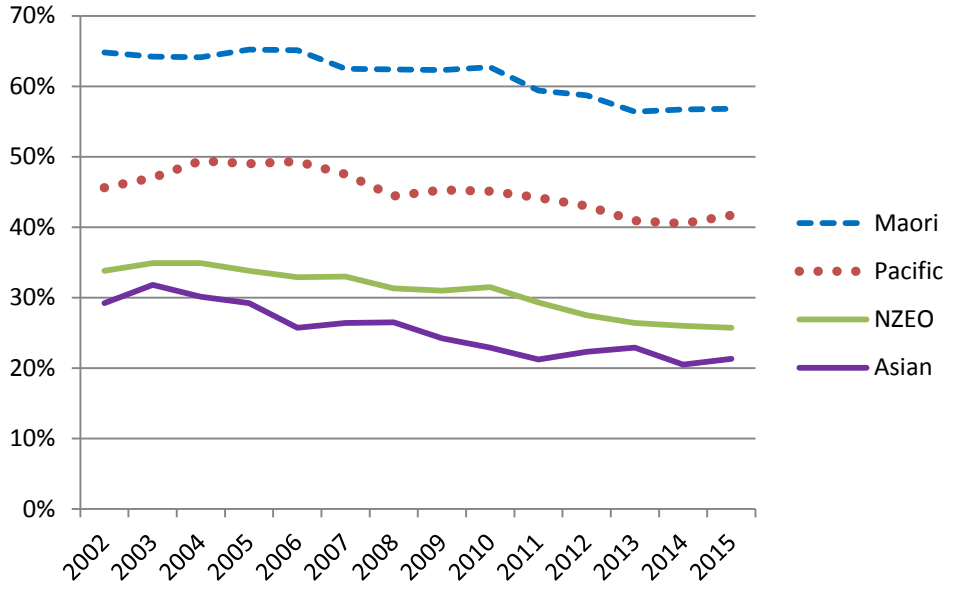
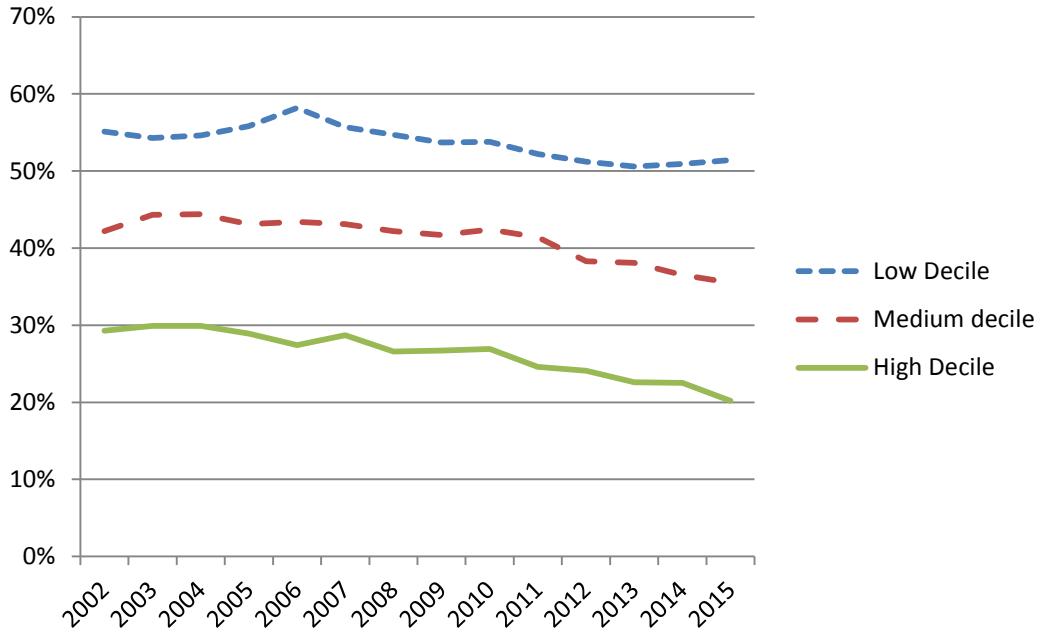
Changes in exposure to risk factors over time, by ethnicity and school decile

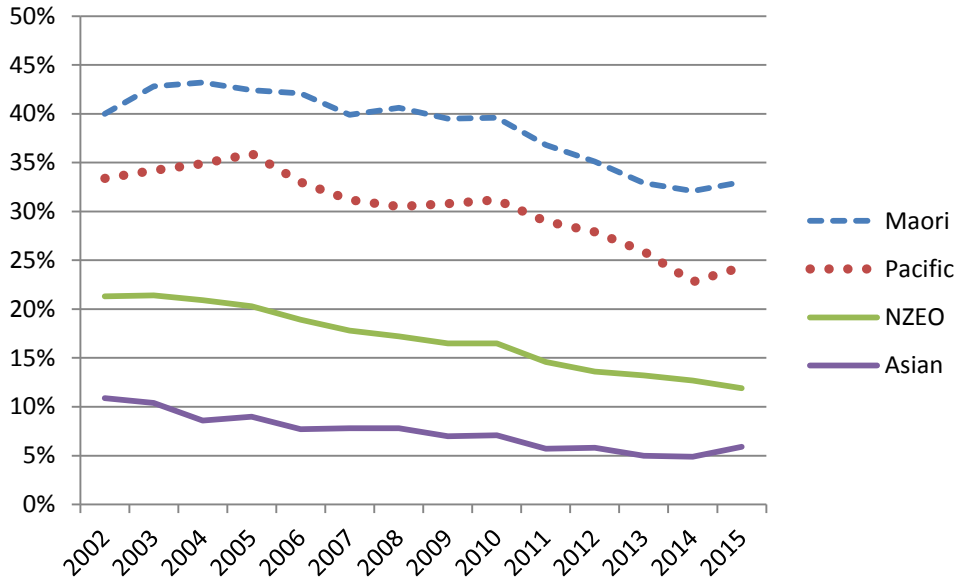
Supplementary Figure S4: Proportion reporting at least one parent smokes by ethnicity, 2002 to 2015



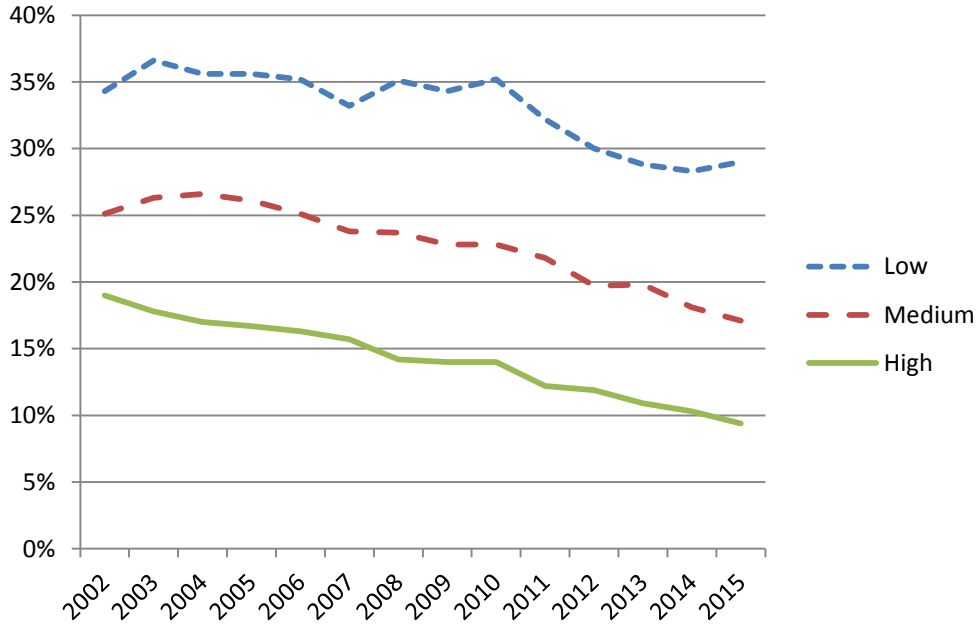
Supplementary Figure S5: Proportion reporting at least one parent smokes by school decile, 2002 to 2015



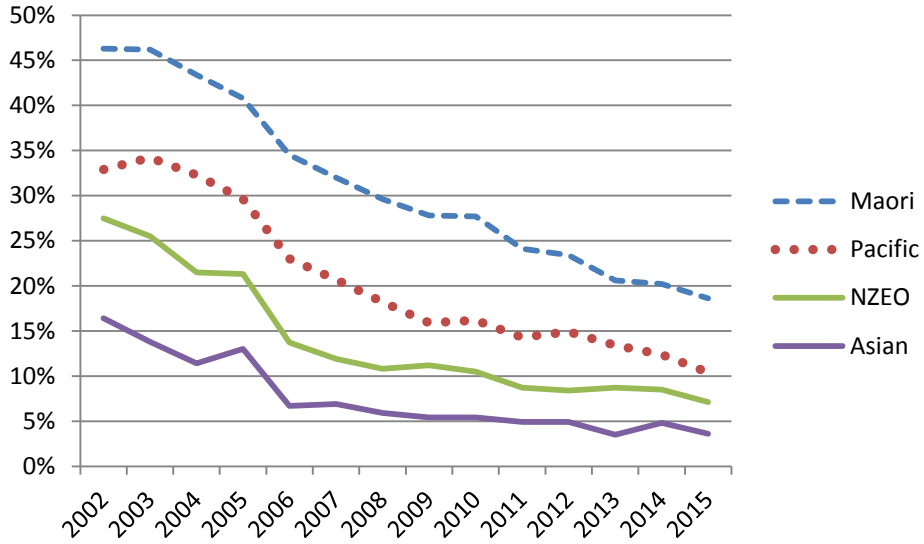
Supplementary Figure S6: Proportion reporting older sibling(s) smoke, by ethnicity, 2002 to 2015.



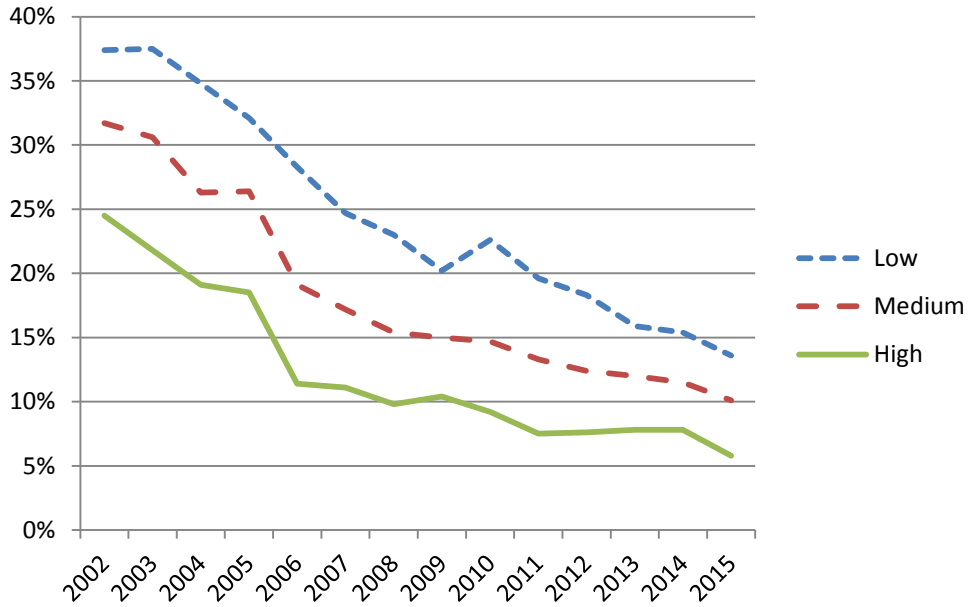
Supplementary Figure S7: Proportion reporting older sibling(s) smoke, by school decile, 2002 to 2015.



