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Pathways Between Socioeconomic Status and Adolescent
Cigarette Smoking:
analysis of data from the
Christchurch Health and Development Study

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Abstract

Background

Cigarette smoking is a major preventable cause of morbidity and premature mortality world-wide. It is well known that the risk of becoming a smoker is socially patterned and differentially distributed in the population. In particular, a socioeconomic gradient in smoking has been frequently described such that rates increase with declining socioeconomic status (SES). A consequence of this gradient is that the health burden of smoking falls disproportionately on lower socioeconomic strata, which in turn is reflected in their lower life expectancy. However, little is known about the causal mechanisms that might account for this gradient in smoking.

Smoking initiation typically occurs during adolescence. Thus, antecedents to adolescent smoking have become an important focus in social epidemiology. Such research, while abundant, has reported inconsistent findings on the socioeconomic patterning of adolescent smoking. Of particular interest to our understanding of the generation of socioeconomic inequalities in health is whether the socioeconomic gradient in adult smoking is replicated amongst their offspring, and if so, what might be the underlying mechanisms contributing to this phenomenon. This thesis addresses these questions by drawing on data collected during the course of the Christchurch Health and Development Study (CHDS) – a longitudinal study of a birth cohort of 1265 children born in the urban region of Christchurch, New Zealand, during 1977.

Objectives

The substantive aim of this thesis was to identify, aided by empirical evidence and sociological theory, plausible mechanisms implicated in the potential causal pathway between parental SES and adolescent smoking, developing a hypothetical model to test on data available from the CHDS.

Results

Initial analyses revealed the presence of a statistically significant linear association between parental SES at birth and respondent smoking at age 21. Stratified analyses by gender and ethnicity, and linear logistic regression models to test for interaction effects, revealed that this association was the same for both sexes and ethnic groups.

Six potential pathways were identified from the literature: parental smoking, parental attitudes to smoking, early smoking experimentation, conduct disorder, educational achievement, and affiliations with smoking peers. After fitting a series of linear logistic regression models, only three were found to mediate the association between parental SES and adult offspring smoking. These were: parental smoking, educational achievement and affiliations with smoking peers. This pattern was replicated and confirmed with a further exercise using log-linear modelling. A log-linear model fitted to the data revealed that the association between parental SES and young adult smoking was explained by three pathways: directly by way of parental smoking and educational achievement, and indirectly via the effect of parental smoking on affiliations with smoking peers.

Conclusion

This analysis of the CHDS data highlights how parental smoking and educational achievement, and to a lesser extent, affiliations with smoking peers, are potential points for intervention in reducing the socioeconomic gradient in young adult smoking. However, the association of these causal mechanisms with parental SES at birth suggests that fundamental socioeconomic differences and inequality are important and may hold promise in addressing health behaviours associated with SES.

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Acknowledgement concerning the data analysis

I would like to note, that while all the statistical models presented in the data analysis section (Chapter 5) were my own work and run on SAS by myself - both Professor David Fergusson and John Horwood provided expert advice on how to select the models presented in this thesis from the numerous models that I actually fitted to the data (and not presented here). I would also like to acknowledge John Horwoods' input in writing the CATMOD procedure for SAS used in the log-linear modelling exercise.

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Chapter 1

Background, aims and public health significance

1.1 Background

As early as 1900, increases in cancer of the lung had been noted in official statistics. Yet, studies of the health consequences of smoking did not appear in the scientific literature until the 1930s, by which time clear trends in mortality and disease incidence had become evident (Rabin and Sugarman 1993; U.S.DHEW 1964). Firm evidence of the magnitude of the health risks associated with tobacco smoking was not widely recognised until the publication of the first report of the British Royal College of Physicians of London; ‘Smoking and Health’ in 1962, and the U.S. Surgeon General’s Report (of the same name), in 1964 (Rabin and Sugarman 1993; Taylor 1984). In the 1964 report it was stated unequivocally that “cigarette smoking is a health hazard of sufficient importance in the United States to warrant appropriate remedial action” (U.S.DHEW 1964: 6). While a number of other associations with smoking and disease were also reported, the causal link between tobacco use and lung cancer in men (and the suspected association with lung cancer in women), was empirically established. The strength of the evidence of the health consequences of tobacco smoking accumulated with each successive report. By 1982, the fourteenth U.S. Surgeon General’s Report concluded, that cigarette smoking was the “chief, single, avoidable cause of death in our society, and the most important public health issue of our time” (U.S.DHHS 1982: xi).

The dissemination of this information to the public has been an important element of health promotion strategies since the 1964 Surgeon General’s Report (Gusfield 1993). Over this period the prevalence of cigarette smoking has declined in most developed countries (Graham 1993; Tyas and Pederson 1998). This has been attributed to the impact on smoking initiation and cessation of a number of factors such as; increased public awareness of smoking as a major health hazard, legislative and policy changes (such as those which have limited access to tobacco and reduced the number of places where smoking is permitted), and a range of individualised smoking cessation programmes (Gusfield 1993). Despite this decline, and the overwhelming evidence from more than 70,000 scientific articles since 1950 that cigarette smoking is an important cause of morbidity and mortality world-wide, cigarette smoking remains a major public health issue (WHO 1999). In developed countries where smoking has been well-established for several decades, about 90% of lung cancer, 15-20% of other cancers, 75% of chronic bronchitis and emphysema, and 25% of deaths from cardiovascular disease at ages 35-69 years, have been attributed to tobacco smoking (WHO 1999: 66). In New Zealand, it is estimated that more than 4500 smoking related deaths occur each year (Cancer Society of New Zealand 1996; Peto *et al.* 1994), and that cigarette smoking is responsible for approximately 17%, or one in six, of all deaths (Cancer Society of New Zealand 1996: 2).

Demographic data from developed countries, including New Zealand, has also revealed characteristics of smokers themselves. Smoking, predominantly a male habit in the 1960s (and earlier), is now almost equally common for both sexes, although recent evidence suggests that smoking initiation among young women is now

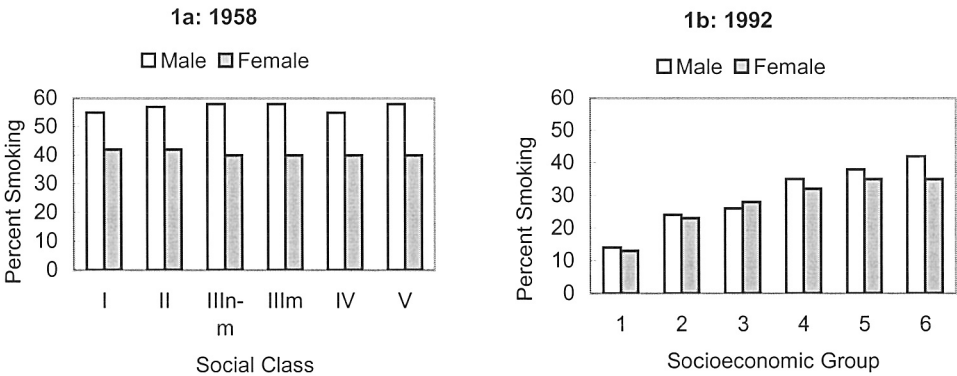
higher than among young men (U.S.DHHS 1989). The picture on the ethnic patterning of smoking is somewhat more complex, and varies greatly between countries. The most striking trend, however, is the emergence over the past thirty or so years of a socioeconomic difference in smoking prevalence.

1.1.1 Socioeconomic patterning of smoking

Among the distinct demographic characteristics of smokers in New Zealand, U.K., U.S., and Australia, low socioeconomic status (SES) is the most commonly observed predictor of the typical smoker (Graham 1993; Pierce 1989; Tyas and Pederson 1998; Whitlock *et al.* 1997). Research from New Zealand and overseas has shown, for instance, that smoking is far more prevalent, tobacco consumption heavier per person and quit rates lower among lower socioeconomic groups (Graham and Der 1999; NHC 1998; NZHIS 1995; Pierce 1989). Furthermore, research from a number of industrialised nations has clearly illustrated a socioeconomic *gradient* in smoking behaviour, with an increased prevalence of smoking for each decrease in socioeconomic status (Benzeval *et al.* 1995; Feinstein 1993; Macintyre 1986).

The socioeconomic gradient in smoking is, however, a relatively recent phenomenon. British data on the social class patterns of smoking (see Figure 1) illustrates how in 1958, before the association between smoking and disease was publicised, smokers were just as likely to come from the upper social classes as the lower and middle social classes. By 1992 a distinctive social class gradient in smoking had emerged (Townsend 1995).

Figure 1: Percentage of adults smoking cigarettes by social class: UK: 1958 and 1992



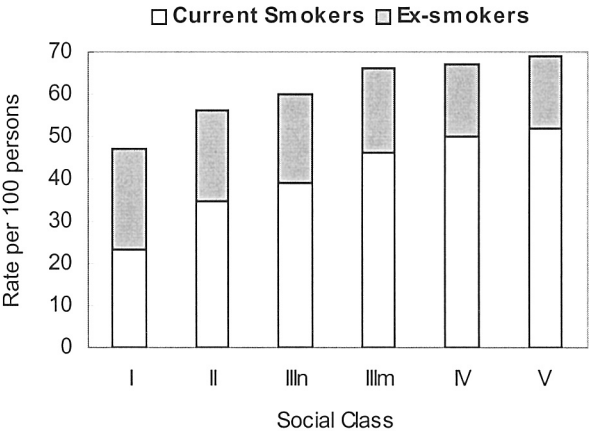
Source: Townsend (1995: 84)

The graph on the left (Figure 1a) depicts smoking prevalence in 1958 by gender for social class (group I is the highest and group V the lowest socioeconomic group). This clearly shows that the proportions of male and female smokers in each of the six social classes were markedly similar, with roughly 40% of females and 55% of males from each social class classified as smokers. The graph on the right (Figure 1b), depicting smoking prevalence by socioeconomic group in 1992, illustrates the now characteristic socioeconomic gradient in smoking prevalence for both males and females, with an increasing proportion of smokers with each decrease in socioeconomic group. The prevalence of smoking in the lowest socioeconomic group is more than twice that of

the highest socioeconomic group. Figure 1 also illustrates a narrowing of the difference in smoking prevalence between men and women in 1992 in comparison with 1958.

Data from the U.S. also reveals the same pattern in the years since 1964, showing greater rates of smoking cessation and reduced initiation among higher socioeconomic groups (Rabin and Sugarman 1993). Unfortunately, the earliest available New Zealand data on smoking prevalence by socioeconomic group is that recorded in the 1976 New Zealand Census of Population and Dwellings. Thus, it is not known if the prevalence of smoking was similar across the socioeconomic strata in New Zealand before public awareness of the adverse health effects of smoking. The socioeconomic patterning of smoking (among males aged 15-64) was nonetheless, evident in New Zealand at the time of the 1976 Census (see ‘current smokers’ Figure 2).