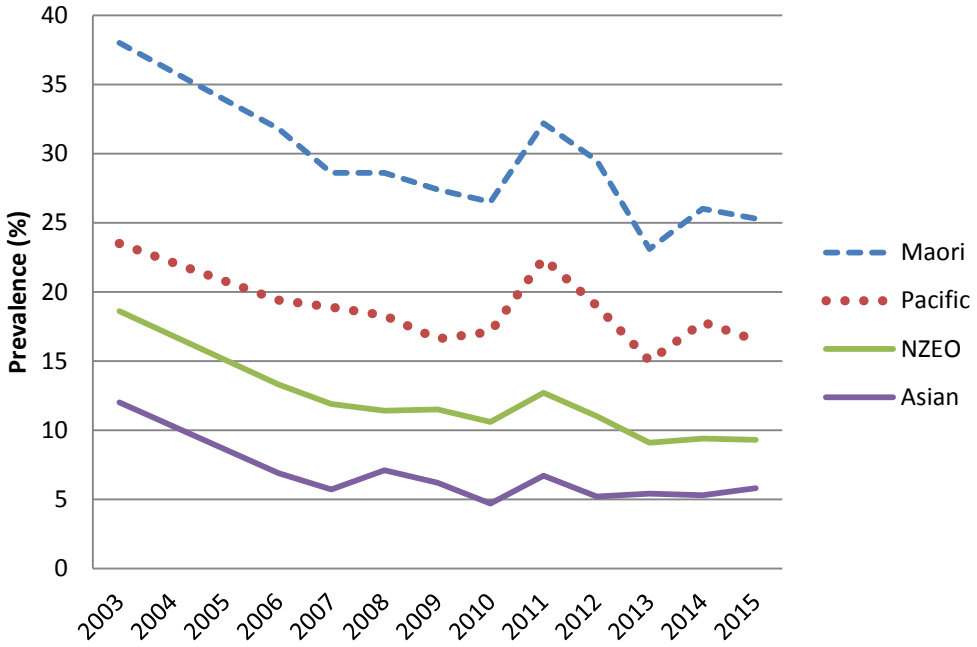
Supplementary Figure S8: Proportion reporting best friend smokes, by ethnicity, 2002 to 2015.



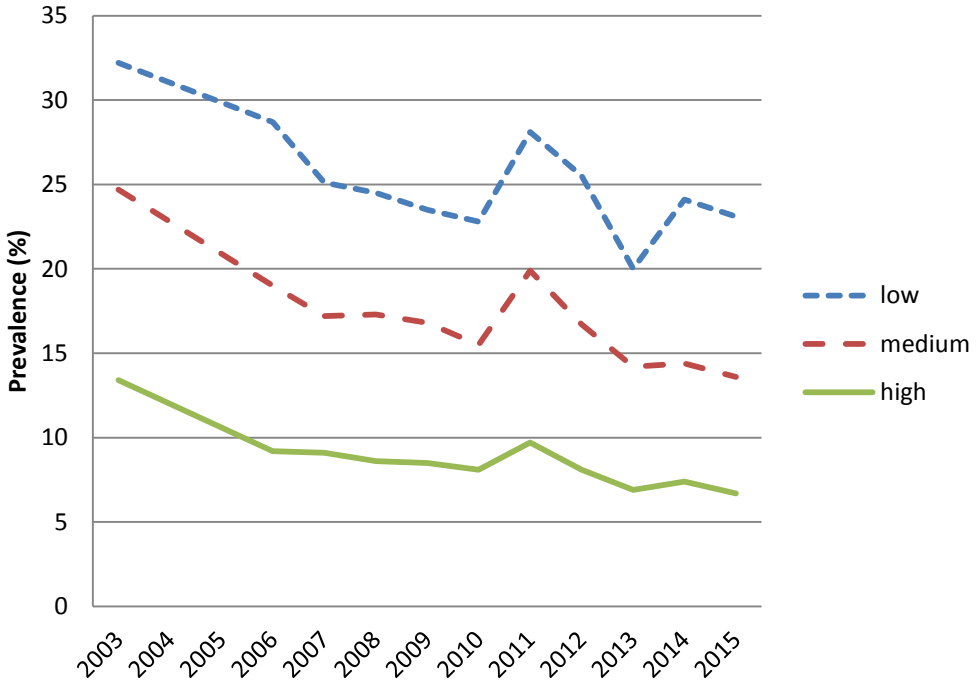
Supplementary Figure S9: Proportion reporting best friend smokes, by school decile, 2002 to 2015.



Supplementary Figure S9: Proportion reporting daily exposure to smoking in the home, by ethnicity, 2002 to 2015.



Supplementary Figure S10: Proportion reporting daily exposure to smoking in the home, by school decile, 2002 to 2015.



Testing for difference over time in odds ratios

To test whether odds ratios changed significantly between 2003 and 2015, we re-ran the GLIMMIX multivariable analysis using the data from years 2003 and 2015 only. We then tested whether there was an interaction effect between year and the risk factors in the model. The results were:

Table S2: Interaction effects between risk factors and year

Type III Tests of Fixed Effects				
Effect	**Num DF**	**Den DF**	**F Value**	**Pr > F**
Age	1	51799	28.01	<.0001
Gender	1	7230	91.49	<.0001
School decile (low, med, high)	2	313.7	6.78	0.0013
Ethnicity (prioritised)	1	45690	1.93	0.1650
Parental smoking	3	51799	10.66	<.0001
Sibling smoking	1	51799	255.85	<.0001
Best friend smoking	1	51799	3280.99	<.0001
Exposure to smoking in the home (none, daily, less than daily)	2	51799	119.98	<.0001
Year	1	17216	451.36	<.0001
Year*parental smoking	3	51799	0.32	0.8078
Year*sibling smoking	1	51799	3.41	0.0650
Year*best friend smoking	1	51799	20.18	<.0001
Year*exposure to smoking in the home.	2	51799	15.30	<.0001

Based on these findings we can conclude that the ORs (i.e. the association between risk factors and regular smoking) differed between 2003 and 2015 for *best friend smoking* and *exposure to smoking in the home,* as both interactions were statistically significant at the P<0.0001 level.

Associations between risk factors and regular smoking in Māori adolescents, 2003 and 2015

Supplementary Table S3: Associations between risk factors and regular smoking among Māori adolescents, 2003 and 2015, expressed as odds ratios and 95% confidence intervals.

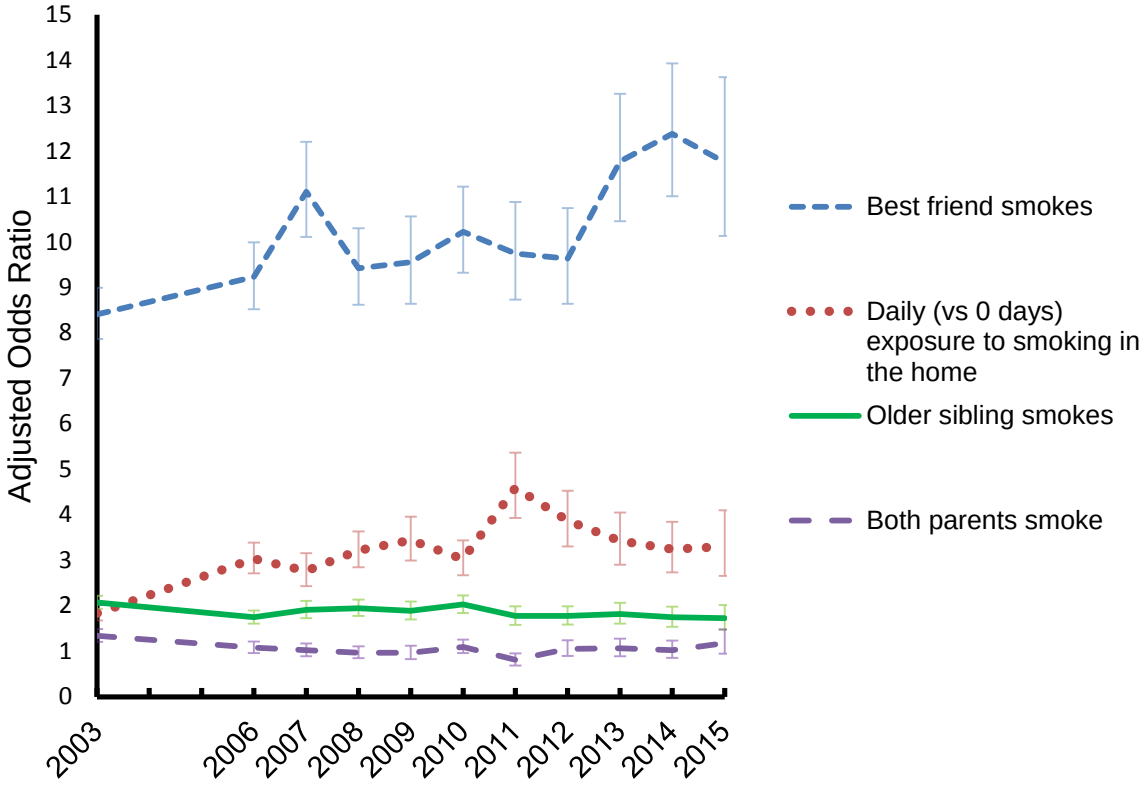
	MĀORI					
	2003 N=5,425			**2015** N=4,215		
	Prevalence of exposure	Model 1 (unadjusted)	Model 4 (fully adjusted)	Prevalence	Model 1 (unadjusted)	Model 4 (fully adjusted)
No exposure to smoking in the home (0 days)	52.0	1.0	1.0	50.6	1.0	1.0
Less than daily (1-6 days) exposure to smoking in the home	38.0	1.9 (1.6, 2.3)	1.6(1.3, 2.0)	25.3	3.7(2.8, 4.8)	2.8 (2.1, 3.9)
Daily (7 days) exposure to smoking in the home	47.6	2.7 (2.4, 3.1)	1.9 (1.6, 2.1)	49.4	6.9 (5.3, 8.9)	4.2 (3.0, 5.9)
Neither parent smokes	36.0	1.0	1.0	43.6	1.0	1.0
Only mother smokes	20.1	2.0 (1.7-2.3)	1.3 (1.1-1.6)	17.5	2.2 (1.6-2.9)	0.8 (0.5-1.1)*
Only father smokes	13.8	1.9 (1.6-2.3)	1.5 (1.2-1.8)	14.0	1.8 (1.3-2.5)	0.9 (0.6-1.3)*
Both parents smoke	30.2	2.7 (2.3-3.1)	1.2 (1.0-1.4)*	24.8	3.7 (2.9-4.7)	0.9 (0.7-1.3)*
Older sibling smokes	42.5	2.4 (2.1-2.7)	1.6 (1.4-1.9)	32.6	2.9 (2.4-3.5)	1.5 (1.2-1.9)
Best friend smokes	46.0	6.8 (6.0-7.8)	5.5 (4.8-6.3)	18.5	11.2 (9.0-13.4)	8.2 (6.5-10.2)

Notes: Fully adjusted model includes age, sex, school decile, past week exposure to smoking in the home (none, less than daily, daily), parental smoking (neither, mother only, father only, both), older sibling smokes, best friend smokes.