Figure 2: Age-standardised New Zealand adult male smoking rates by social class: 1976

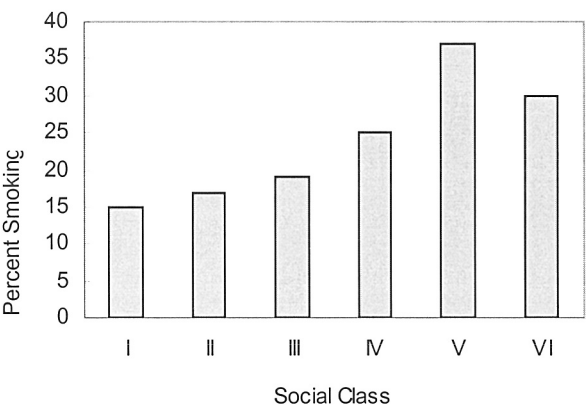


Source: Pearce *et al.* (1985:12)

It is apparent from Figure 2, where as many as 50% of social class I (the highest) compared to only 25% of social class VI (the lowest social class) were classified as former smokers, that smoking *cessation* was related to social class. This suggests that the socioeconomic gradient in smoking is also a relatively recent phenomenon in New Zealand, as it is in other industrialised nations (Pearce *et al.* 1985).

In the twenty years since 1976 the overall prevalence of smoking in New Zealand has declined from 36%, to 24% (Borman *et al.* 1999: 461). Yet, the socioeconomic gradient in smoking is still evident. Figure 3 illustrates the prevalence of smoking as recorded by the 1996 Census for those in the full-time workforce aged 21-69 by Elley-Irving social class (Davis *et al.* 1997).

Figure 3: Smoking prevalence in 1996 by Elley Irving social class



Source: Davis *et al.* (1997: 63)

The prevalence of smoking among the lowest socioeconomic group VI, at around 30%, is twice that of the highest socioeconomic group I, at 15%. However, disturbing an otherwise linear trend of increases in smoking for each decrease in SES, is the anomalous drop in smoking among the lowest socioeconomic group. This may be due to a higher proportion of unemployed people in 1996 in comparison with 1976, who are more likely to come from lower socioeconomic groups (SNZ 1996), and who are typically *excluded* from socioeconomic analyses which rely on occupational data. This is plausible, given that in 1996, the prevalence of smoking among the unemployed and ‘actively seeking work’ was 41% compared with 25% for full-time workers (Borman *et al.* 1999). Furthermore, Maori, for whom rates of smoking are considerably higher than the general population (SNZ 1998), are also over-represented among those who are unemployed (Te Puni Kokiri 1998).

Whilst the socioeconomic gradient in cigarette smoking has been frequently described internationally and in New Zealand, and cigarette smoking is known to be responsible for much of the socioeconomic gradient in morbidity and mortality from smoking related illnesses (Graham 1993; NHC 1998; NZHIS 1995a; Pearce *et al.* 1993; Whitlock *et al.* 1997), very little is understood about why smoking is patterned in this way (Stronks *et al.* 1997). In other words, *what is it about SES or social class that causes not only the higher prevalence of smoking among lower socioeconomic groups, but also the socioeconomic gradient in smoking?* This is the essential research question that underpins this thesis.

1.1.2 Socioeconomic patterning of adolescent smoking

Given that habitual cigarette smoking is typically established during adolescence (Glendinning *et al.* 1994; Townsend 1995; U.S.DHHS 1994), a substantial body of literature has necessarily focused on the correlates and predictors of adolescent smoking (Conrad *et al.* 1992; Fergusson and Horwood 1995; Fergusson *et al.* 1995; Moncher *et al.* 1991). Apart from SES, the most common etiological factors identified in association with adolescent smoking include; parental and peer smoking behaviour (and attitudes), educational attainment, early experimentation with smoking, and rebelliousness and conduct problems (Chassin *et al.* 1984; Conrad *et al.* 1992; Fergusson and Horwood 1995; Fergusson *et al.* 1995; Moncher *et al.* 1991; Stanton and Silva 1991; Stanton *et al.* 1989). While there appears to be consistent empirical support for the findings in that patterns of early smoking experimentation and adolescent peer affiliations are major predictors of regular cigarette smoking among adolescents and young adults (Conrad *et al.* 1992; Moncher *et al.* 1991; Stanton and Silva 1991), evidence on the influence of other etiological factors remains inconsistent (Conrad *et al.* 1992; Moncher *et al.* 1991; Tyas and Pederson 1998).

Inconsistencies also characterise the socioeconomic patterning of *adolescent* smoking, with some studies reporting a strong association between SES and adolescent smoking, others reporting only a weak association, and a few reporting no association at all (such inconsistencies have been noted by: Conrad *et al.* 1992; Glendinning *et al.* 1994; Tyas and Pederson 1998). Glendinning *et al.* (1994), for example, found no direct association between parental social class and adolescent smoking behaviour, but marked differences relating to adolescents' own (achieved) SES position. This contradicts the findings of a number of other studies which have demonstrated an inverse association between SES and adolescent smoking behaviour, or at the very least, a higher prevalence of smoking among lower socioeconomic youth (Conrad *et al.* 1992; Stanton *et al.* 1989; Tyas and Pederson 1998). Such inconsistencies are not surprising given the wide variations in: research design (in particular between cross-sectional and longitudinal studies), the covariates investigated, the measurement of variables (especially that of SES), and in analyses.

There are likely to be a number of explanations for these apparent discrepancies. For instance, a socioeconomic differential in smoking may not occur until early adulthood, due, as some studies have found, to increased quitting by young smokers from higher socioeconomic groups (Reid *et al.* 1995). Yet, while research on adolescent smoking has contributed to our understanding of the factors that predispose an individual adolescent to initiate smoking, it cannot, nor was it intended to explain the possible social patterning of adolescent smoking by SES. It is far from clear whether such predictors of individual smoking are associated with, or directly or indirectly (via some other pathway) linked to SES.

1.2 Aims

The principal aim of this thesis was to explore pathways between parental SES and adolescent smoking, with a view to providing some insight into the causes of the socioeconomic gradient in adult smoking behaviour. This has been achieved by undertaking an analysis of existing data collected from a cohort of 1011 individuals born in the Christchurch urban region in 1977. Data have been collected from these individuals annually to the age of 16, after which further collections were carried out at 18 and 21. This longitudinal study, known originally as the Christchurch Child Development Study and subsequently renamed as the Christchurch Health and Development Study (CHDS), has collected an extensive range of data from multiple sources. These included direct assessment, as well as parent and respondent interviews, teacher reports, and medical and police records (Fergusson *et al.* 1989). This data constitutes one of the richest sources of information available in New Zealand for examining the dynamics of ‘growing up’ in New Zealand including the relationship between parental SES and adolescent smoking. The longitudinal nature of this data is ideally suited to an examination of the effects of parental SES (as measured at birth) on adolescent smoking through a variety of potential mechanisms (or intervening variables). Such mechanisms have been identified from a review of the literature on adolescent smoking and located within a plausible theoretical framework and (hypothetical) model of likely pathways between parental SES and adolescent smoking. This model is then empirically tested with data available from the CHDS, using appropriate statistical methods. The principal aim of this thesis was to determine the significant pathways between parental SES and adolescent smoking. To this end the thesis will proceed by:

1. Determining the socioeconomic patterning of adolescent smoking by parental SES.
2. Assessing the socioeconomic patterning of adolescent smoking by parental SES for key sub groups, specifically; gender and ethnicity.
3. Identifying the relative influence and temporal sequencing of key intervening variables, for example; parental smoking, affiliations with smoking peers, educational attainment, and those otherwise identified in the path model, on the association between parental SES and adolescent smoking.

1.3 Public health significance

1.3.1 Patterns of adolescent smoking in New Zealand

There is a paucity of reliable data on the prevalence of adolescent smoking in New Zealand. Two sources of data in particular are commonly used to gauge changes in patterns of adolescent smoking over time; surveys of fourth form secondary school students, and the New Zealand Census of Population and Dwellings. Both of these sources are problematic. While secondary school surveys have provided data on the prevalence of smoking among those aged 14 and 15, many more adolescents begin smoking at a later age than this. For example, the Dunedin Health and Development Study (a longitudinal study which has collected data annually on a cohort of over a thousand people born in Dunedin the 1970s), recorded a two-fold increase in smoking prevalence from age 15 to 18 (McGee and Stanton 1993). Data from the census, on the other hand, records only the smoking prevalence of those aged 15 and over, and thus excludes younger adolescents. Census data on the prevalence of adolescent smoking may also be biased by the potential under-reporting of smoking by adolescents, given that

they may be subjected to parental surveillance when completing the census questionnaire. For these reasons, the focus here is on the smoking patterns of those aged 20-24 as reported in the census. While this group is not strictly the adolescent population, it does capture all those who began smoking in adolescence and who continue to smoke.

Data from the New Zealand censuses show that, for those aged 20-24, there has been a decline in smoking prevalence from 40% in 1976 to 32% in 1996 (SNZ 1998: 90). The 1996 Census further reveals significant variation in smoking among adolescents. For instance, in 1996, higher rates of smoking were observed among young females aged 20-24 (33%) than young males (31%), and the rates for young Maori, aged 15-24, were almost twice that (42%) of non-Maori at 23% (SNZ 1998: 90). Much of the ethnic difference in smoking prevalence was found to be due to the smoking behaviour of young Maori women, with 54% recorded as smokers (SNZ 1998: 89). Changes in the definition of ethnicity between the censuses may have had some impact on the figures quoted above. However, it is not known what effect this could have had on the reported differential in smoking prevalence between Maori and non-Maori.

Data on the socioeconomic pattern of smoking among adolescents in New Zealand are not plentiful, but where parental SES has been measured the findings have been consistent. Silva (1996), Stanton and Silva (1991), Ree (1986), Mitchell (1983), and Stanhope (1975) have all found an association between parental SES and adolescent smoking. A comparison of New Zealand high school students' smoking behaviours in 1968 and 1981 by Michell (1983) has also revealed that while a socioeconomic pattern in adolescent smoking was evident in 1981 (at least among non-Maori adolescents), it was not apparent in 1968. This suggests that the SES gradient in adolescent smoking, as in the case of adults, is a relatively recent phenomenon.

The studies by both Mitchell (1983) and Ree (1986) reported an association between parental SES and adolescent smoking for European but not Maori. It should be noted that it has been difficult to determine the association between Maori ethnicity and adolescent smoking, because of the concentration of Maori in lower socioeconomic strata (Chapple 2000; Te Puni Kokiri 1998) and because of inadequate sample sizes of Maori in many surveys. Mitchell (1983), for example, noted that a socioeconomic pattern in smoking was not reported for Maori due to their under-representation amongst the highest Elley-Irving socioeconomic groups.

1.3.2 Socioeconomic health inequalities

Tobacco is highly addictive. One U.S. study estimated that the median age of cessation for those who began smoking in adolescence was thirty-three years of age for men and thirty-seven years of age for women (Pierce and Gilpin 1996). The effects of smoking on health are also highly adverse. Given these facts the current demographic differences in adolescent smoking will potentially translate in to gender, ethnic and socioeconomic health inequalities in later life. Smoking, on its own, may not be responsible for the differentials in morbidity and mortality from smoking-related illnesses, since a number of other social, economic and environmental factors also contribute to differences in health outcomes associated with smoking (Davey-Smith and Shipley 1991). Nevertheless, it is well recognised as a *critical* and *preventable* cause of much of the poorer health and premature death suffered disproportionately by lower socioeconomic groups (NHC 1998; Zhang *et al.* 2001).

Are such health inequalities considered unfair or unjust? This depends on whether such inequalities are seen to be a direct result of individual choice or whether factors beyond the individual's control play an important role (Woodward and Kawachi 1998). Arguably, cigarette smoking can be likened to any other hazardous leisure pursuits, such as skiing, sky-diving or motor vehicle racing, in that individuals who engage in such behaviours do so with the understanding of the risks they run. Adverse health outcomes resulting from such leisure pursuits are unlikely to be considered as unfair or unreasonable. There are two features of smoking, however, that substantially affect the degree of choice involved. Firstly, unlike a number of other behaviours, tobacco smoking is pharmacologically addictive (U.S.DHHS 1994). The evidence is that the great majority of smokers, are aware of the health dangers of smoking and have experienced great difficulty in giving up, and wish they had never started (Rabin and Sugarman 1993). This is testimony to the diminished role of choice experienced by those who continue smoking. Secondly, smoking initiation overwhelmingly occurs during adolescence, a time at which individuals lack the maturity to appreciate fully, not only the long-term consequences of smoking, but also the addictive properties of tobacco. It is doubtful that a model of informed choice can apply in such circumstances.

Furthermore, the individual decision to initiate or quit smoking does not occur in a social vacuum. There are a wide variety of environmental, social and cultural contexts which “. . . exert important influences on both the processes of choice and the types of behavioural options which are available, and indeed, appropriate” (Lynch *et al.* 1997: 810). In fact it has been argued that the differential distribution of health behaviours across socioeconomic groups should be viewed from a life-course perspective “. . . as the cumulative responses of different classes of people to conditions imposed by social structure” (Lynch *et al.* 1997: 810). Health inequalities resulting from cigarette smoking are, in this sense, also unjust since the ability to exercise choice about smoking initiation and cessation is structurally and culturally constrained by factors such as age, gender, ethnicity and SES.

1.3.3 Smoking intervention and prevention programmes

For public health efforts to successfully reduce the prevalence of adolescent smoking further, they need to be informed by a much richer understanding of the pathways between SES and adolescent smoking. Part of the reason why health promotion efforts have been unsuccessful in lowering the smoking prevalence among adolescents, particularly among adolescents from lower socioeconomic backgrounds, is that, despite the abundant research on determinants of smoking, we do not understand why those from lower socioeconomic backgrounds take up smoking at a greater rate than their higher SES counterparts. An extensive array of research has identified peer smoking, adolescent educational achievement, conduct disorder, and risk taking/rebelliousness as possible ‘causes’ of adolescent smoking, but as so little of it has been of longitudinal design, the ability to explore factors such as the role-modelling effect of parental smoking on their adolescent offspring has been severely limited.

Clearly, the most straightforward explanation of the link between parental SES and adolescent smoking is one in which parental smoking leads to adolescent smoking. Thus, adolescents from lower socioeconomic backgrounds smoke because their parents do. If this explanation of the socioeconomic gradient in adolescent smoking were

sufficient, then the success of health promotion efforts would depend upon the effectiveness of interventions to reduce parental smoking, or the targeting of mechanisms linking parental smoking to adolescent smoking. Access to cigarettes, attitudes toward smoking, and knowledge of the health effects, are all potential mechanisms through which parental smoking may influence adolescent smoking. However, it is likely that there are other socioeconomic factors (or correlates) which predispose adolescents from lower SES groups to smoke, quite independently of parental smoking. Thus, it is plausible that the higher prevalence of smoking among adolescents from lower SES groups is partly due to structural factors. This implies that targeting smoking prevention programmes to those from lower SES groups may have little effect if social or economic policies to improve the socioeconomic position of lower SES groups are not also in place.

If, however, parental smoking has little or no effect on adolescent smoking, and it was found, for example, that peer smoking behaviour had a substantial effect on adolescent smoking, then health promotion efforts would have greater efficacy by focusing on peer influence. Potential mechanisms that could be seen as mediating between peer and adolescent smoking are factors such as educational achievement, self-esteem or self-efficacy, and refusal skills or, more broadly, opportunities for socialising in non-smoking environments, or the accessibility of alternative activities that confer adult status. It is also possible, that, rather than peers influencing the individual, the individual actively seeks out peers with similar behavioural orientations – a phenomenon known as differential association. If differential association is in fact more critical than peer influence, then public health efforts which attempt to increase adolescent self-esteem or teach ‘refusal skills’ to adolescents will be met with limited success. In reality, it is probable that both peer selection and peer influence are operating and that they may be mutually reinforcing, making smoking initiation unlikely among non-smoking peer groups, and cessation difficult among peer groups where smoking is firmly established. Alternatively, it may be that both parental and peer smoking contribute to the socioeconomic patterning of adolescent smoking, in which case successful interventions would be more likely to result from addressing both mechanisms.

Another issue also examined in this thesis is whether the association between SES and adolescent smoking is similar or different for males and females and for Maori and non-Maori. Particularly salient in New Zealand, is the issue of whether the higher prevalence of smoking among Maori is due to their over-representation among lower SES groups or due to cultural mechanisms associated with ethnicity. If the higher rates of smoking are found to be due to ethnic or cultural factors, this would indicate that programmes tailored for Maori may have the most benefit, whereas if the higher prevalence of smoking is found to be due to the concentration of Maori among lower socioeconomic groups, then broader social and economic policies which improve the socioeconomic position of Maori may be indicated. Similarly, if the association between SES and adolescent smoking differs for males and females, then it may indicate the need for intervention programmes to be sensitive to gender dimensions in the smoking acquisition process, as well as those that are primarily socioeconomic in nature.

These scenarios – the possible influence of parental and peer smoking (as explanations of the parental SES and adolescent smoking association), and the importance of examining gender and ethnic dimensions in this association – are only a few examples of the potential value to public health of asking the research question that motivated this thesis. In particular, it is argued that health promotion efforts in the areas of smoking intervention and prevention will be better equipped, and more effective in addressing the higher rates of smoking among

lower SES adolescents, if informed by an understanding of the mechanisms discussed here. Once public health efforts, armed with this information, are successful in reducing socioeconomic differences in smoking prevalence, we will, in time, see a substantial reduction in socioeconomic inequalities in health outcomes arising from tobacco smoking.

Chapter 2

Determinants of smoking: potential pathways between parental SES and adolescent smoking

2.1 Historical Overview

Research on the ‘determinants’ of smoking was in its infancy at the time of the publication of the U.S. Surgeon General’s Report in 1964. Included in the 1964 Report were two chapters, one on the ‘Morphological Constitution of Smokers’ and one on the ‘Psychosocial Aspects of Smoking’ illustrating the very first attempts to understand the ‘determinants’ of smoking (U.S.DHEW 1964). These early attempts focused on factors such as; body weight and shape, a range of demographic factors (age, sex, race, SES, religion, and marital status), personality factors (particularly extroversion and neuroticism), and a number of psychosocial and social/environmental variables (for example, parental smoking and peer cultures). The predominant theoretical explanation for tobacco smoking at the time was informed by psychoanalytic theory, in which psychoanalysts suggested that smoking, like thumb-sucking, “. . . was a regressive oral activity related to the infant’s pleasure at his mother’s breast” (U.S.DHEW 1964: 367). Reported associations between adult smoking and male childhood thumb-sucking, number of months breast feeding, together with the increased food consumption, weight gains and use of chewing gum upon cessation of smoking, provided the (then) necessary evidence to support the recommendation that the “oral hypothesis warrants further investigation” (U.S.DHEW 1964: 368). The 1964 report also mentioned, albeit briefly, a hypothesis linking smoking to social class norms. However, while acknowledging that smoking may in part be determined by such social factors they concluded that “Since man [*sic*] is not a passive target of such forces but an active participant, no possible explanation can omit consideration of the way in which he [*sic*] reacts to and, in turn, creates such forces . . .” (U.S.DHEW 1964: 365). Nevertheless, while a number of potential determinants and theoretical explanations of smoking were explored, the scientific evidence at the time was largely inconclusive, and only suggestive of possible associations (U.S.DHEW 1964). Thus, the broad and non-specific conclusion: “. . . smoking – its beginning, habituation, and occasional discontinuation – is to a large extent psychologically and socially determined” (U.S.DHEW 1964: 377).

With each advance in the evidence of the adverse health effects of smoking came an expanding body of research on the determinants of smoking. Thus, in the publication of the twelfth Surgeon General’s Report in 1979, nine of twenty-three chapters were devoted to determinants of smoking and smoking cessation (U.S.DHHS 1989). Strikingly, many of the determinants of smoking identified in the earliest 1964 report have featured strongly in the literature since. In particular, associations between smoking and educational achievement, SES, rebelliousness, stress and self-esteem, all suggested in the initial report, have been explored in numerous studies. A voluminous body of literature from research investigating other ‘risk’ factors for adolescent smoking has similarly accumulated.

The literature is also characterised by a move away from cross-sectional study designs and correlates of adolescent smoking, toward prospective studies that seek to identify ‘predictors’ or ‘risk factors’ for adolescent smoking. Advances in statistical analysis have paved the way for relatively recent explorations into the causal mechanisms or pathways between various known ‘risk factors’ and adolescent smoking. The current picture, broadly speaking, is one of multiple influences on adolescent smoking in which pharmacological, social, cultural, structural, environmental, and individual personality variables interact differently according to SES, gender, ethnic group, age, and stage in the smoking acquisition process (U.S.DHHS 1989).

As the intention of this thesis was to explore the association between parental SES and adolescent smoking, rather than to identify all possible determinants of adolescent smoking per se, this literature review is limited to considering only those factors which can be plausibly identified as potential pathways between parental SES and adolescent smoking. The review revealed that there were six factors in particular that can be considered critical ‘determinants’ of adolescent tobacco smoking, while at the same time being theoretically and empirically viable mechanisms linking parental SES to adolescent smoking. These are: parental smoking, parental attitudes towards and knowledge of the health effects of smoking, early smoking experimentation, deviant and risk-taking behaviours, affiliations with smoking peers, and adolescent educational achievement. However, before considering further, the evidence for each of these factors as determinants of adolescent smoking and as potential causal pathways, it is important to first examine the role of two additional factors which, although *not directly implicated as mechanisms in the causal pathway* between parental SES and adolescent smoking, can be considered to have important influences on adolescent smoking. In short, a consideration of the potential moderating effects of gender and ethnicity on the association between SES and adolescent smoking.

2.2 Gender and ethnicity as determinants of adolescent smoking

2.2.1 Gender

Historically, tobacco smoking has been a male phenomenon, most common amongst affluent men and rare amongst women (Gusfield 1993). In the early nineteenth century, tobacco smoking was, as described by Gusfield: “. . . a major symbol and sign of the adult male in American life” (Gusfield 1993). While pipes and cigars were the dominant means of tobacco smoking for men, with the introduction of manufactured cigarettes to the consumer market prior to the 1920s tobacco became more accessible to women in particular (Gusfield 1993). Yet, smoking amongst women did not become widespread or publicly acceptable until after World War II, by which time a much greater number of men – many of whom as soldiers during the war were encouraged by the government provision of free cigarettes – had also adopted the smoking habit (Chapman-Walsh *et al.* 1995; Gusfield 1993). By the 1960s, smoking was no longer the reserve of affluent men. Approximately one third of women and over half of all men, from all social classes, had become regular consumers of tobacco in one form or another (Chapman-Walsh *et al.* 1995). The decline in smoking did not occur until the 1970s following the greater public awareness of the health consequences of smoking. It was over this period of reduced smoking initiation and increased cessation by those from higher SES groups (as noted in Chapter 1), resulted in the emergence of the now characteristic SES gradient in smoking. An associated development was a greater reduction in smoking prevalence amongst men relative to women.