All associations are statistically significant at the 95% confidence level except those marked with an asterisk (*)

Trends in associations between risk factors and regular smoking, 2003 to 2015.

Supplementary Figure S11: Fully adjusted odds ratios for key risk factors, 2003 to 2015.



STROBE Statement—Checklist of items that should be included in reports of ***cross-sectional studies***

	Item No	Recommendation	Page no.
Title and abstract	1	(*a*) Indicate the study's design with a commonly used term in the title or the abstract	1
		(*b*) Provide in the abstract an informative and balanced summary of what was done and what was found	2
Introduction			
Background/rationale	2	Explain the scientific background and rationale for the investigation being reported	3-6
Objectives	3	State specific objectives, including any prespecified hypotheses	6
Methods			
Study design	4	Present key elements of study design early in the paper	6-7
Setting	5	Describe the setting, locations, and relevant dates, including periods of recruitment, exposure, follow-up, and data collection	6-8
Participants	6	(*a*) Give the eligibility criteria, and the sources and methods of selection of participants	7-8
Variables	7	Clearly define all outcomes, exposures, predictors, potential confounders, and effect modifiers. Give diagnostic criteria, if applicable	9-10
Data sources/ measurement	8*	For each variable of interest, give sources of data and details of methods of assessment (measurement). Describe comparability of assessment methods if there is more than one group	*9-10*
Bias	9	Describe any efforts to address potential sources of bias	NA
Study size	10	Explain how the study size was arrived at	7-8
Quantitative variables	11	Explain how quantitative variables were handled in the analyses. If applicable, describe which groupings were chosen and why	9-10
Statistical methods	12	(*a*) Describe all statistical methods, including those used to control for confounding	10-11
		(*b*) Describe any methods used to examine subgroups and interactions	10-11
		(*c*) Explain how missing data were addressed	7
		(*d*) If applicable, describe analytical methods taking account of sampling strategy	10.
		(*e*) Describe any sensitivity analyses	NA
Results			
Participants	13*	(a) Report numbers of individuals at each stage of study—eg numbers potentially eligible, examined for eligibility, confirmed eligible, included in the study, completing follow-up, and analysed	7-8
		(b) Give reasons for non-participation at each stage	NA
		(c) Consider use of a flow diagram	NA
Descriptive data	14*	(a) Give characteristics of study participants (eg demographic, clinical, social) and information on exposures and potential confounders	8+Supl
		(b) Indicate number of participants with missing data for each variable of interest	NA
Outcome data	15*	Report numbers of outcome events or summary measures	11

Main results	16	(*a*) Give unadjusted estimates and, if applicable, confounder-adjusted estimates and their precision (eg, 95% confidence interval). Make clear which confounders were adjusted for and why they were included	12-15
		(*b*) Report category boundaries when continuous variables were categorized	NA
		(*c*) If relevant, consider translating estimates of relative risk into absolute risk for a meaningful time period	NA
Other analyses	17	Report other analyses done—eg analyses of subgroups and interactions, and sensitivity analyses	11-16 +Suppl
Discussion			
Key results	18	Summarise key results with reference to study objectives	13-14
Limitations	19	Discuss limitations of the study, taking into account sources of potential bias or imprecision. Discuss both direction and magnitude of any potential bias	16-17
Interpretation	20	Give a cautious overall interpretation of results considering objectives, limitations, multiplicity of analyses, results from similar studies, and other relevant evidence	21
Generalisability	21	Discuss the generalisability (external validity) of the study results	20
Other information			
Funding	22	Give the source of funding and the role of the funders for the present study and, if applicable, for the original study on which the present article is based	21

*Give information separately for exposed and unexposed groups.

Note: An Explanation and Elaboration article discusses each checklist item and gives methodological background and published examples of transparent reporting. The STROBE checklist is best used in conjunction with this article (freely available on the Web sites of PLoS Medicine at http://www.plosmedicine.org/, Annals of Internal Medicine at http://www.annals.org/, and Epidemiology at http://www.epidem.com/). Information on the STROBE Initiative is available at www.strobe-statement.org.

BMJ Open

Why has adolescent smoking declined dramatically? Trend analysis using repeat cross-sectional data from New Zealand 2002-2015.

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Manuscript ID	bmjopen-2017-020320.R2
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Date Submitted by the Author:	05-Jul-2018
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Primary Subject Heading:	Smoking and tobacco
Secondary Subject Heading:	Public health, Paediatrics, Epidemiology
Keywords:	PAEDIATRICS, PREVENTIVE MEDICINE, PUBLIC HEALTH, EPIDEMIOLOGY

Why has adolescent smoking declined dramatically? Trend analysis using repeat cross-sectional data from New Zealand 2002-2015.

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Word Count: 3874

ABSTRACT

Objectives: Adolescent smoking has declined in New Zealand and in many other countries since the late 1990s, yet the reasons for the decline are not well understood. We investigated the extent to which established risk factors for adolescent smoking (parental, sibling and peer smoking, and exposure to smoking in the home) explained the downward trend.

Design: Trend analysis of repeat cross-sectional data from an annual nationally representative survey.

Setting: New Zealand

Participants: Secondary school students aged 14-15. N=398,221

Outcome measure: Regular (at least monthly) smoking

Methods: For each risk factor (parental smoking, best friend smoking, older sibling smoking, and past week exposure to smoking in the home) we plotted prevalence of exposure, 2002 – 2015. Next, using multivariable logistic regression, we modelled the trend in regular smoking (expressed as an odds ratio for year) adjusting for age, sex, ethnicity and socioeconomic position. The risk factors were added to the model – individually and collectively - to test whether they attenuated the odds ratio for year.

Results: Exposure to all risk factors except 'past week exposure to smoking in the home' decreased between 2002 and 2015. We observed a strong downward trend in regular smoking among adolescents (odds ratio = 0.88 per year, 95% CI 0.88, 0.88, p<.001). 'Best friend smoking' was the only risk factor that significantly attenuated the trend. However, due to

circularity, this factor provides an unsatisfactory explanation for population level smoking decline.

Conclusions: The established risk factors that we explored do not appear to have contributed to the remarkable decline in adolescent smoking in New Zealand between 2003 and 2015. Further research is needed to assess the possible contribution of factors outside our model, such as changes in the policy context, the social meaning of smoking and broader social and economic conditions.

ARTICLE SUMMARY

Strengths and limitations of this study

- The survey has a large sample size (N=20,443 - 31,833 per year), allowing precise population estimates based on individual-level data.
- Due to data limitations, the study only includes a small number of risk factors, however the risk factors included have consistently been found to be among the strongest and most important predictors of adolescent smoking.
- Our study design (using repeat cross sectional data) does not enable causal inferences to be drawn; rather our study draws on existing knowledge about the predictors of adolescent smoking initiation.

INTRODUCTION

Smoking is a leading cause of preventable illness and premature death[1] and a key driver of health disparities between ethnic and socioeconomic groups.[2 3] Long-term tobacco use typically begins with experimental smoking in adolescence,[4] and, internationally, considerable research and policy attention has focused on understanding and preventing smoking uptake in this age group.

The dramatic decline in adolescent smoking observed since the late 1990s in many high income countries is good news from a public health perspective. In New Zealand (NZ), for example, regular smoking (defined as at least monthly) among 14-15 year olds declined from a peak of 29% in 1999 to 5% in 2015, with decreases across all main ethnic groups, and a convergence between boys and girls over the period.[5] Over the same period, the proportion who had never smoked (i.e. not even a few puffs) rose from 32% to 79%.[5] However, as in other countries,[6] ethnic and socioeconomic disparities remain pronounced. For example Māori (indigenous) smoking prevalence in this age group was 11% in 2015 compared to 4% among non-Māori.[7]

Other countries including the USA, England and Australia have also experienced a decline in adolescent smoking from the late 1990s, following a sharp rise in the early 1990s.[8] It is important to understand the causes of this decline in order to help ensure it is sustained, and to enable replication in other countries. Yet little research has focused on explaining this phenomenon. Public health interventions such as increases in tobacco tax or smokefree environment legislation may have played a role,[9-11] but they do not fully explain the observed trends, since declines in adolescent smoking have occurred almost simultaneously in countries with widely differing regulatory contexts. This, and the fact that other adolescent risk behaviours (e.g. alcohol use, teen pregnancy) have also declined over a similar time period,[8] suggests broader social or cultural changes rather than specific tobacco control policies may contribute to this international trend.

Such shifts could be generated by new technologies, for example. When the use of cell phones rose and smoking fell among adolescents in the late 1990s, a causal association was hypothesised.[12] More recently, attention has turned to other new technologies – smartphones

and social media - and their potential role in driving generational change in attitudes and behaviour.[13] There is face validity to the idea that these new technologies may have changed the way young people socialise or project their identity, displacing the role of smoking or providing less opportunity for it. However, this hypothesis is challenged by a consistent body of evidence showing a *positive* association between smoking and cell phone/ internet/social media use at the individual level.[14-23]

Other major changes since the mid-90s that could potentially impact on youth behaviour include changes in parenting;[24-26] changes in the school environment and ethos; [27-29] and broad economic and labour market conditions resulting in young people leaving school and achieving independence later than previous cohorts.[30 31] But before exploring these macro level explanations for smoking decline, an initial step is to determine the extent to which the observed trends can be explained by changing exposure to established individual-level predictors of smoking initiation.

Proximal risk factors for adolescent smoking have been studied extensively. Parental, sibling and peer smoking have consistently been identified as key risk factors,[4 32] with the Surgeon General's 2012 evidence review concluding that the evidence is suggestive of a causal role for peer influences, and a potential causal role for parental smoking.[4] The review found that smoking by older siblings influences smoking in adolescents more consistently than does smoking by parents.[4] Exposure to smoking in the home, although a less studied factor, has also been shown to predict smoking in adolescents independent of parental smoking status in longitudinal and cross sectional studies.[33-37] Studies suggest second hand smoke exposure may biologically predispose children to nicotine dependence [38-42] in addition to providing pro-smoking socialization.[34] Could declining exposure to these proximal risk factors explain the dramatic decline in adolescent smoking since the turn of the century?

Despite extensive risk factor research, few studies have explored how exposure to risk factors has changed over time, or how such changes may be contributing to changes in adolescent smoking at the population level. The current study explores trends in exposure of 14-15 year olds to known risk factors for adolescent smoking (parental, sibling and peer smoking and exposure to smoking in the home) and investigates the extent to which these risk factors could explain the declining trend in adolescent smoking in NZ from 2003 to 2015.

METHODS

Data

We used repeat cross-sectional data from the ASH NZ Year 10 Snapshot Survey series, an annual school-based survey of 14-15 year olds, which is administered by Action on Smoking and Health NZ (ASH NZ) and is part of the New Zealand Youth Tobacco Monitor. The questionnaire includes a set of 'core' questions that have remained consistent over time to enable trend monitoring, and additional questions which change from year to year. Years included in the current study were 2002-2015, since key variables of interest were unavailable prior to 2002. Furthermore, exposure to smoking in the home was not included in the questionnaire in 2002 or 2004-5, and therefore multivariable trend analysis includes only data from 2003 and 2006-15.