In contrast to the 1970s, the gender difference in smoking prevalence in many ‘Westernised’ nations is currently marginal, with some nations reporting a slightly higher prevalence of smoking among adult females, and others a slightly higher prevalence amongst males (Chapman-Walsh *et al.* 1995). Amongst *adolescents*, the gender difference is also slight in ‘Westernised’ countries, with only minor differences reported (Tyas and Pederson 1998). In New Zealand, young females (aged 20-24) currently have rates of smoking that are just two percent higher than that of young males (SNZ 1998), and a similarly higher prevalence of smoking has been recently reported amongst young women in the U.S. (U.S.DHHS 1989). Nonetheless, gender is unlikely to be a significant ‘determinant’ of adolescent smoking in any substantive causal sense, despite it being perhaps significant in public health terms. Indeed Tyas and Pederson (1998) concluded from their critical appraisal of over 200 studies examining ‘determinants’ of smoking that gender was the only variable *not* predictive of adolescent smoking.

What causal or explanatory role, then, does gender play? Gender cannot be considered as a *mediating* variable (Baron and Kenny 1986) in any causal pathway *between* parental SES and adolescent smoking, simply because SES can not ‘cause’ gender. Thus, gender cannot be an explanation of the association between SES and adolescent smoking because it is *exogenous* (or external) to the causal model. This does not rule out the possibility that gender may have important *moderating* effects on the other intervening variables, as later discussions will reveal, nor does it rule out the possibility, as some research has found, that the *association* between SES and adolescent smoking may *vary according to gender*. Such effects, known as *interaction effects* or *effect modification* – technically referring to the ‘heterogeneity of effect’ or the interdependence of two or more variables (Rothman and Green 1998: 329-330) – are important to consider if one is to understand the relationship between gender, SES, and pathways to adolescent smoking. A number of studies examining the socioeconomic patterning of smoking by gender have, for instance, found that the association between adult smoking and SES varied according to gender. Research conducted in the U.S. has reported a relatively wider gender difference in smoking prevalence amongst the lowest SES group – where the prevalence of smoking amongst males was considerably higher than that of females – compared to the highest SES group, where rates of smoking amongst males and females were much more similar (Chapman-Walsh *et al.* 1995). A number of other studies have shown a clear SES gradient in male smoking, and inconsistent findings on the SES gradient in female smoking (Rahkonen *et al.* 1995). Whether the association between SES and adolescent smoking is the same for both sexes, in New Zealand is unclear – although, if the situation in New Zealand is at all similar to that reported in the U.K. (Lucas and Lloyd 1999), one would expect the socioeconomic gradient in adolescent smoking to be similar for males and females. Hence, although gender is unlikely to be a significant *predictor* of adolescent smoking, and while it can not be directly implicated in the *causal pathway*, its inclusion in the analysis is critical to exploring the potential moderating effects of gender on the association between SES and adolescent smoking.

2.2.2 Ethnicity

While the association between Maori ethnicity and adolescent smoking is clearly evident in New Zealand, the association between ethnicity and adolescent smoking has been reported in only a few studies internationally (Tyas and Pederson 1998; U.S.DHHS 1989). The indigenous North American aboriginal peoples have been found to have rates of smoking that are consistently the highest of any ethnic group in the U.S. (Tyas and Pederson 1998). American whites, followed by Hispanics, were reported to have the next highest rates of smoking, while African Americans have comparatively lower rates (U.S.DHHS 1989). The reason for this is not clear. A possible clue may lie in research into the association between smoking amongst migrant groups and level of acculturation, which suggests that increased levels of smoking are found among those with higher levels of acculturation (Tyas and Pederson 1998). In New Zealand, indigenous Maori have the highest rates of smoking of any ethnic group in the country (Borman *et al.* 1999). Given similarly high rates of smoking among the U.S. indigenous ethnic groups, it is possible that there may be some aspect of the circumstances of indigenous groups – as opposed to minority ethnic or migrant groups – that contributes to their comparatively higher smoking prevalence.

Broughton (1996), in tracing the historical development of smoking among Maori since European colonisation, has illustrated how rapidly smoking became ingrained in Maori culture. The early use of tobacco as currency among Maori, for example, was followed in the 1920s by the depiction of Maori (and Maori children) smoking tobacco in paintings, postcards, posters and cigarette cards, “. . . perpetuating the association between Maori and smoking” (Broughton 1996: 80). Smoking also became very common amongst Maori women in contrast to their European counterparts for whom smoking was a cultural taboo. According to Broughton this was consistent with status differences, “. . . from the European point of view it was quite acceptable for Maori women to smoke pipes in Victorian and Edwardian times as this merely verified their inferior social, cultural and racial status” (Broughton 1996: 94). An historically constructed cultural norm of smoking among Maori, and among Maori women in particular (Broughton and Lawrence 1993), thus, may play an important role in legitimising smoking among Maori.

Nevertheless, when considering the causal model under investigation, ethnicity must be considered as sharing the same status as gender, that is, technically, an *exogenous variable*. This is purely because SES cannot have a causal relationship to ethnicity (although for an argument that SES influences ethnic self-identification, see, Chapple 2000). However, ethnicity may *moderate* the effects of the intervening mechanisms in the postulated causal pathway. For instance, parental smoking may have a different effect for Maori compared to non-Maori (that is the strength of the association between SES and adolescent smoking may vary according to ethnicity). As cited earlier the only New Zealand research in this area has reported a socioeconomic gradient in smoking for non-Maori but not for Maori adolescents (Mitchell 1983; Ree 1986). This appears to be an artefact of the low numbers of Maori in the highest SES groups (Mitchell 1983). This statistical artefact – the under-representation of Maori among high SES groups – has made it difficult to explore further whether the higher prevalence of smoking among Maori is explained by socioeconomic or cultural factors, or some combination.

One possible explanation for the higher rates of smoking among Maori is that it is due to their over-representation amongst *lower* SES groups. Thus, SES may *confound* the association between *ethnicity* and

smoking. Confounding by a third variable may exist when that variable is associated with both the exposure and the outcome, and is not a mechanism in the causal pathway (Rothman and Green 1998:125). This is possible, since ethnicity is associated with both SES (Chapple 2000; Te Puni Kokiri 1998) and adolescent smoking (SNZ 1998), and is not in the causal pathway. Conversely, it could also be argued that the association between SES and adolescent smoking is due to the higher rates of smoking among Maori. In other words, *ethnicity* may act as a *confounder* of the association between SES and adolescent smoking.

Again, as with gender, it is important to consider the role of ethnicity, not as an intervening mechanism or *mediating* variable (Baron and Kenny 1986) linking parental SES to adolescent smoking, but as an exogenous or *moderating* variable which may influence the association between SES and adolescent smoking, and which may act as a *potential* confounder of the SES – adolescent smoking association.

2.3 Intervening mechanisms between parental SES and adolescent smoking

2.3.1 Parental smoking

One of the most straightforward explanations for the socioeconomic gradient in adolescent smoking is that it reflects a similar gradient in parental smoking. In other words, adolescents in lower socioeconomic groups are much more likely to be exposed to parents and family members who smoke (Glendinning *et al.* 1994). These parental patterns may influence adolescent smoking via a number of mechanisms: by increasing the accessibility of tobacco in the home, through a direct role-modelling effect, or through familial socialisation processes in which certain attitudes to, or knowledge about, the effects of smoking are transmitted. Since it is difficult to measure directly the accessibility of tobacco in the home, this potential mechanism is assessed more indirectly and implied in the homes of smoking parents. The most frequently presumed mechanism, and that considered to be the most important is the influence of role-modelling.

In a review of eleven prospective studies by Conrad *et al.* (1992) parental smoking was found to be predictive in only five studies. Yet, in apparent contradiction to this finding, Tyas and Pederson (1998) found that parental smoking was associated with a significantly increased risk of adolescent smoking in twice as many studies as those reporting no association. They also found inconsistent reports by a number of studies examining separately the effects of maternal and paternal smoking, with some reporting “. . . both to be significant, non-significant, or each one significant while the other was not” (Tyas and Pederson 1998: 412). Some of the studies reviewed by the authors found that the influence of paternal smoking was only significant for girls and not for boys (Tyas and Pederson 1998), suggesting that gender may interact with the association between parental and adolescent smoking. Other studies have found that the influence of parental smoking was stronger for younger than older adolescents (Chassin and Presson 1986; U.S.DHHS 1994).

One explanation for such inconsistencies can be attributed to the fact that parental smoking has been measured at different stages of the life-course. In cross-sectional, and a number of longitudinal, studies, parental smoking has been measured at the stage of adolescence, while in some birth cohort studies, parental smoking has been measured at birth. A further difficulty is the assessment of parental smoking only at one point in time, regardless of life stage. Thus, assessing parental smoking at birth is consistent with the idea that this is likely to

be the crucial stage of influence, while measuring parental smoking during adolescence suggests that the important effect is from current parental smoking (Bauman *et al.* 1990). Yet, it is apparent that studies utilising these measures of parental smoking actually sought to measure the role-modelling effects of parental smoking, but to do so necessarily requires a measurement of lifetime exposure to parental smoking, prior to the uptake of smoking in adolescence (Bauman *et al.* 1990). However, as a number of parents will have given up smoking before their offspring enter adolescence, and as smoking cessation is associated with SES, the effects of parental smoking on adolescent patterns are likely to have been obscured.

Bauman *et al.* (1990), using a measure of *lifetime* parental smoking, found parental smoking to be as strongly correlated with adolescent smoking as affiliations with smoking peers. They suggest that the influence of peer smoking on adolescent smoking initiation has been overestimated, while the effects of parental smoking have been underestimated. Linzer (cited in Bauman *et al.* 1990), in the analysis of earlier studies where smoking and *ex-smoking* parents were classified together, found that the smoking behaviour of adolescents whose parents were ex-smokers was much more similar to that of adolescents whose parents were current smokers. The implication of this, if true, is that smoking *cessation* among parents may have little positive effect on the smoking outcomes of their children.

It is possible that the influence of parental smoking cessation on adolescent smoking may be a function of the timing of cessation. The role-modelling effect of parental smoking on very young children may be negligible. Farkas *et al.* (1999), seeking to explore whether parental smoking cessation discouraged adolescent smoking, and whether there was a dose-response relationship between exposure to parental smoking and adolescent smoking, analysed the lifetime smoking histories of 4500 U.S. adolescents from two-parent families. Their analyses revealed that adolescents whose parents had stopped smoking were one-third less likely to have been smokers than those who had a *currently* smoking parent, and that adolescent smokers whose parents had quit smoking were twice as likely to have quit smoking than those who had a currently smoking parent (Farkas *et al.* 1999). If however, family instability and marital breakdown are more common among lower SES families, resulting in a greater proportion of *sole parent* families, then the results of the study by Farkas and colleagues may be biased by the under-representation of lower SES families. It is also possible that the effects of parental smoking cessation on adolescent smoking may be different in higher SES families. Also reported was evidence of a dose response relationship between exposure to parental smoking and adolescent smoking, with earlier parental smoking cessation associated with lower likelihood of the child ever smoking (Farkas *et al.* 1999). Yet, it remains to be seen whether parental smoking cessation actually prevents the onset of smoking among adolescents, or whether it merely delays it. Nevertheless, if one seeks to examine the role-modelling effects of parental smoking (and the potential impact of parental smoking cessation), then a measure of lifetime exposure to parental smoking is preferable. In the case of cross-sectional data, it is preferable to have some recall measure of smoking as well as a measure of current smoking.

However, it is unlikely that parental smoking alone explains all of the SES-adolescent smoking association, and it is probable that there are other socioeconomic effects quite independent of the parental smoking effect. Green and colleagues, noting that it is “. . . accepted lay wisdom that parents are an important influence on their children’s behaviour”, set out to determine which of parental smoking and parental SES was the more important determinant of adolescent smoking (Green *et al.* 1991: 745). While Flay and colleagues have argued that “SES

has been shown to be consistently correlated with smoking in young people not by virtue of any direct effects of social class but because a greater proportion of working-class parents smoke” (cited in Green *et al.* 1991:752), *both* social class and parental smoking behaviour have been found to be *independently* associated with young people’s smoking (Green *et al.* 1991). Thus, it is likely that, in addition to parental smoking, there are other mechanisms associated with SES that are implicated in the causal pathway.

2.3.2 Parental knowledge and attitudes towards smoking

Along with the role-modelling effect of parental smoking, a number of studies have examined the effects of parental attitudes towards, and knowledge about, the effects of smoking (Conrad *et al.* 1992; Fergusson and Horwood 1995; Fergusson *et al.* 1995; Tyas and Pederson 1998). Knowledge of what constitutes a healthy lifestyle and of the implications of various behaviours is arguably a prerequisite for living healthfully. If such knowledge about the ill-effects of smoking were not available, it would be hard to consider it as a ‘risk-taking’ behaviour. Yet, given the widespread publicity about the adverse health effects of smoking, it would be very surprising to find many adults unaware of the health hazards of tobacco smoking in Western societies, although understanding might vary considerably.

A number of measures of parental attitudes towards, and knowledge about, smoking have been utilised in the literature. These include: parental approval or disapproval of offspring smoking, agreement or disagreement with a range of policies on tobacco smoking, and general to specific knowledge of the consequences of smoking (Conrad *et al.* 1992; Tyas and Pederson 1998). In the research reviewed by Conrad *et al.* (1992), parental approval, supportiveness of smoking and positive attitudes towards smoking, were predictive in less than half the studies including such measures. The validity of some findings has been compromised in many cases because the data collected has been on adolescent *perceptions* of parental attitudes, rather than directly by parental interview. It should be noted, however, that in the critical appraisal of literature on determinants of adolescent smoking undertaken by Tyas and Pederson (1998), it was found that having parents in favour of smoking was predictive of later adolescent smoking in the majority of studies in which this was assessed.

The influence on adolescent smoking of parental attitudes or knowledge may largely depend upon the smoking status of the parent. Of all the studies reviewed on adolescent smoking, none have actually examined whether parental attitudes and knowledge of the health hazards of smoking are related to parental smoking status. Whether parental attitudes and knowledge acts as a separate intervening mechanism in the causal chain, or merely a correlate of parental smoking status, remains to be determined.

2.3.3 Early smoking experimentation

A well-documented finding in the psychological literature is the predictive power of prior behaviour (Conrad *et al.* 1992). The established link between early smoking experimentation and subsequent smoking in adolescence is an instance of this (Chassin *et al.* 1990; Fergusson and Horwood 1995; Stanton and Silva 1991). It has been suggested that “such continuities may arise . . . because of a result of learning processes in which the young person progressively learns how to smoke and how to acquire cigarettes” (Fergusson *et al.* 1995: 648). A

younger age of smoking initiation, and early childhood smoking experimentation, have been found in numerous studies to be associated both with higher rates of regular smoking in later years (Burt *et al.* 2000; Chassin 1984; Collins *et al.* 1987; Fergusson and Horwood 1995; Harrell *et al.* 1998; Knoke and Burke 1983), and with lower rates of cessation (Breslau and Peterson 1996). An age and gender interaction effect has also been reported, with females initiating smoking at an older age than males (McGee and Stanton 1993). Yet, knowledge about continuities in behaviour over time tells us little about why such behaviours occur in the first place. Thus, predicting later behaviour on the basis of earlier behaviour does not, of itself, get us any closer to understanding etiology, although obviously it helps us appreciate the maintenance of smoking patterns.

For early smoking experimentation to act as an intervening mechanism in the link between parental SES and adolescent smoking, one would expect that, in addition to the link with adolescent smoking, there would also be an empirical link with parental SES. Giovino's (1999) finding, for example, that adolescents from lower socioeconomic backgrounds begin smoking earlier than their higher SES counterparts, could be taken to suggest that early smoking experimentation may also be related to parental SES (although such an association has not been reported in any of the literature reviewed here). Exactly why those from lower SES backgrounds may be more likely to engage in early smoking experimentation is unclear, although investigators from the CHDS have found that early smoking experimentation was correlated with parental smoking practices (Fergusson and Horwood 1995), suggesting a possible causal relationship between parental smoking and early smoking experimentation.

Thus, one potential explanation of the association between parental SES and adolescent smoking may be that it is, in part, explained by continuities in smoking behaviour over time, where children from lower SES backgrounds experiment with smoking earlier than their higher SES counterparts due to their relatively greater exposure to parental smoking in early childhood (and the associated greater access to tobacco in the home). An examination of the role of early smoking experimentation as a potential intervening mechanism is, therefore, justified on such grounds.

2.3.4 Affiliations with smoking peers

There is substantial empirical support for the effects of peer smoking on adolescent smoking (Conrad *et al.* 1992; Tyas and Pederson 1998). In fact, peer smoking, is often cited as the single most important determinant of adolescent smoking (Tyas and Pederson 1998; Urberg *et al.* 1991). In various studies the measurement of peers has included; best friend, friends, and classmates or similarly aged peers (Tyas and Pederson 1998). The smoking behaviour of 'best friend' has found to be more correlated with adolescents' own smoking status than that of 'friends', and the prevalence of smoking among 'friends' more strongly correlated with adolescent smoking than that of classmates, or more generally, peers of the same age (Tyas and Pederson 1998).

Pathways through which peer smoking may influence adolescent smoking include: increased access to cigarettes (and the associated opportunities for experimentation), peer attitudes (or norms) towards, and knowledge of the health effects of tobacco smoking and peer influence. Increased access to tobacco, as with parental smoking, has tended to be an implied, rather than a directly-measured, mechanism. Peer attitudes, although examined in many studies have not been found to be predictive of adolescent smoking (Conrad *et al.* 1992; Tyas and Pederson

1998). For this reason they are not considered any further in this thesis. Studies examining retrospectively the smoking histories of youth have tended to find that experimentation with smoking is to an important extent a social phenomenon, involving offers of cigarettes from peers rather than isolated incidents of individual experimentation (Lucas and Lloyd 1999). Thus, it is peer influence that is the most often cited link between adolescent and peer smoking. Hence, the focus in a number of health promotion initiatives on the development of 'refusal' skills, and the improvement of self-esteem and self-efficacy, to counteract the effects of peer influence (Botvin *et al.* 1980; Charlton *et al.* 1999; Evans *et al.* 1978; O'Loughlin *et al.* 1998; U.S.DHHS 1989, 1994).

Some authors have noted that age interacts with peer influence, with older age groups more subject to peer influence (Tyas and Pederson 1998). This result has not been replicated by Chassin *et al.* (1996) and Bauman (2001), however. Urberg *et al.* (1991), have suggested that this inconsistency is due to the measurement of peer influence, and to the confounding effect of age-related increases in adolescent smoking (Fergusson *et al.* 1995). The study by Urberg *et al.* (1991) found that older age groups appeared to be more influenced by peers when using a measure of the *proportion* of friends who smoked, while younger age groups appeared more influenced by their peers when using as a measure the *difference* in smoking behaviour of the adolescent in comparison with that of their best friend. Using both measures, they also noted that young boys were the most influenced by peer smoking. Aside from the issue of whether there are age and gender interactions with peer influence, most studies, including that undertaken by Urberg *et al.* (1991), have been of insufficient duration or design to separate out the effects of peer *influence* from peer *selection* (that is, differential association).

Peer influence and differential association

The association of peer with adolescent smoking does not necessarily imply a causal association, in the sense of smoking peers 'recruiting' other adolescents into smoking. It is equally feasible that adolescents seek out peers similar in behavioural orientation to themselves. The direction of causality and the role of selective or *differential association* can only be determined in prospective studies. Engels *et al.* (1997), in a three-wave longitudinal study of over a thousand Dutch secondary school students, examined the extent to which there was peer group homogeneity in smoking behaviour. They stated that "longitudinal research on similarities in cigarette smoking, drug use, sexual behaviour and attitudes to delinquency explicitly show that selective association provides a comparable, or even better interpretation of homogeneity in friendships than influence processes" (Engels *et al.* 1997: 802). The outcome of their study revealed that there was indeed peer group homogeneity in smoking behaviour, and, further, that it was selective association rather than peer influence or peer pressure that was the underlying mechanism producing similarities in peer-group smoking behaviour (Engels *et al.* 1997).

An analysis of longitudinal data from the CHDS reported that both differential association with peers and peer influence had important effects on adolescent smoking (Fergusson *et al.* 1995). Fergusson and colleagues (1995), after examining continuities in cigarette smoking between childhood and adolescence, found two peer-mediated pathways between early smoking experimentation (prior to age 13) and smoking at age 16. In the first peer-mediated pathway, children who engaged in early smoking experimentation (prior to 13 years of age) were more likely to associate at age 15 with peers who smoked. Affiliations with smoking peers at age 15 were

strongly predictive of adolescent smoking at age 16 years (Fergusson *et al.* 1995). In the second pathway, early smoking experimentation was predictive of smoking at age 14, which was then predictive of affiliations with smoking peers at age 15. This in turn, was associated with smoking at age 16 (Fergusson *et al.* 1995). Thus, there may be a number of pathways linking early smoking experimentation and peer smoking, as well as bi-directional influences between peer and adolescent smoking, the importance of which has also been noted by Chassin *et al.* (1984), and by the U.S.DHHS (1989).

Another important consideration is the effect of peer influence and differential association when parental smoking is taken into account. Surprisingly few studies have included both of these measures. As noted by Green *et al.* (1991), the influence of peer smoking on adolescent smoking may be substantially reduced after controlling for parental smoking. Glendinning *et al.* (1994), upon examining data from the Young People's Lifestyle and Leisure Project, explored the effects on adolescent smoking of parental and peer smoking. As expected, they found both peer and parental smoking to be associated with adolescent smoking. However, they also found that young people were more likely to smoke if their friends smoked, *irrespective* of parental smoking (Glendinning *et al.* 1994). In contrast to the finding by Green *et al.* (1991) – where the effect of peer smoking was reduced after accounting for parental smoking – the finding by Glendinning and colleagues suggested the reverse: that is, that peer smoking was the stronger influence (Glendinning *et al.* 1994). They also found that parental smoking behaviour remained an important influence where smoking among friends was uncommon (Glendinning *et al.* 1994). They concluded from this that “parental influences remain strong where smoking is uncommon among friends but that they are greatly reduced where smoking is common among friends” (Glendinning *et al.* 1994: 1455). Moreover, the associations of peer and parental smoking with adolescent smoking were found to be independent of social class background (Glendinning *et al.* 1994). The authors noted that it was “surprising to find that young people's smoking was not more clearly linked to parental social class given the marked social class gradients in adult smoking in general and parental smoking in particular” (Glendinning *et al.* 1994: 1450).