All public and private schools with Year 10 students were invited to participate in the ASH NZ Year 10 Snapshot each year. Table 1 shows the sample size and student response rate (as a proportion of the total NZ Year 10 population) by year. Non-response was almost entirely at the school level, with school response rates ranging from 44-67%. [43] (The lower school response rate in 2015 was due to limited resources for liaising with schools that year).

Following previously published ASH NZ analyses, our analysis was restricted to respondents aged 14 or 15 at the time of the survey. For consistency between descriptive and

multivariable (i.e. adjusted) analyses, only respondents with complete data for all variables (smoking status, parental smoking, sibling smoking, best friend smoking, age, gender, ethnicity, school decile, and school ID, and for 2003 and 2006-15 exposure to smoking in the home) were included in the analyses. In addition, only schools with at least 20 respondents were included so that results were based on stable estimates of smoking in each school. Table 1 shows the number of valid survey responses received based on the ASH NZ criteria for inclusion (i.e. those with complete data for age (14/15 years), sex, ethnicity and smoking status), and the number included in our study (after exclusions above), by year. After application of our additional inclusion criteria 96% (398,221/ 414,254) of valid responses were included.

Table 1. Sample size and student response rate by year

Year	NZ Year 10 population	Valid survey responses*	Valid survey responses that met all study inclusion criteria	% Year 10 population that met all study inclusion criteria
2002	58,812	29,173	28,088	50%
2003	61,028	32,705	31,377	54%
2004	62,852	31,630	30,807	46%
2005	64,619	32,561	31,833	51%
2006	63,086	32,844	31,690	52%
2007	62,012	25,978	25,109	42%
2008	61,485	30,903	29,682	50%
2009	61,355	25,757	24,755	42%
2010	61,210	32,832	31,696	54%
2011	59,562	26,856	26,028	45%
2012	59,627	31,983	30,396	43%
2013	57,929	28,340	27,014	49%
2014	59,612	31,125	29,303	47%
2015	59,528	21,567	20,443	36%
Total	**852,717**	**414,254**	**398,221**	**47%**

*Valid survey responses = those with complete data for age (14 or 15), sex, ethnicity and smoking status

The final included sample (N=398, 221) comprised approximately half the Year 10 population each year, and closely resembled the population in respect of demographic characteristics. A detailed comparison of the final included sample and population, by year, is provided in a Supplementary Table S1, showing that the sample is broadly representative albeit with modest but consistent under-representation of Māori and students from low decile schools. (School decile is a school-level measure of the socio-economic position of a school's student community, explained further below.[44])

Fieldwork was undertaken earlier in the year in 2011 and subsequently, meaning respondents were 2-3 months younger on average in 2011 and subsequent years, than in 2010 and prior years. Excluding the timing of fieldwork and changes to non-core questions, there has been consistency in survey instruments, administration and data management across included years.

In participating schools, the one-page survey is completed in class time under the supervision of teaching staff. Individual students may choose not to participate. To protect the confidentiality of students' responses, identifying information is not collected, and teachers are requested not to check the completed surveys. Completed surveys are returned to ASH NZ, which oversees data entry, cleaning and coding.

The survey was approved, as a component of the NZ Youth Tobacco Monitor, by the Ministry of Health Multiregional Health and Disability Ethics Committee in 2007. Further details on survey methodology are available elsewhere.[43]

Variables

The outcome variable, 'regular smoking' (Y/N) was defined as smoking at least monthly, based on the question 'How often do you smoke now?' The answer categories were: 'I have

never smoked/I am not a smoker now'; 'At least once a day'; 'At least once a week'; 'At least once a month'; and 'Less often than once a month'.

Smoking status of mother, father, older sibling(s), and best friend were based on the question 'Which of the following people smoke?' with a dichotomous variable (current smoker, yes/no) created for each. Previous research shows that maternal smoking is more strongly associated with adolescent smoking initiation than paternal smoking,[45] therefore we examined exposure to maternal and paternal smoking separately. For the purposes of multivariable analysis, parental smoking was grouped into one variable, coded 0 = neither parent smokes, 1 = only mother smokes, 2 = only father smokes, 3 = both parents smoke.

Past week exposure to smoking in the home was based on the question 'During the past 7 days, on how many days have people smoked around you in your home?' Response categories were 0 days, 1-2 days, 3-4 days, 5-6 days and 7 days. For descriptive analysis only, we recoded the responses into three categories: 'Daily exposure' (7 days) 'less than daily exposure' (1-6 days) and 'no exposure' (0 days).

In previous research, using the same data set, we confirmed that all the above risk factors were significantly associated with adolescent smoking, and that these associations remained significant throughout the study period.[46]

Demographic variables were age (14 or 15 years old), sex (male or female), ethnicity (prioritised Māori, Pacific, Asian, NZ European/other [NZEO]); and school decile. School decile is calculated by the Ministry of Education for purposes of funding allocation, and is a school-level measure of the socioeconomic position (SEP) of a school's student community. Details of how school decile is calculated are available from the Ministry of Education.[44] For descriptive analysis only, we grouped school decile into low (deciles 1-3: most deprived),

medium (4-7), and high (8-10: least deprived). Each school also had an identification number (school ID) which was assigned to all respondents from that school.

Analysis

To describe trends we used SPSS (IBM Corp. Released 2016. IBM SPSS Statistics for Windows, Version 24.0. Armonk, NY: IBM Corp.) to tabulate prevalence of regular smoking and prevalence of exposure to risk factors (overall and by sex, ethnicity and school decile) for each year. We then quantified the mean annual absolute change in proportion of respondents exposed to each risk factor using weighted linear regression (to adjust for differing variance by year by giving more weight to more accurate estimates of prevalence) with year as the independent variable. The weights were $1/SD^2$ of the proportions.

Next, for the years 2003 and 2005-2015, we conducted trend analyses based on individual-level data using multivariable logistic regression. We used SAS/STAT software (Version 9.4 of the SAS system for Windows. Copyright © 2002-2012, SAS Institute Inc., Cary, NC, USA). To test the extent to which the risk factors of interest accounted for the change over time in adolescent smoking, we modelled regular smoking as a function of survey year, adjusting for demographic factors (age, sex, ethnicity and school decile), and including school ID as a random effect to account for clustering at the school level (Model 1). We then added the risk factors of interest to Model 1, first individually then collectively. Attenuation of the odds ratio (OR) for year, which was tested using Z tests to compare log odds, would indicate that the risk factor (partially) accounted for the trend over time.

Initially we modelled the trend using year as a continuous variable, which provided a single OR describing average annual change in the odds of regular smoking compared with the reference year, 2003. This approach assumes a linear trend over time, which may not be

valid, so we also modelled the trend using year as a categorical variable. This provided an OR for regular smoking for each survey year 2006-2016, compared with the reference year.

To test whether the results were the same for Māori adolescents as for the sample as a whole, we re-coded ethnicity into Māori (yes/no) and repeated the trend analysis above for Māori only.

Patient and Public Involvement

Patients were not involved in the design or conduct of this study, and nor were members of the general public.

RESULTS

Prevalence of regular smoking

There was a long-term decline in prevalence of regular smoking among 14-15 year olds from 22% in 2002 to 5% in 2015 (Figure 1). Based on weighted linear regression, the overall regular smoking rate reduced by an average of 1.2 % per year (in absolute terms) from 2002-2015.

Supplementary Figures S1, S2 and S3 show trends in prevalence of regular smoking stratified by ethnicity, school decile and gender, and indicate that smoking has declined in all demographic groups. Furthermore ethnic, SEP, and gender disparities have narrowed over time in absolute terms.

Changes in exposure to risk factors over time

Trends in exposure to risk factors are shown in Figure 1. Parental smoking (Fig 1a) declined only modestly over the study period with maternal and paternal smoking both declining by an

average rate of 0.5% per annum. As shown in Figure 1b smoking among older siblings declined slightly more, at an average rate of 0.7% per annum, and 'best friend smokes' had the highest rate of decline at 1.5% per annum. Past week exposure to smoking in the home did not change significantly over the study period. Prevalence of daily exposure to smoking in the home fell from 22% to 13% overall (an average decrease of 0.6% per annum), however less than daily exposure increased over the study period.

Supplementary Figures S4 to S10 show exposure to risk factors over time by ethnicity and school decile. They show that trends in exposure to risk factors followed a similar pattern in all ethnic and SEP subgroups, but disparities in levels of exposure were marked at all years.

Trend analyses

Results of the trend analyses are shown in Table 2. We observed a strong downward trend in regular smoking among 14-15 year olds, with an OR of 0.88 per year (95% CI 0.88, 0.88, p<.001) based on the linear trend. When 'best friend smokes' was added to the model (Model 1 +Best friend smokes) the size of the OR declined significantly (Model 1), indicating that this risk factor partially (but not fully) accounted for the declining trend in adolescent smoking between 2003 and 2015. None of the other risk factors, when added to Model 1, significantly attenuated the OR for year relative to the reference year, indicating that, individually, they did not contribute to the trend.

Table 2: Results of multiple logistic regression analyses examining the impact of risk factors on the trend in regular smoking in adolescents.

Year	Model 1: OR for Year Partially adjusted (95% CI)	Model 1 + Best Friend smokes (95% CI)	Model 1 + Exposure to smoking in home (95% CI)	Model 1 + smoke parent4	Model 1 + smoke Sibling	Model 1 + all risk factors
2003	1					
2006	0.62	0.83*	0.58	0.60	0.61	0.75*

	(0.59, 0.64)	(0.78, 0.87)	(0.55, 0.61)	(0.57, 0.63)	(0.58, 0.64)	(0.71, 0.79)
2007	0.56 (0.53, 0.59)	0.78* (0.74, 0.83)	0.54 (0.52, 0.57)	0.55 (0.52, 0.58)	0.56 (0.54, 0.59)	0.72* (0.68, 0.76)
2008	0.51 (0.48, 0.53)	0.74* (0.70, 0.79)	0.49 (0.47, 0.52)	0.50 (0.48, 0.53)	0.51 (0.48, 0.53)	0.68* (0.64, 0.72)
2009	0.47 (0.44, 0.49)	0.69* (0.65, 0.73)	0.46 (0.43, 0.49)	0.46 (0.44, 0.49)	0.47 (0.45, 0.50)	0.63* (0.59, 0.67)
2010	0.40 (0.38, 0.42)	0.58* (0.54, 0.61)	0.40 (0.38, 0.42)	0.39 (0.37, 0.41)	0.40 (0.38, 0.42)	0.53* (0.50, 0.57)
2011	0.36 (0.34, 0.38)	0.55* (0.51, 0.58)	0.33 (0.31, 0.35)	0.36 (0.34, 0.38)	0.37 (0.35, 0.39)	0.47* (0.44, 0.51)
2012	0.28 (0.27, 0.30)	0.42* (0.39, 0.45)	0.26 (0.25, 0.28)	0.29 (0.27, 0.30)	0.29 (0.27, 0.31)	0.37* (0.35, 0.40)
2013	0.27 (0.25, 0.28)	0.41* (0.39, 0.44)	0.28 (0.26, 0.29)	0.27 (0.26, 0.29)	0.28 (0.26, 0.30)	0.39* (0.37, 0.42)
2014	0.24 (0.22, 0.25)	0.36* (0.34, 0.39)	0.23 (0.22, 0.25)	0.24 (0.23, 0.26)	0.25 (0.24, 0.27)	0.34* (0.32, 0.36)
2015	0.21 (0.19, 0.22)	0.34* (0.32, 0.37)	0.20 (0.19, 0.22)	0.21 (0.20, 0.23)	0.22 (0.21, 0.24)	0.31* (0.29, 0.34)
All years combined, using year as a continuous variable						
Linear trend (2003 - 2015)	0.88 (0.88, 0.88)	0.91* (0.91,0.92)	0.88 (0.88, 0.88)	0.88 (0.88,0.89)	0.88 (0.88, 0.89)	0.91* (0.90, 0.91)

Model 1 is adjusted for age, gender, ethnicity and school decile.