Fergusson *et al.* (1995), in their analysis of continuities in smoking in the CHDS, also included alongside peer smoking a measure of parental smoking behaviour. As previously noted, they found two peer-mediated pathways between early smoking experimentation and smoking at age 16 (Fergusson *et al.* 1995). Interestingly, they also found a third, *direct* pathway between early smoking experimentation and later smoking that was independent of intervening smoking behaviour and peer affiliations (Fergusson *et al.* 1995). Upon further examining the antecedents of early smoking experimentation, the effects of parental smoking, along with a range of other social and contextual factors were considered. They found that the apparent associations between parental smoking practices and smoking in adolescence were explained by the correlation between parental smoking and early smoking experimentation and the correlation between parental smoking and affiliations with smoking peers at age 15 (Fergusson *et al.* 1995). Thus, it appears, that parental smoking leads to adolescent smoking via the indirect paths of early smoking experimentation *and* affiliations with smoking peers, *and* via a direct path to smoking at age 16.

Peer influence and differential association are clearly implicated as pathways between parental SES and adolescent smoking. Furthermore, it is likely that peer influence or selection may moderate or exacerbate the

effects of parental smoking on adolescent smoking. Where the role of peers is implicated in the causal pathway it remains to be seen whether such peer affiliations are determined in any way by parental SES.

2.3.5 Deviant and risk-taking behaviours

Various risk-taking behaviours have frequently been documented as associated with, and predictive of, smoking in adolescence (Burt *et al.* 2000; Collins *et al.* 1987; Conrad *et al.* 1992; Tyas and Pederson 1998). A wide range of measures of risk-taking and associated health-lifestyles have been utilised in the research on determinants of adolescent smoking. For younger children, deviant or rebellious behaviour has often been measured using the psychiatric definition of *conduct disorder* (see APA 1987). A wider range of measures have been applied to adolescents where risk behaviours have included more extreme behaviours such as; other substance use, unsafe sexual behaviour, dangerous driving behaviour (drinking and driving, not wearing seat belts), criminal behaviour and violence, through to more general health lifestyle factors such as diet, exercise, and dental care (Conrad *et al.* 1992; Tyas and Pederson 1998). These measures have been conceptualised as health behaviours, rebelliousness, risk-taking, and problem or deviant behaviour. The potential mechanisms through which deviant behaviour may lead to adolescent smoking are nevertheless, complex.

Risk behaviours and unhealthy lifestyles, or patterns of problem-prone behaviour, are frequently interrelated, such that individuals engaging in one unhealthy or risk-taking behaviour are commonly found to engage in others (Chassin and Presson 1986; Conrad *et al.* 1992; Tyas and Pederson 1998). Knowledge that risk behaviours tend to cluster together in individuals, while providing some insight into the wider context in which adolescent smoking occurs, is not in itself helpful for explaining why certain adolescents are more deviant-prone than others. Explanations for deviant behaviour range along a continuum from largely genetic in origin (or personality-based), to models in which it is viewed as a socially learned behaviour, to broader explanations that see deviance as arising out of adverse structural conditions and to a large extent socially constructed. Assuming for a moment that the association between deviant behaviour and later adolescent smoking can be taken as a given, for it to act as a mechanism in the pathway between SES and adolescent smoking, one would expect some empirical linkages between parental SES and deviant behaviour. Such linkages may occur as a result of socialisation processes, in which children imitate behavioural patterns exhibited by their parents.

As with adolescents, health-related and risk-taking behaviours tend to occur together in adults (Tyas and Pederson 1998). While the socioeconomic patterning of health behaviours has not been extensively studied in New Zealand or elsewhere, there is evidence that such a pattern exists for a number of health-related behaviours other than smoking, such as poor diet (Acheson 1998; Blaxter 1990; Lynch *et al.* 1997; Marmot *et al.* 1995) inadequate exercise (Blaxter 1990; Lynch *et al.* 1997; Macintyre 1986; NZHIS 1995), and hazardous levels of alcohol consumption (Acheson 1998; MOH 1999, 2000). Moreover, a socioeconomic dimension to other health behaviours such as infant feeding practices (Acheson 1998; Horwood *et al.* 1985; Silva and Stanton 1996), sexual behaviours and contraceptive use (Dickson 1996; Lynskey and Fergusson 1993), has also been reported. Lynch *et al.* (1997) have also documented that a large number of adult health-related behaviours among a sample of over 2000 Finnish men were not only associated with their own social class, but also associated with the social class of those men's parents. While not implying that all or even the majority of those from lower socioeconomic backgrounds are likely to exhibit deviant behaviour, it is plausible, given the associations

between parental behaviour and SES, that deviant behaviour is more common among adolescents from lower SES groups. Thus, as with parental smoking, the role-modelling effect of parental health and risk behaviours is likely to be an important influence on the behavioural repertoires of their offspring. However, while this may explain why some youth are more deviant-prone than others, and how socioeconomic differences in deviant behaviour may arise, it does not explain why deviance is linked to adolescent smoking behaviour.

One potential mechanism through which deviance may be linked to adolescent smoking is through affiliation with smoking peers. As noted previously, like-minded peers tend to associate with each other, and deviant peer affiliations are no exception (Fergusson and Horwood 1998; Fergusson *et al.* 1999). This is not to say that the basis of friendships depends upon homogeneity of smoking or other risk-taking behaviours alone. Clearly, not all those engaging in deviant behaviours also smoke, nor do all those who smoke necessarily engage in other deviant behaviours. Nonetheless, affiliation with smoking peers is a much more common occurrence among those who engage in deviant behaviour. Thus, while propensity towards smoking is more common amongst deviant-prone adolescents, greater affiliations with smoking peers arising out of deviance is likely, in turn, to reinforce such pre-existing tendencies towards smoking. Another potential explanation of the association between deviant behaviour and adolescent smoking, is via the empirical linkages between deviant behaviour and educational achievement (see below).

2.3.6 Educational achievement

A correlation between low adolescent educational achievement and adolescent smoking has been well documented (Conrad *et al.* 1992; Glendinning *et al.* 1994; Tyas and Pederson 1998; U.S.DHHS 1994, 1998). Measures of educational achievement have included; school exam pass rates, highest educational qualification upon leaving secondary school, educational aspirations, disaffection with school, and school leaving age, as well as more general measures of the extent to which adolescents participate in school activities such as sports, music and clubs (Conrad *et al.* 1992; Tyas and Pederson 1998).

Sociological literature has long recognised the association between parental SES and educational achievement and this association has been documented in New Zealand over a period of time (Cockerham *et al.* 1997; Lauder and Hughes 1990; Lauder *et al.* 1992; Lauder *et al.* 1985; Robinson 1982). Young people from lower SES homes typically have lower levels of educational achievement and more modest educational aspirations, while those from high socioeconomic backgrounds typically have higher educational aspirations and perform better at school, consistent with the higher educational attainment and expectations of their parents (Mitchell and McManus 1982; Robinson 1982). While the review of prospective studies by Conrad *et al.* (1992) found that a number of educational variables were predictive of adolescent smoking, it is not clear whether this association occurs through a direct effect on adolescent smoking *independent* of family socioeconomic background.

One possible explanation for the relationship is that higher educational achievement or ability may go hand in hand with greater understanding of the adverse health consequences of smoking. However, such a mechanism linking adolescent educational achievement with adolescent smoking is unlikely, in light of the large number of studies finding little or no association between knowledge of the health effects of smoking and actual smoking behaviour among adolescents (Conrad *et al.* 1992; Miller and Slap 1989; Tyas and Pederson 1998). Given that

adolescents relate to like-minded peers, it is also possible that young people associate with those who have similar educational aspirations and levels of educational ability or achievement. In this case, the reported relationship between adolescent educational achievement and adolescent smoking may be due to the link between peer affiliations and educational achievement. To an important extent peer affiliations are socially structured. Thus, the type of school one goes to, and its general socioeconomic profile, are likely to be closely related to the socioeconomic position of the family. The large socioeconomic differences between those who attend private and public schools in New Zealand is one such example of how 'choice' of peer group can be influenced by parental SES. Such socioeconomic differences also exist within the range of public schools in New Zealand, as is evident by the different decile ratings of such schools (MOE 1997). Even within the same schools, early streaming into different classes on the basis of academic achievement ensures that similarly achieving students are streamed in the same class, making the likelihood of socialising with others of similar background all the more likely.

Another potential explanation for the association between educational achievement and adolescent smoking may be that those with low educational achievement become disaffected with school and, in turn, become rebellious or 'deviant'. Conventional wisdom from theories on the sociology of education holds that teachers, either consciously or unconsciously, treat children differentially according to social class background (Hurrell 1995). One argument suggests that schools are essentially middle class institutions designed to reproduce the existing social hierarchy by preparing students differentially for their prospective careers. Such differential preparation for the workforce, although legitimised by the concept of educational achievement, has favoured middle class (predominantly white) children over working-class children, irrespective of actual educational ability (Ball 1986). In addition to teacher bias on the basis of social class, racial bias from teachers has also been reported to influence teacher assessment of student ability (Pumphrey and Verma 1990). For young people in circumstances characterised by such low teacher and parental expectations, deviant behaviour may simply be an expression of disaffection and disillusionment with a culturally inappropriate school system. Thus, the process of schooling itself may contribute to social class patterns in deviant behaviour, which in turn affects educational achievement.

Alternatively, it may be that it is deviance or rebelliousness itself which leads to the differential treatment of students by teachers. Evidence to support this argument can be found in a study by Hurrell (1995), in which multivariate techniques were used to assess whether teachers discriminated between students on the basis of social class, gender and ethnicity, after taking into account pupil behaviour. After accounting for behavioural differences, Hurrell found no ethnic or social class discrimination by teachers, but some discrimination on the basis of gender, resulting in more punishment for boys than girls (Hurrell 1995). Thus, it may be the link between rebelliousness and adolescent smoking, rather than education itself, that explains the association between education and smoking in adolescence. In any case, whether or not teachers treat students differently on the basis of social class or deviant behaviour, there is certainly enough evidence to suggest that educational achievement and deviant behaviour are empirically related. The causal direction of this relationship remains unclear, although in the longitudinal cohort study by Power *et al.* (1991) deviant behaviour in middle adolescence was found to be predictive of later lower educational achievement and subsequent downward social mobility in early adulthood.

Finally, education is also important in one other respect. It is associated with adolescents' social class of destination. Consider, for example, the findings by Glendinning and colleagues where:

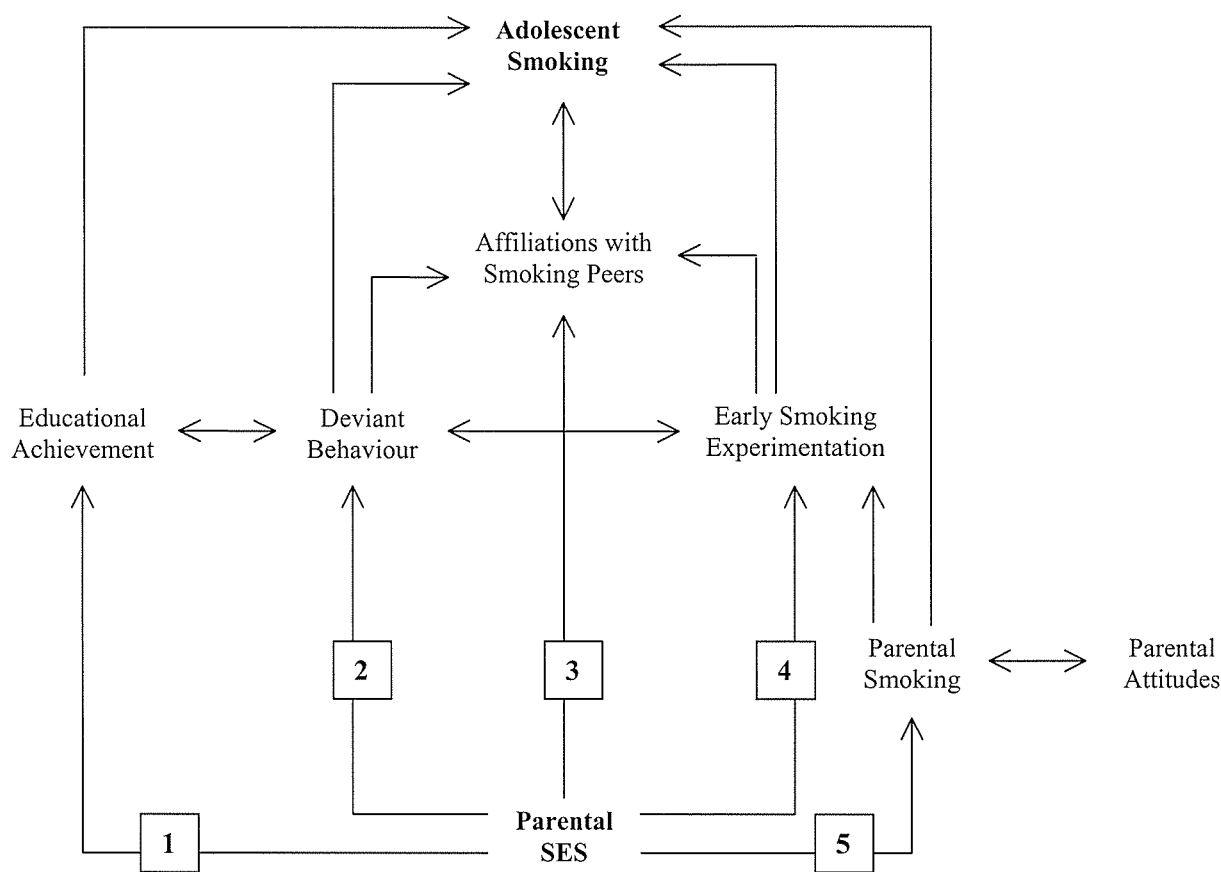
. . . young people who came from middle-class home backgrounds and who themselves occupied relatively advantaged social positions towards the end of adolescence were less likely to smoke, whereas those young people who remained in relatively disadvantaged social positions outside successive generations were more likely to do so. By contrast the smoking behaviour of young people who were socially mobile in or out of the middle classes between successive generations was markedly different from that which might be regarded as 'typical' of their social class of origins, for example young people who currently occupied more advantaged social positions but who came from relatively less advantaged social class family backgrounds were the least likely to smoke (Glendinning *et al.* 1994: 1459).

That adolescent smoking was found to be related more to adolescent's own educational achievement, taken as indicative of their social class of destination, rather than that of their origin, suggests that young peoples' smoking status may be linked more closely to their own educational and occupational trajectories, than their parents'. The finding by Glendinning *et al.* (1994), that for some adolescents, smoking was related to *social mobility*, suggests that educational achievement may act as a moderating influence on other factors which predispose an adolescent to refrain from or initiate smoking. Thus, in the causal model of the potential pathways between parental SES and adolescent smoking, it is therefore critical to examine the role of educational achievement as a potential mechanism linking SES to adolescent smoking, as well as possible interrelationships between education, peer affiliations, and deviant behaviour.

2.4 Model of pathways between parental SES and adolescent smoking

Six key variables – parental smoking, parental attitudes/knowledge, early smoking experimentation, deviant behaviour, affiliations with smoking peers, and adolescent educational achievement – have been drawn from the literature on the determinants of smoking. These variables are both theoretically and empirically justified as potential mechanisms in the causal pathway between parental SES and adolescent smoking. Gender and ethnicity, although exogenous to the causal pathway, are the two additional variables that also need to be considered in relation this association. A hypothetical path model, summarising the potential pathways between SES and adolescent smoking, is depicted in Figure 4.

Figure 4: Hypothetical model of pathways between parental SES and adolescent smoking



The model depicted in Figure 4 shows five main pathways between SES and adolescent smoking. Pathway one, leads from parental SES, via educational achievement, to adolescent smoking. The double-headed arrow between education and deviant behaviour suggests that the two may be related, but the direction of causation could go either way. Pathway two is from SES, via deviant behaviour, which may lead *directly* to adolescent smoking or may lead on to a secondary path, via affiliations with smoking peers (or educational achievement), to adolescent smoking. Again, the double arrow linking deviant behaviour and early smoking experimentation suggests, that while the two are expected to be associated, the direction of causation is unknown. In the third pathway SES leads to affiliations with smoking peers directly, which in turn, leads to adolescent smoking. Pathway four is primarily from SES to early smoking experimentation, and then to adolescent smoking directly, or indirectly via affiliations with smoking peers. In the fifth pathway, SES is linked to parental smoking, which leads to adolescent smoking directly, or through the mechanisms of early smoking experimentation or affiliations with smoking peers. The double arrow linking parental attitudes to parental smoking suggests that the two are likely to be associated and that either one may lead directly or indirectly to smoking in adolescence.

The above model is primarily a result of critically reviewing the literature from the social epidemiology of adolescent smoking. The vast majority of this literature has been informed by psychological theory, emphasising behavioural predispositions and the role-modelling effects of parental behaviour within a framework of social, cognitive and developmental learning theories (Ajzen and Fishbein 1970; Bandura 1963; Jessor and Jessor 1977). While this may be sufficient to explain why certain adolescents initiate smoking when others do not, it is

not *entirely* adequate for explaining why there are social patterns to smoking among adolescents. Before considering exactly how this model is to be operationalised, it is therefore critical to explore some background *sociological* theory as to how and why this phenomenon occurs.

Chapter 3

Theoretical underpinnings

3.1 Introduction

The term socioeconomic status has so far been represented by a wide range of social and economic indicators. The literature reviewed previously in Chapter 2, has, for example, utilised measures of: occupational social class (U.K. and New Zealand literature in particular), educational achievement (or years of schooling), parental income, wealth or status (U.S. in particular), material possessions, and a number of measures of poverty (such as receiving a welfare benefit), and deprivation. In this chapter the theoretical foundation provided by Max Weber on social stratification will be explored, and the term socioeconomic status defined within this framework. This results in essentially a social class definition of SES – an interpretation that underpins the remainder of this thesis when referring to SES. As a theoretical explanation for the social class gradient in cigarette smoking, particular emphasis is given to the Weberian concept of ‘lifestyles’ and the interplay between ‘life chances’ and ‘life choices’ as conceptualised by Bourdieu.

3.2 SES and socioeconomic stratification

In health and epidemiological research, it is standard practice to include SES alongside other critical demographic variables, such as age, sex, and race or ethnicity. Yet, the absence of any concrete definition of the term SES has resulted in the widespread, often ad hoc, use by researchers of an increasing number of variables believed to be indicative of socioeconomic position:

Socioeconomic status is typically used as a shorthand expression for variables that characterise the placement of persons, families, households, census tracts, or other aggregates with respect to the capacity to create or consume goods that are valued in our society. Thus socioeconomic status may be indicated by educational attainment, occupational standing, social class, income (or poverty), wealth, and tangible possessions—such as home appliances or libraries, houses, cars, boats, or by degrees from elite colleges and universities. At some times, it has also been taken to include measures of participation in social, cultural, or political life (Hauser and Warren 1997: 178).

The lack of consensus as to the measurement of SES is apparent by the use of widely varying constructs throughout the literature. While there is strong empirical support that certain indicators (such as income, education and occupation) are highly correlated with each other and predictive of a wide range of health outcomes and health-related behaviors, a number of researchers have also documented ‘independent’ effects of each indicator, suggesting that some indicators hold more explanatory power than others for specific health outcomes. However, the rationale for preferring one particular measure of SES to another is often not explicitly stated. There are always some difficulties encountered when using SES measures. For instance, determining

SES by occupation often excludes adolescents and those outside the labour force (where education may be a preferable measure), and income or wealth measures are difficult to obtain and subject to considerable change with age. Nor is education an ideal measure in that, unless it is translated into income via the occupational structure, it does not necessarily equate with economic position or living standards. Although practical limitations explain in part why certain measures of SES have been adopted, what is clearly absent in much of the research investigating the relationship between SES and health outcomes (or behaviours) is any justification informed by social stratification theory.

As outlined by Davis and colleagues, the concept of SES must be understood in terms of the processes of socioeconomic stratification as: “*the patterned unequal distribution of opportunities, advantages, resources and power among subgroups or a given population [in which] distinct socioeconomic strata . . . exhibit differential life chances, living standards and associated cultural practices*” (Davis *et al.* 1997:8). Such a perspective originates from classical sociological theories of social class and, more specifically, from Max Weber’s theory of social stratification.

3.3 Weberian theory of social stratification

Weber distinguished three critical dimensions of social stratification: class, status and party. In contrast to the writings of Karl Marx – for whom a social class was defined by its relation to the means of production – a class by Weber’s definition is defined by a group of people who share a ‘common economic situation’. A ‘common economic situation’ equates with a shared *market situation* in which those with similar levels of education and skills compete with other groups in the labour market for higher returns in the form of salaries and other valued rewards (Grusky 1994). At the same time, Weber acknowledged the existence of two additional dimensions to stratification, *status* groups – who were defined by the sharing of similar *lifestyles*, as characterised by consumption patterns and prestige – and ‘*party*’ groups, characterised by similarity in *political affiliations*, interests and power (Weber 1968). Theoretically and empirically each of the three dimensions of social stratification may be independent of each other, coexist, or be partially or wholly interdependent (Grusky 1994). While less emphasis was given by Weber to the party dimension of social stratification, suggesting that class and status groups were in fact much more influential in shaping the social hierarchy, it is not immediately apparent whether Weber believed *class* or *status* to be the primary axis of social stratification, or whether they were seen to be of equal importance (Grusky 1994). Considerable disagreement among scholars of Weber’s text has since arisen over whether class or status is the more important force underlying stratification, and the relationship between class, status and party has been conceptualised in various ways.

One of the consequences of this uncertainty is the use of different measures of social class inspired by Weberian theory. In practice, prestige-based scales (commonly utilised in the U.S.) have a close resemblance to, and emphasis on, status (the subjective aspect of socioeconomic differentiation) as the pivotal factor underlying stratification. Occupational socioeconomic scales, by contrast, are based on *objective* income and (arguably objective) educational characteristics of those in specified occupations, emphasising instead the class (economic) dimension of stratification. In reality, both measures are highly correlated, suggesting a strong association between the economic and social status characteristics of occupations (Grusky 1994; Hauser and Warren 1997).

Nevertheless, considerable insight into Weber's understanding of the interplay between class and status, and how this can account for the socioeconomic patterning of lifestyles and associated behaviours, has been highlighted by Cockerham *et al.* (1997) in a recent article exploring the concept of *health lifestyles* within a Weberian framework. Such a framework also provides a critical insight into the socioeconomic gradient in smoking behaviour.