*OR > Model 1 OR ($p < .05$)

When all four risk factors were entered into the model together (Model 1+ all risk factors), the attenuation of the OR was significant ($p<.05$) but the magnitude of the change was no greater than for 'Model 1 + Best friend smokes'.

The pattern of results described above was observed regardless of whether year was used as a categorical variable (modelling change relative to 2003 for each year), or a continuous variable (modelling the linear trend, to give an annual average change over the study period, as shown in the final row of Table 2). The same patterns were also seen in Māori respondents (see Supplementary Table S2).

There was a residual effect of year (i.e. unexplained change over time indicated by an OR for year that was significantly less than 1) in all the models, including the fully adjusted model. This suggests that there were factors outside our fully adjusted model that were influencing the change over time in smoking prevalence.

DISCUSSION

Exposure to best friend smoking declined strongly during the 2002-2015 period, whilst exposure to other established risk factors for smoking decreased more modestly, if at all. There was no change in past week exposure to smoking in the home overall (i.e.1-7 days), but daily exposure fell significantly, whilst less than daily exposure increased. The primary aim of the study was to determine whether these known risk factors explained (in statistical terms) the dramatic decline in adolescent smoking seen recently in NZ. Despite declining exposure to many of the included risk factors, modelling showed that most of the factors we explored (parental and sibling smoking, and exposure to smoking in the home) did not account for the trend in any measurable way, either individually or collectively.

Only 'best friend smokes' appeared to contribute to the declining trend in adolescent smoking. This was unsurprising, given that exposure to this risk factor declined markedly over the study period, and a previous study using the same data set has shown that the smoking status of respondents' best friend was by far the strongest risk factor for regular smoking in NZ 14-15 year olds of the factors we explored,[46] However research on peer

influence suggests that causality is likely to be bidirectional, and the association is due, in part, to smokers seeking out other smokers as friends.[4] Furthermore, at the population level, it would be a circular to suggest that declining best friend smoking explained the decline in adolescent smoking, since survey respondents and their best friends belong to the same cohort of adolescents in which smoking is declining. The question remains: if decreases in best friend smoking are resulting in reduced risk of adolescent smoking at the individual level, what is driving the decline in best friend smoking?

One possibility is that, since younger adolescents are strongly influenced by peers and adolescents slightly older then themselves, a virtuous cycle may have developed whereby a decline in adolescent smoking at time 1 has led to a subsequent decline in adolescent smoking at time 2 and so on. Further research, perhaps drawing on communicable disease methodology, could test this hypothesis and explore how the 'social transmission' of smoking (and other health risk behaviours) influences population prevalence over time. Should this hypothesis prove to be correct, the trigger for the sudden change from rapidly rising to rapidly falling adolescent tobacco use from the late 1990s to the early 2000s still remains to be identified.

Our findings suggest that there are other factors influencing the decline in adolescent smoking that this study did not address. For example, it is possible that changes in the social meaning of smoking[47 48] and the policy context[11 49] – factors that were not included in our analyses - may have played a role in triggering adolescent smoking decline. For example, policy responses to rising adolescent smoking in the 1990s may have influence teen smoking in NZ, as they appear to have done in Australia,[10 11] the UK[6] and the US.[9] In New Zealand such policy responses included raising the legal age of tobacco purchase from 16 to 18 years of age in 1997, a tax increase which raised the price of a packet of 20 cigarettes by 13% in 1998,[50] and the 'Why start?' mass media campaign which ran from 1996-1998. It is plausible

that, collectively, these measures contributed to the denormalisation of smoking, which, together with any specific intervention effects, may have been the trigger for adolescent smoking decline both in NZ and other jurisdictions. Mass media campaigns from 2000 focusing on second hand smoke and a 2004 ban on smoking in pubs and all other indoor workplaces likely contributed to the ongoing denomalisation of smoking (in particular indoor smoking) in NZ,[51] and may underpin the observed decline in daily exposure to smoking in the home. As Simon Chapman has pointed out, denormalisation involves an 'interplay of continuous, uncontrolled, unmeasured, and sometimes unmeasurable variables intended to influence [tobacco] consumption', and cannot be reduced to the sum of its parts.[52]

However if tobacco denormalisation, along with the other factors discussed above, explains the decline in adolescent smoking, is it simply a coincidence that adolescent alcohol use, teen pregnancy and juvenile crime have also declined over the same period? Or does this suggest there are additional over-arching influences that are impacting on a range of adolescent risk-taking behaviours?

As far as we are aware, this is the first study to explore trends in exposure to known risk factors with the purpose of better understanding the drivers of the decline in adolescent smoking prevalence. Definitively establishing the reason(s) for the decline in adolescent smoking is not possible using repeat cross-sectional data (or indeed via any single study). However, trend analysis using statistical modelling allowed us to explore the relationships between survey year, risk factors and outcomes, and thereby (potentially) account for changes over time in statistical terms. This approach has allowed us to rule out hypothesised explanations for population level change over time, and adds to the evidence base about the most likely explanations for the decline of smoking in young people.

Strengths of the study include the large sample size, and demographic similarity between the sample and the Year 10 population, suggesting response bias was not a substantial issue. Systematic under- and over-representation were found to be relatively consistent over time and therefore unlikely to affect trend analysis, which was the focus of our study. The methods for the ASH NZ survey were broadly consistent between years, with minor changes (e.g. a change in fieldwork timing from 2011) unlikely to contribute significantly to the trends observed. Since there is strong similarity between NZ and other countries at a late stage in the tobacco epidemic in terms of trends in adolescent smoking and known risk factors, it is likely that our conclusions may be generalisable to similar countries, but this remains to be confirmed through further research.

Given the complex array of factors at various levels that are known to influence smoking uptake, one of the limitations of our study was the limited number of risk factors for which consistent data was available. Clearly, there are other contributing factors, and our study was unable to explore these. The study was based on self-report questionnaire data, with its inherent limitations (e.g. potential for social desirability bias, and misinterpretation of questions resulting in misclassification); however recent biomarker testing of a sub-sample of ASH NZ Year 10 participants indicated that the survey provides an accurate population estimate of smoking prevalence.[53] We used school decile as a proxy for SEP, since more direct measures were unavailable. Because school communities are heterogeneous, it is an imperfect measure at the individual level, and residual confounding by SEP is possible in our adjusted analyses.

Conclusions

In summary, our findings suggest that the remarkable decline in adolescent smoking in New Zealand cannot be explained by declining exposure to parental smoking, sibling smoking or past week exposure to smoking in the home. These factors have not contributed measurably

to the trend, either individually or collectively. Declining 'best friend smoking' partially accounts for declining adolescent smoking in our statistical model, but this finding contributes little to our understanding of the drivers of population level decline since respondents and their best friends largely come from the same population. It is clear that factors other than those in our model are at play, with changes in the social meaning of smoking, the policy context and broader socio-cultural changes all potential contributors. Further research is needed to identify other contributing factors and determine their relative importance.

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Contributorship statement

All authors contributed to the conception and design of the study. The analysis was executed by DS and JB, with oversight by RE. JB drafted the manuscript with input from all authors.

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Competing interests

None declared.

Data sharing statement

The data is owned by ASH New Zealand, and cannot be shared by the authors.

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Fig 1a. Trends in adolescent and parental smoking prevalence, 2002-2015

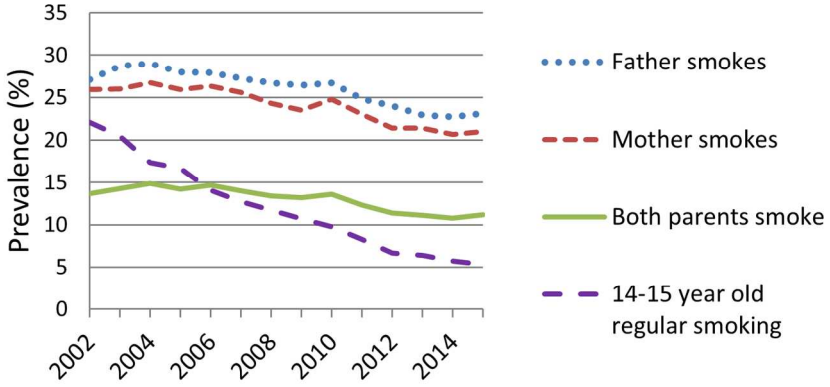


Fig 1b. Trends in exposure to other risk factors, 2002-2015

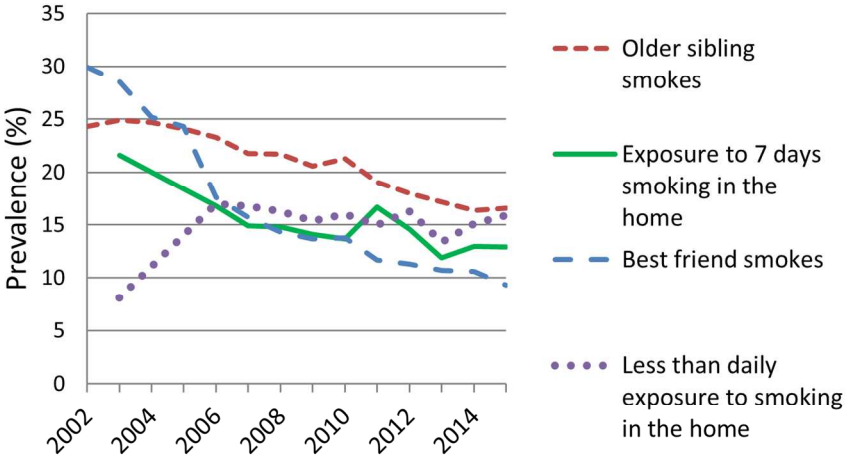


Fig 1: Prevalence of regular smoking and risk factors in 14-15 year olds, 2002 to 2015.

137x178mm (300 x 300 DPI)

SUPPLMENTARY MATERIAL

Characteristics of the sample

Supplementary Table S1: Sample characteristics and comparison with Year 10 population (% absolute difference)

			Age		Sex		School Decile			Ethnicity			
		Total	14 years	15 years	Male	Female	Low	Medium	High	NZEO	Maori	Pacific	Asian
2002	**N**	28088	13161	14927	13911	14177	4505	13007	10576	19395	4670	1833	2190
	%		47%	53%	50%	50%	16%	46%	38%	69%	17%	7%	8%
	dif from pop				*-1%*	*1%*	*-4%*	*2%*	*4%*				
2003	**N**	31377	14585	16792	15492	15885	5272	14274	11831	20716	5425	2174	3062
	%		46%	54%	49%	51%	17%	45%	38%	66%	17%	7%	10%
	dif from pop				*-2%*	*2%*	*-3%*	*3%*	*5%*				
2004	**N**	30807	18668	12139	14996	15811	5889	13373	11545	19888	5694	2244	2981
	%		61%	39%	49%	51%	19%	43%	37%	65%	18%	7%	10%
	dif from pop				*-2%*	*2%*	*-1%*	*1%*	*4%*				
2005	**N**	31833	15901	15932	15211	16622	4719	15374	11740	21176	5435	2172	3050
	%		50%	50%	48%	52%	15%	48%	37%	67%	17%	7%	10%
	dif from pop				*-3%*	*3%*	*-3%*	*2%*	*5%*				
2006	**N**	31690	19996	11694	15642	16048	4416	15935	11339	20161	6237	2252	3040
	%		63%	37%	49%	51%	14%	50%	36%	64%	20%	7%	10%
	dif from				*-2%*	*2%*	*-4%*	*4%*	*4%*	*2%*	*-2%*	*-1%*	*1%*

	pop												
2007	**N**	25109	16213	8896	11988	13121	3558	11062	10489	16187	4653	1804	2465
	%		65%	35%	48%	52%	14%	44%	42%	64%	19%	7%	10%
	dif from pop				*-4%*	*4%*	*-4%*	*-2%*	*10%*	*5%*	*-3%*	*-2%*	*0%*
2008	**N**	29682	18395	11287	14462	15220	4732	12875	12075	17913	5552	2863	3354
	%		62%	38%	49%	51%	16%	43%	41%	60%	19%	10%	11%
	dif from pop				*-3%*	*3%*	*-2%*	*0%*	*3%*	*1%*	*-3%*	*1%*	*1%*
2009	**N**	24755	15977	8778	11607	13148	3235	11029	10491	15410	4304	2211	2830
	%		65%	35%	47%	53%	13%	45%	42%	62%	17%	9%	11%
	dif from pop				*-4%*	*4%*	*-5%*	*1%*	*4%*	*4%*	*-5%*	*0%*	*2%*
2010	**N**	31696	19725	11971	16636	15060	5111	14049	12536	18806	6315	3041	3534
	%		62%	38%	52%	48%	16%	44%	40%	59%	20%	10%	11%
	dif from pop				*1%*	*-1%*	*-2%*	*1%*	*2%*	*2%*	*-3%*	*0%*	*1%*
2011	**N**	26028	21390	4638	12462	13566	3720	10843	11465	15669	4995	2453	2911
	%		82%	18%	48%	52%	14%	42%	44%	60%	19%	9%	11%
	dif from pop				*-3%*	*3%*	*-4%*	*-1%*	*6%*	*3%*	*-4%*	*0%*	*0%*
2012	**N**	30396	25098	5298	14918	15478	4982	12461	12953	17878	5800	3048	3670
	%		83%	17%	49%	51%	16%	41%	43%	59%	19%	10%	12%
	dif from pop				*-2%*	*2%*	*-1%*	*-2%*	*4%*	*4%*	*-3%*	*1%*	*1%*
2013	**N**	27014	22126	4888	13546	13468	4049	10875	12090	16146	5111	2628	3129
	%		82%	18%	50%	50%	15%	40%	45%	60%	19%	10%	12%
	dif from pop				*-1%*	*1%*	*-3%*	*-2%*	*5%*	*3%*	*-4%*	*0%*	*1%*
2014	**N**	29303	24206	5097	14164	15139	4359	12836	12108	16847	5779	2952	3725

	%		83%	17%	48%	52%	15%	44%	41%	57%	20%	10%	13%
	dif from pop				*-3%*	*3%*	*-3%*	*2%*	*2%*	*2%*	*-4%*	*1%*	*1%*
2015	**N**	20443	16630	3813	10155	10288	3540	10024	6879	12123	4215	1884	2221
	%		81%	19%	50%	50%	17%	49%	34%	59%	21%	9%	11%
	dif from pop				*-2%*	*2%*	*-2%*	*5%*	*-2%*	*4%*	*-3%*	*0%*	*-1%*

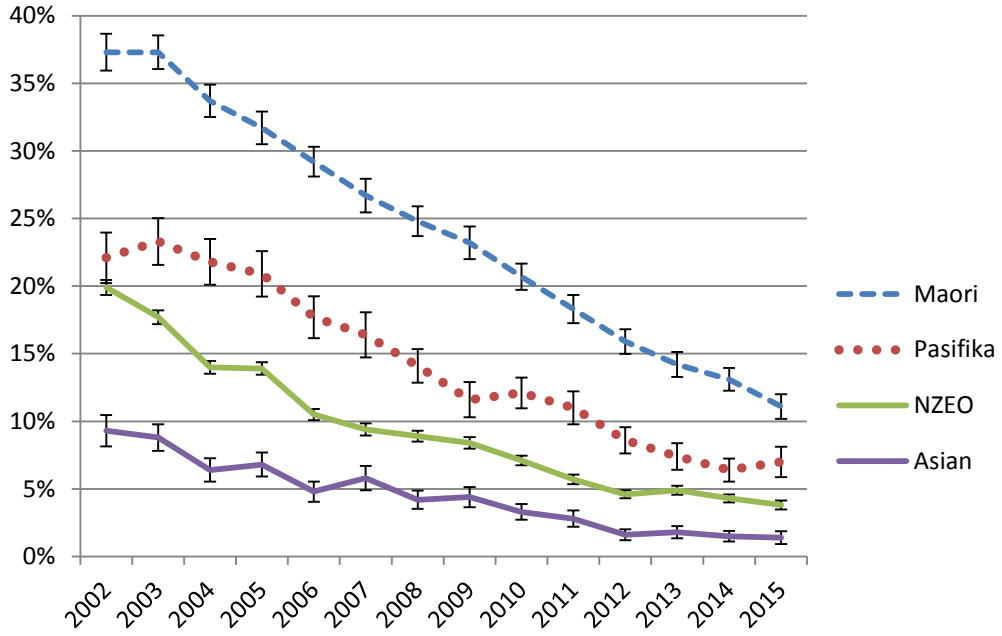
Notes: Population data is based on Ministry of Education (MoE) records which are available online at: https://www.educationcounts.govt.nz/statistics/schooling/student-numbers/6028.

Ethnicity data is not available from MoE prior to 2006, so ethnic comparison of population and sample is not possible 2002-2005.

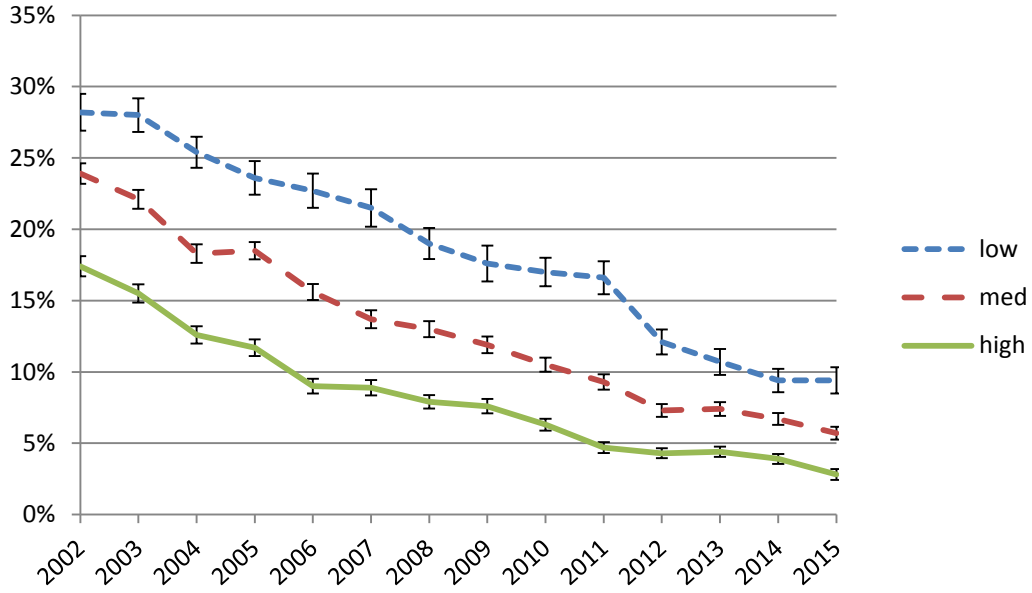
Comparison between sample and population on age of students (14 or 15 years) is not shown since MoE data is measured at the midpoint in the year, whereas survey fieldwork dates differ slightly from year to year, and as a result meaningful comparison is not possible.

Smoking prevalence by ethnicity, school decile and gender

Supplementary Figure S1: Prevalence of regular smoking in 14-15 year olds by prioritised ethnicity, 2002 to 2015



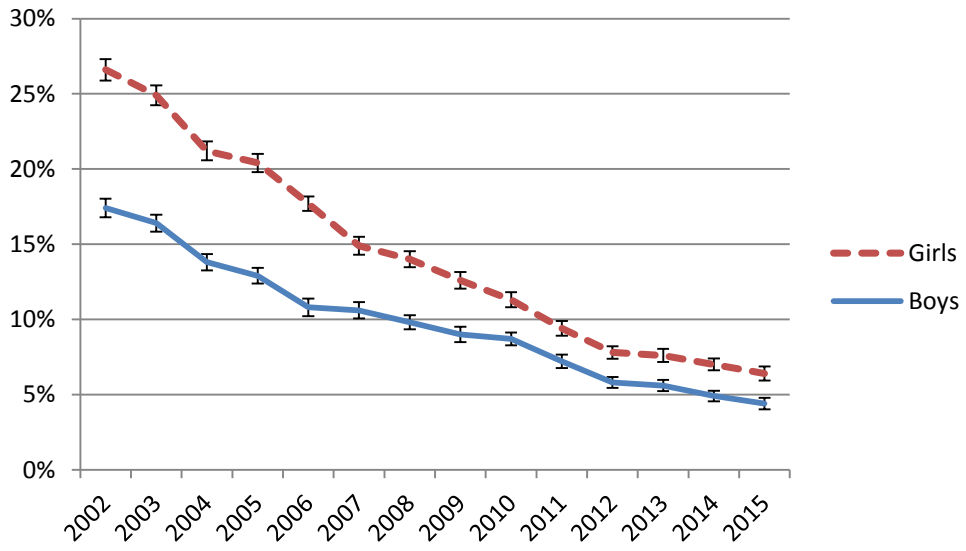
Supplementary Figure S2: Prevalence of regular smoking in 14-15 year olds by school decile, 2002 to 2015



Notes: Low decile = most deprived. High decile = least deprived.

School decile is calculated by the Ministry of Education for purposes of funding allocation, and is a school-level measure of the socioeconomic position of a school's student community. Details of how school decile is calculated are available on the Ministry of Education website: https://www.education.govt.nz/school/running-a-school/resourcing/operational-funding/school-decile-ratings/

Supplementary Figure S3: Prevalence of regular smoking in 14-15 year olds by gender, 2002 to 2015



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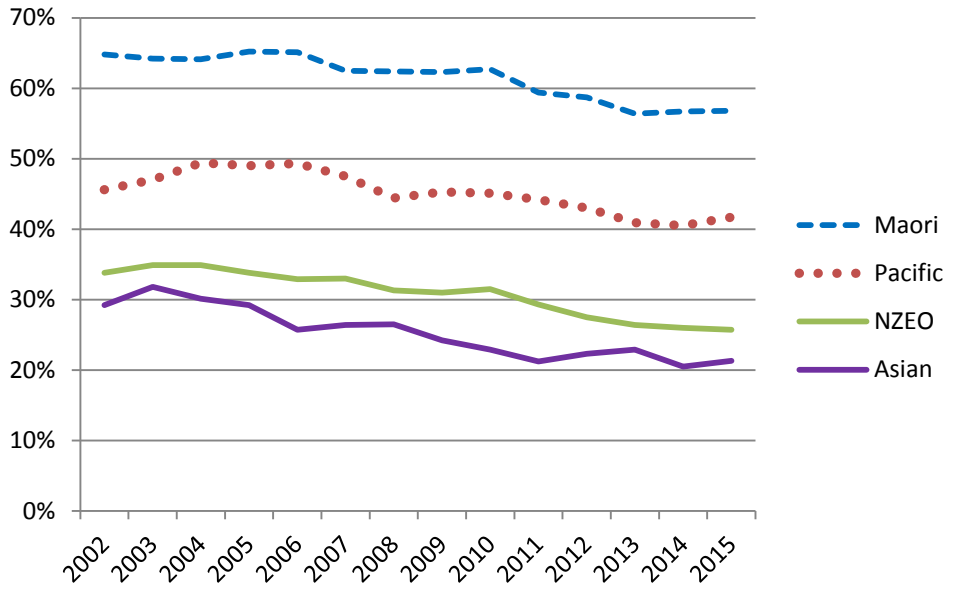
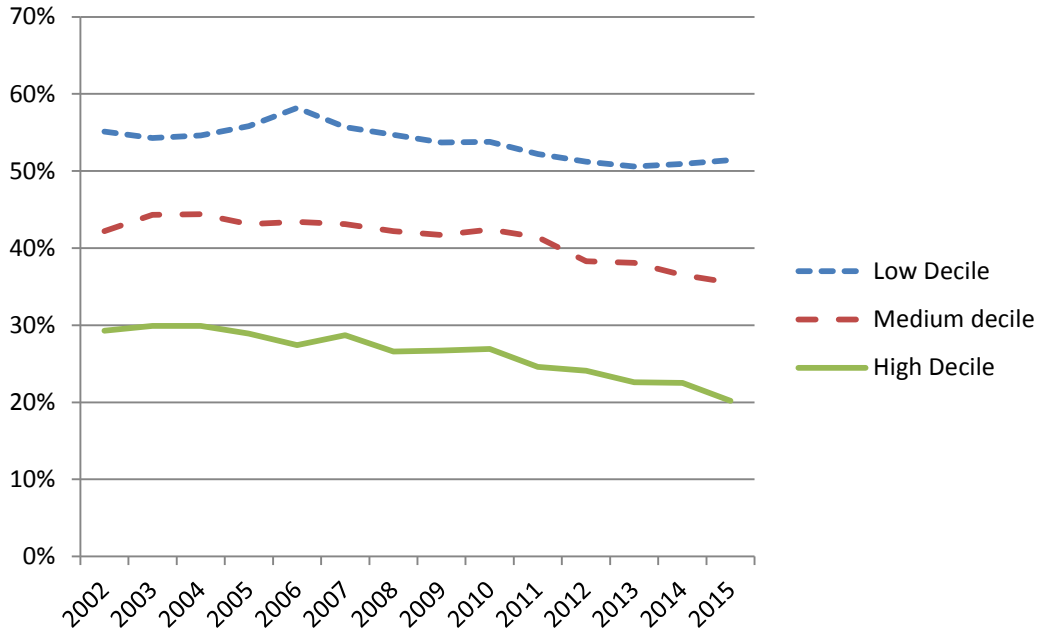
Changes in exposure to risk factors over time, by ethnicity and school decile

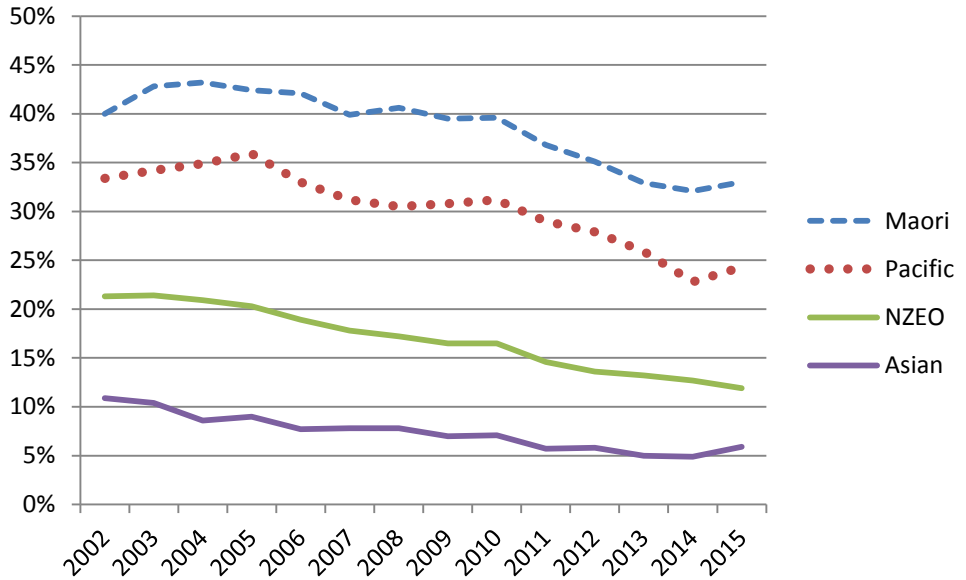
Supplementary Figure S4: Proportion reporting at least one parent smokes by ethnicity, 2002 to 2015



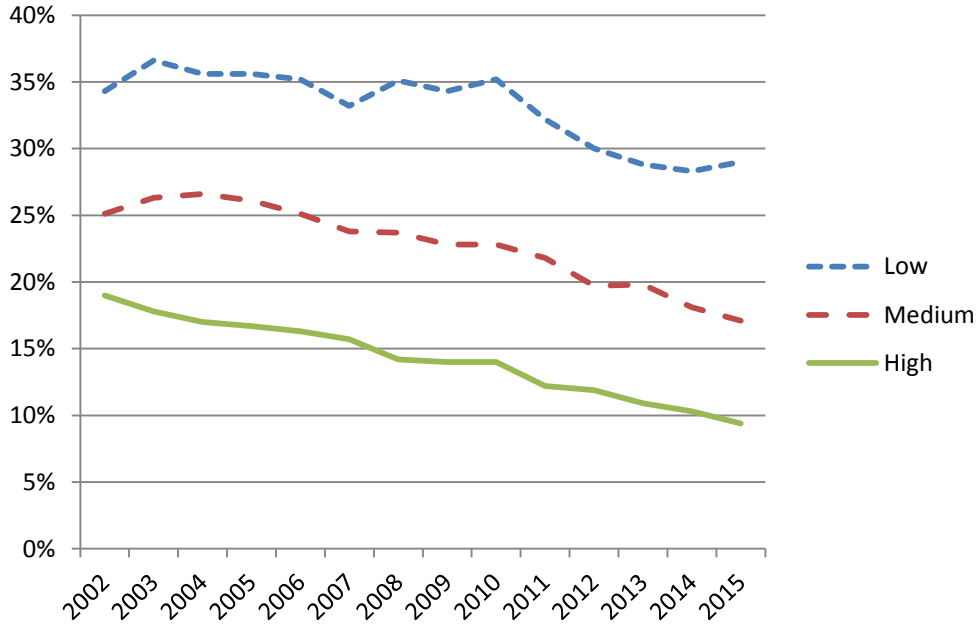
Supplementary Figure S5: Proportion reporting at least one parent smokes by school decile, 2002 to 2015



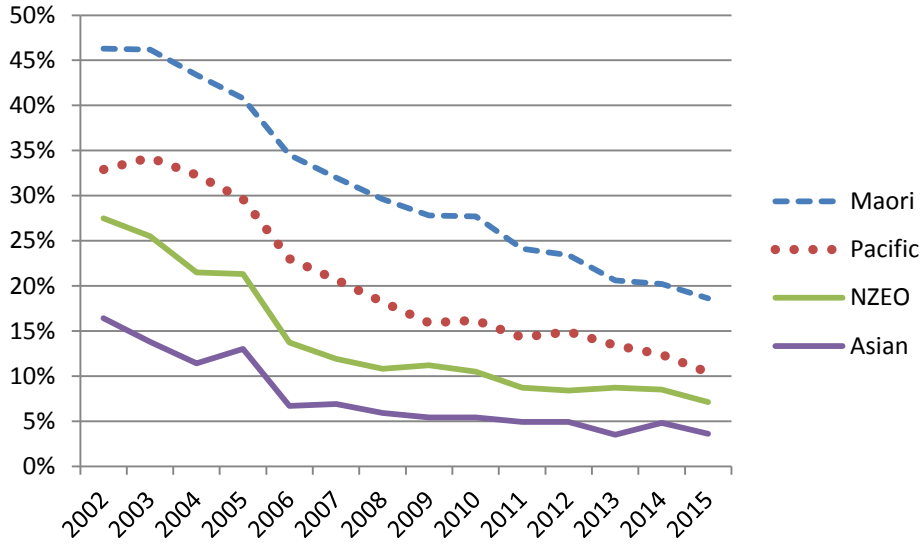
Supplementary Figure S6: Proportion reporting older sibling(s) smoke, by ethnicity, 2002 to 2015.



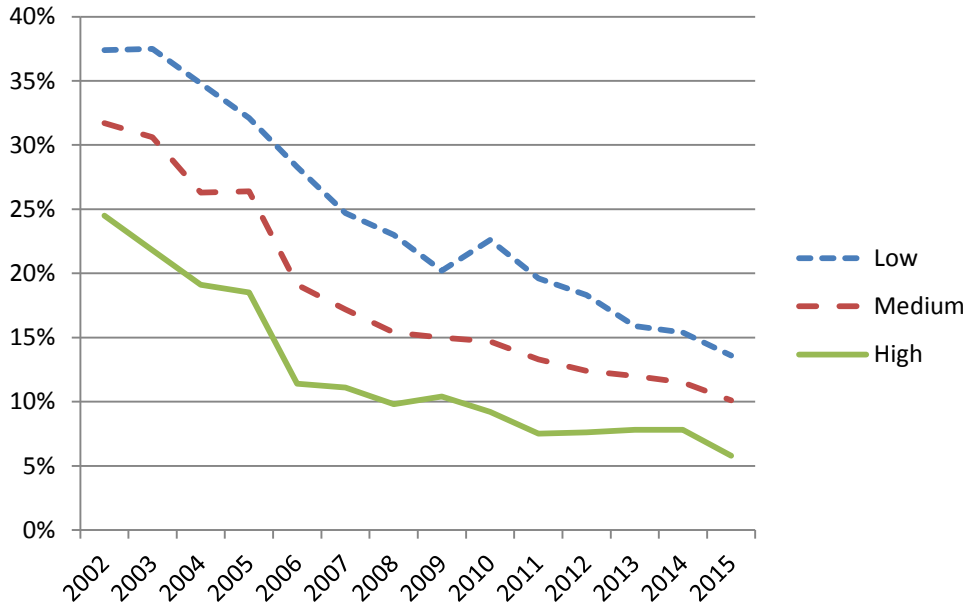
Supplementary Figure S7: Proportion reporting older sibling(s) smoke, by school decile, 2002 to 2015.



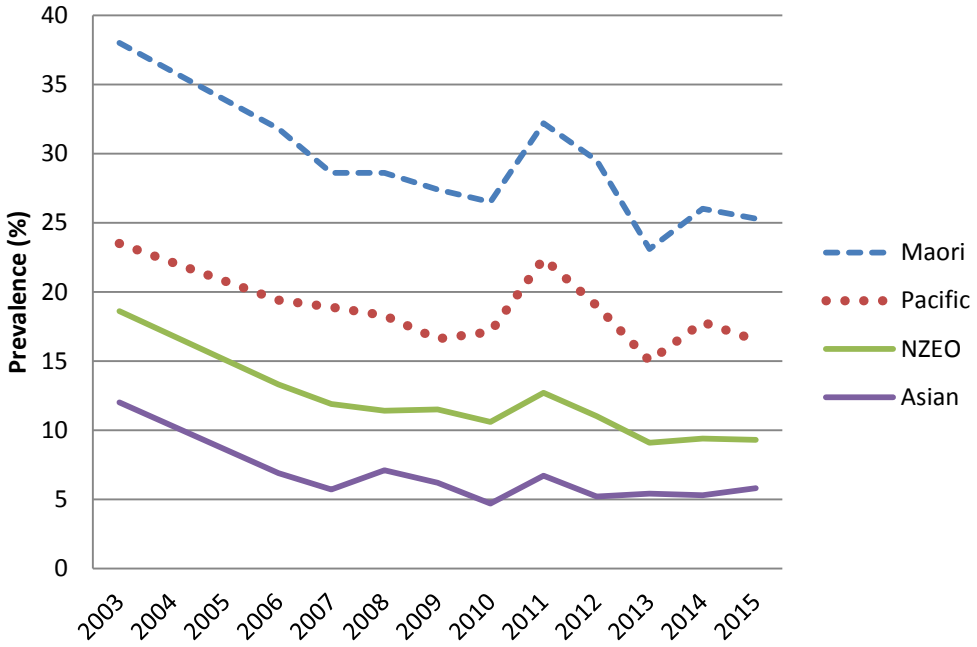
Supplementary Figure S8: Proportion reporting best friend smokes, by ethnicity, 2002 to 2015.



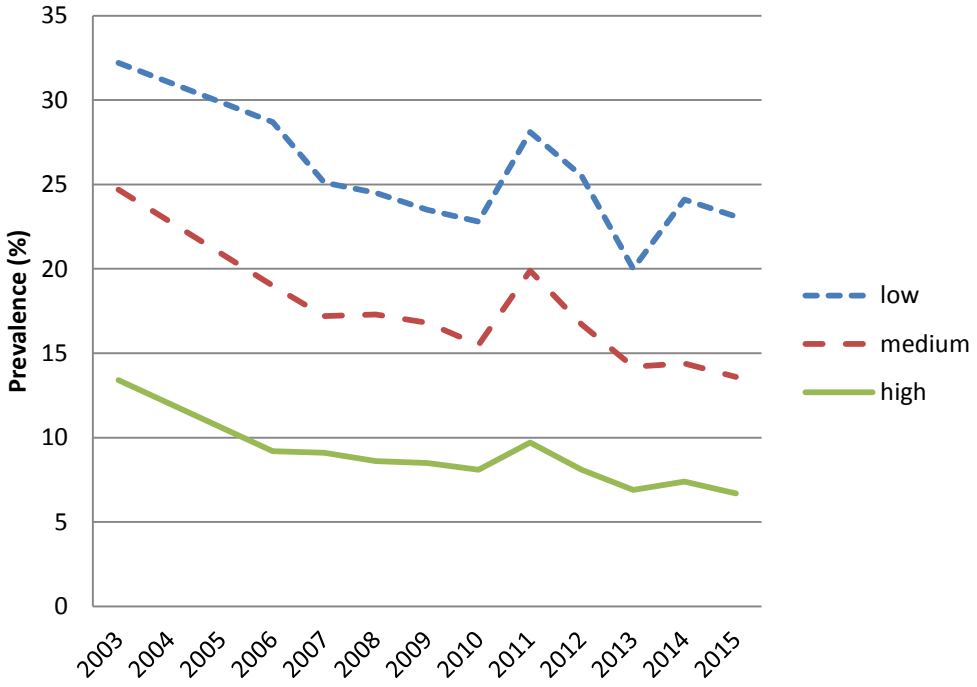
Supplementary Figure S9: Proportion reporting best friend smokes, by school decile, 2002 to 2015.



Supplementary Figure S10: Proportion reporting daily exposure to smoking in the home, by ethnicity, 2002 to 2015.



Supplementary Figure S10: Proportion reporting daily exposure to smoking in the home, by school decile, 2002 to 2015.



Trend analysis for regular smoking in Māori adolescents

To test whether trend analysis results for Māori (indigenous) adolescents differed from those for the adolescent population as a whole, we stratified the sample by ethnicity (Māori/non-Māori) and re-ran our trend analyses on the Māori sub-sample. Table S2 shows ORs for each survey year relative to 2003 (baseline), adjusted for age, gender, and decile (Model 1); then for each named risk factor separately, and finally adjusted for all risk factors together. The final row shows the results for all years combined, with year entered in the model as a continuous variable.

Supplementary Table S2: Trend analyses for Māori sub-sample

Year	Model 1 OR for Year, Partially adjusted	Model 1 + Best Friend	Model 1 + smoke in home	Model 1 + smoke parent4	Model 1 + smoke Sibling	Model 1 + all risk factors
2003	1					
2006	0.73 (0.67, 0.79)	0.89* (0.81, 0.97)	0.68 (0.62, 0.74)	0.70 (0.65, 0.77)	0.72 (0.66, 0.78)	0.80 (0.73, 0.88)
2007	0.64 (0.58, 0.70)	0.78* (0.71, 0.87)	0.61 (0.55, 0.67)	0.62 (0.57, 0.69)	0.63 (0.58, 0.70)	0.73 (0.66, 0.81)
2008	0.57 (0.52, 0.62)	0.75* (0.68, 0.82)	0.55 (0.49, 0.60)	0.56 (0.51, 0.61)	0.56 (0.51, 0.62)	0.69* (0.62, 0.76)
2009	0.52 (0.48, 0.58)	0.70* (0.63,0.78)	0.50 (0.46, 0.56)	0.51 (0.46, 0.56)	0.52 (0.47, 0.57)	0.65* (0.58, 0.72)
2010	0.46 (0.42, 0.50)	0.59* (0.54, 0.65)	0.45 (0.41, 0.49)	0.45 (0.41, 0.49)	0.45 (0.41, 0.49)	0.55* (0.50, 0.61)
2011	0.42 (0.38, 0.46)	0.58* (0.52, 0.64)	0.38 (0.34, 0.41)	0.42 (0.38, 0.46)	0.42 (0.38, 0.46)	0.51* (0.45, 0.56)
2012	0.34 (0.31, 0.38)	0.47* (0.42, 0.52)	0.31 (0.28, 0.34)	0.34 (0.31, 0.38)	0.35 (0.31, 0.38)	0.42* (0.37, 0.46)
2013	0.31 (0.28, 0.34)	0.44* (0.40, 0.49)	0.31 (0.28, 0.34)	0.31 (0.28, 0.34)	0.31 (0.28, 0.35)	0.42* (0.38, 0.47)
2014	0.28 (0.26, 0.31)	0.40* (0.36, 0.45)	0.27 (0.24, 0.30)	0.29 (0.26, 0.32)	0.29 (0.26, 0.32)	0.37* (0.33, 0.42)
2015	0.23 (0.21, 0.26)	0.34* (0.34, 0.39)	0.22 (0.19, 0.25)	0.23 (0.21, 0.26)	0.24 (0.21, 0.27)	0.32* (0.28, 0.36)
Linear trend 2003-2015	0.89 (0.88, 0.89)	0.92* (0.91, 0.92)	0.89 (0.88, 0.89)	0.89 (0.88, 0.90)	0.89 (0.89, 90)	0.91* (0.90, 0.92)

*OR > Model 1 OR ($p < .01$)

STROBE Statement—Checklist of items that should be included in reports of ***cross-sectional studies***

	Item No	Recommendation	Page no.
Title and abstract	1	(*a*) Indicate the study's design with a commonly used term in the title or the abstract	1
		(*b*) Provide in the abstract an informative and balanced summary of what was done and what was found	2
Introduction			
Background/rationale	2	Explain the scientific background and rationale for the investigation being reported	3-6
Objectives	3	State specific objectives, including any prespecified hypotheses	6
Methods			
Study design	4	Present key elements of study design early in the paper	6-7
Setting	5	Describe the setting, locations, and relevant dates, including periods of recruitment, exposure, follow-up, and data collection	6-8
Participants	6	(*a*) Give the eligibility criteria, and the sources and methods of selection of participants	7-8
Variables	7	Clearly define all outcomes, exposures, predictors, potential confounders, and effect modifiers. Give diagnostic criteria, if applicable	9-10
Data sources/ measurement	8*	For each variable of interest, give sources of data and details of methods of assessment (measurement). Describe comparability of assessment methods if there is more than one group	*9-10*
Bias	9	Describe any efforts to address potential sources of bias	NA
Study size	10	Explain how the study size was arrived at	7-8
Quantitative variables	11	Explain how quantitative variables were handled in the analyses. If applicable, describe which groupings were chosen and why	9-10
Statistical methods	12	(*a*) Describe all statistical methods, including those used to control for confounding	10-11
		(*b*) Describe any methods used to examine subgroups and interactions	10-11
		(*c*) Explain how missing data were addressed	7
		(*d*) If applicable, describe analytical methods taking account of sampling strategy	10.
		(*e*) Describe any sensitivity analyses	NA
Results			
Participants	13*	(a) Report numbers of individuals at each stage of study—eg numbers potentially eligible, examined for eligibility, confirmed eligible, included in the study, completing follow-up, and analysed	7-8
		(b) Give reasons for non-participation at each stage	NA
		(c) Consider use of a flow diagram	NA
Descriptive data	14*	(a) Give characteristics of study participants (eg demographic, clinical, social) and information on exposures and potential confounders	8+Supl
		(b) Indicate number of participants with missing data for each variable of interest	NA
Outcome data	15*	Report numbers of outcome events or summary measures	11

Main results	16	(*a*) Give unadjusted estimates and, if applicable, confounder-adjusted estimates and their precision (eg, 95% confidence interval). Make clear which confounders were adjusted for and why they were included	12-15
		(*b*) Report category boundaries when continuous variables were categorized	NA
		(*c*) If relevant, consider translating estimates of relative risk into absolute risk for a meaningful time period	NA
Other analyses	17	Report other analyses done—eg analyses of subgroups and interactions, and sensitivity analyses	11-16 +Suppl
Discussion			
Key results	18	Summarise key results with reference to study objectives	13-14
Limitations	19	Discuss limitations of the study, taking into account sources of potential bias or imprecision. Discuss both direction and magnitude of any potential bias	16-17
Interpretation	20	Give a cautious overall interpretation of results considering objectives, limitations, multiplicity of analyses, results from similar studies, and other relevant evidence	21
Generalisability	21	Discuss the generalisability (external validity) of the study results	20
Other information			
Funding	22	Give the source of funding and the role of the funders for the present study and, if applicable, for the original study on which the present article is based	21

*Give information separately for exposed and unexposed groups.

Note: An Explanation and Elaboration article discusses each checklist item and gives methodological background and published examples of transparent reporting. The STROBE checklist is best used in conjunction with this article (freely available on the Web sites of PLoS Medicine at http://www.plosmedicine.org/, Annals of Internal Medicine at http://www.annals.org/, and Epidemiology at http://www.epidem.com/). Information on the STROBE Initiative is available at www.strobe-statement.org.