3.3.1 Lifestyles and socioeconomic stratification

Lifestyles, as defined by Cockerham and colleagues, consist of "self-selected forms of consumerism, involving particular choices in food, bodily dress and appearance, housing, automobiles, work habits, forms of leisure, and other status orientated behaviour" (Cockerham *et al.* 1997: 321). For Weber, lifestyles (as characterised by patterns of consumption), are primarily the social and cultural expression of groups of individuals who share a similar social status in society. Such lifestyles were thought by Weber to be a result of the interplay between what he termed *life choices* and *life chances* (Cockerham *et al.* 1997; Elstad 2000). Life chances do not refer merely to good or bad luck but, by Weber's definition, to chances arising out of *structural conditions* which are *primarily socioeconomic in nature* – such as education, income, and employment opportunities (market situation or class) – whereas *life choices* represent behaviours and consumption patterns selected by the individual, but within the constraints of his or her *life chances* (Cockerham *et al.* 1997; Elstad 2000). The concept of life chances also involves "rights, norms and social relationships – the probability that others will respond in a certain manner" (Cockerham *et al.* 1997: 325). In other words: "people have needs, goals, identities, and desires that they match against their chances and probabilities of acquiring; they then select a lifestyle based upon their assessments and the realities of their circumstances" (Cockerham *et al.* 1997: 325). It is in this context that a picture emerges in which class (economic situation) influences lifestyle – not *just* through material conditions or living standards, but also through the *norms and socialisation processes* associated with them. In other words, there is a cultural dimension (or class culture) arising out of material conditions which affects the selection of particular lifestyles.

3.3.2 Health lifestyles and socioeconomic stratification

The Weberian concept of lifestyles can be used to explain what may be termed *health lifestyles*, which have been similarly conceptualised as "collective patterns of health-related behaviour based on choices from options available to people according to their life chances." (Cockerham *et al.* 1997: 338). Health-related behaviours encompass a whole range of activities such as diet, exercise, stress-alleviating practices, alcohol, drug and tobacco use, as well as other methods of minimising health risks, such as the use of seatbelts, immunisation services and health check-ups. Extending the Weberian explanation of lifestyles to that of health lifestyles, health-related behaviours can be similarly conceptualised as a consequence of the interplay between social structure and life choices. Thus, while individual choice certainly plays a role in whether one adopts or discards certain *health*-related behaviours, the range and type of choices available is to a great extent determined by the socioeconomic conditions in which one lives and works – including the social norms and socialisation processes that stem from them. This is not to say that there will be a socioeconomic gradient for all health-related behaviours (although as discussed in Chapter 2 there is evidence of a socioeconomic gradient for a considerable

number of health-related behaviours), as many other factors, such as ethnicity, gender, age and religion are also implicated in the adoption of particular lifestyles. The extent to which individual choice or social structure plays the greater role in shaping health-related behaviours is, however, a matter of considerable debate.

Recent attempts to understand the socioeconomic gradient in health lifestyles have drawn not only on the sociology of Weber, but also that of Bourdieu (Cockerham *et al.* 1997). These key theorists accept that both social structure and individual choice play important roles in shaping lifestyles, although the extent to which they emphasise one over the other varies considerably. Cockerham *et al.* (1997) have argued that Weber's position was one which emphasised the role of individual choice over social structure in determining lifestyles, where life choices are merely 'constrained' by structural conditions rather than wholly determined by them. Thus, from such a position, while smoking may be more congruent with certain styles of life, the decision to smoke or not is largely considered an individual one. Bourdieu on the other hand, in reexamining Weber's concept of class and status (Mennell *et al.* 1992), emphasised the primacy of social structure over that of life choices (Cockerham *et al.* 1997). Bourdieu's position on the issue of the structural (social class) patterning of lifestyles is that they are *both* a consequence of class, and a means to ensure reproduction of the class hierarchy. Thus, smoking would be considered as arising out of the class structure, while at the same time, in combination with other factors that constitute one's health behavioural repertoire, acting as a mechanism that maintains the class structure. If one examines the current evidence, such a perspective has considerable plausibility in explaining socioeconomic gradients in health lifestyles in which smoking behaviour is an important characteristic.

Central to Bourdieu's argument for the importance of social structure over that of individual choice is the role of *habitus*, which, broadly speaking, refers to the social, cultural, physical and economic environment one inherits at birth. In discussing the influence of habitus on lifestyle selection, Bourdieu emphasises the role of social class in determining preferences and tastes, via the transmission of four types of capital: economic (monetary), symbolic (status and prestige), social (social relations between people), and cultural (various kinds of legitimate knowledge (Ritzer, 1996)). For instance, Bourdieu's work in *Distinction* (1984) demonstrated, from an analysis of the expenditure patterns of the various social classes the existence of distinct social class preferences in food, clothing, musical and aesthetic taste, leisure time activities, and other avenues of cultural participation. A recent analysis of cultural consumption practices in the U.S. has also revealed that leisure activities, musical and cultural tastes are associated with social class (Katz-Gerro 2000). However, consumption is not entirely a function of economic capital, but also of the priority given to, or the utility of, particular goods to people in a particular socioeconomic situation.

Bourdieu has highlighted how consumption practices and health lifestyles fulfill various utilitarian functions depending (amongst other things) upon one's socioeconomic circumstances (Grusky 1994). Food preferences, for instance, were found by Bourdieu to be dependent upon each social class's understanding of the effect of food on their body: "the working-class is more attentive to male strength and tends to choose foods that are cheap, nutritious and abundant, while the professions are more concerned about body forms and opt more for food that is light, tasty and low in calories" (Cockerham *et al.* 1997: 326). Class-based food preferences have also been documented as a common feature of contemporary society (Acheson 1998; Blaxter 1990; Lynch *et al.* 1997; Marmot *et al.* 1995), and have been noted among adolescents (Karvonen *et al.* 1999). Similar class-based differences in the choice of exercise patterns have also been reported. Turner (1992) has noted that "weight

lifting articulates working-class bodies, while jogging and tennis produce a body which is more at ease in the middle class milieu or habitus” (Turner 1992: 88). For Bourdieu, preferences arising out of the habitus of one’s social class constitute a ‘*class-based worldview*’ which underpins not only health-related behaviours, but also other class-based practices as well (Cockerham *et al.* 1997: 327). Recent evidence from New Zealand research examining educational decision-making processes among high school students has, for instance, illustrated how educational choices are underpinned by a class-based rationality consistent with the concept of a class-based worldview (Lauder *et al.* 1992). Furthermore, for Bourdieu, the class-based worldview operates “more or less routinely – even unconsciously”, in that the selection of particular lifestyles often occurs in a largely unthinking manner (Cockerham *et al.* 1997: 327). Put simply, the habitus of one’s social class “channels behaviour down paths that appear reasonable to the individual” (Cockerham *et al.* 1997: 327).

3.3.3 Smoking and socioeconomic stratification

Is also possible that smoking serves utilitarian functions associated with one’s position in the class structure. It may be one of the few rites of passage into adulthood readily available to young people from poorer backgrounds where other opportunities for conferring adult status are rare – as has been suggested by Green (1991), and confirmed in recent qualitative research (Daykin 1993; Laurier *et al.* 2000). Alternatively, smoking may serve to alleviate stresses associated with the harsher economic conditions that characterise life for those in lower SES groups – as has been suggested by Graham (1993) from research undertaken in the U.K. Tobacco smoking also has symbolic value. Once a marker of prestige – distinguishing the upper classes from the rest of society (Chapman-Walsh *et al.* 1995; Gusfield 1993) – it is now a symbolic marker generally associated with low social status. Research into adolescent behaviour has further suggested that smoking acts as a symbol for group identity (Mosbach and Leventhal 1988), and smoking has been cited as a means by which social relations between people are established or maintained (Graham 1993). Thus, smoking may be conceptualised as a rational response to certain conditions associated with the socioeconomic structure. Increasingly, however, as well as serving as a mechanism for establishing social relations between people, smoking has become a means for social exclusion and, in some instances, physical segregation (Gusfield 1993). Thus, in line with Bourdieu’s argument that lifestyles are both a consequence of class and a means to ensure the reproduction of classes, smoking can be seen as aiding in establishing and maintaining distinctions between classes, as well as reinforcing homogeneity within classes.

Yet, we know from historical evidence that certain class-based consumption practices are subject to change over time, and this is especially true in the case of health-related behaviour. Health behaviours known to contribute to heart disease were once more common among the higher social classes, to the extent that it was considered a disease of affluence (House *et al.* 1990). Over the last few decades, lower SES groups have “increasingly adopted (and higher socioeconomic groups have increasingly discarded) lifestyles and behaviours (cigarette smoking, high fat diets, heavy alcoholic drinking, and sedentary lives) that have been identified over the past few decades as major risk factors for morbidity, disability and mortality” (House *et al.* 1990: 386). This shift in class-based health behaviours has been attributed to the greater cultural capital of higher SES groups, which has inclined them to accept (and to act upon), the predominating *lifestyle* explanation of disease causation (Cockerham *et al.* 1997).

Certainly, in the area of health and health-related behaviour it has been argued that good health has become an ‘accomplishment’, and acting healthfully the ‘embodiment of class’ (Cockerham *et al.* 1997: 333). As noted by Gusfield:

. . . refraining from smoking becomes necessary to the style of health-oriented, rational, risk-averse people. Following the prescription of medical science demonstrates that they are people of virtue. It shows they take a measured, self-controlled attitude toward their lives and behaviour. They are people who have heard the calls of medical research and have the will to avoid the indulgences . . . (Gusfield 1993:62-63).

Although as Elstad has pointed out:

The function of lifestyles as distinguishing ways of life, signalling how a social position is different from other positions, points to the struggle between positions for esteem, status and prestige in society. In this way, lifestyles can take the form of more or less conscious attempts to underline the dividing lines in society, and to make the social hierarchy more conspicuous. Lifestyles are therefore markers of social location and group identities, making the divisions evident and reinforcing and reproducing the social hierarchies. Such lines of reasoning can for instance lead to the interpretation of the more healthy lifestyles in the privileged strata as not primarily aiming at health at all, but rather as ways of claiming superiority over other social positions (Elstad 2000: 61).

Of course, the extent, to which particular behaviours (other than smoking) of higher SES groups are actually ‘healthier’ or, just considered healthier because they are middle-class behaviours, is a matter for further consideration.

3.4 Implications for understanding pathways between parental SES and adolescent smoking

The mechanisms identified in the previous chapter from the literature on the social epidemiology of adolescent smoking have suggested that factors acting as potential mechanisms in the causal pathway between parental social class and young peoples’ smoking behaviour are likely to be: parental smoking and attitudes toward smoking, deviant behaviour, affiliations with smoking peers, early smoking experimentation, and educational achievement. While much of the literature has examined determinants of adolescent smoking from a psychological perspective, emphasising social, cognitive and developmental learning theories, these theories are not necessarily incompatible with social-class explanations of health-related behaviour, in that young people learn to behave in a certain manner, through cognitive and developmental stages, throughout the socialisation process. What social stratification theories emphasise, however, is the *class context* in which these social behaviours are learned.

Many of the social ‘risk’ factors for smoking, such as parental smoking and parental attitudes towards smoking, can be likened to epidemiological ‘exposures’ varying according to the relative social-class position of the family. Thus, higher social classes can be seen as having a much greater likelihood of exposure to behaviours

and norms that are generally opposed to smoking, middle class families do so to a lesser extent, while families at the bottom rung of the social hierarchy have norms and behaviour that are generally much more conducive to smoking. In such a context, it is not surprising that a mechanism such as early smoking experimentation – which one would expect would be much more prevalent in lower social classes than higher social classes because of parental smoking and attitudinal norms – has been linked to later smoking in adolescence. This may be an important pathway between parental SES and adolescent smoking. Increased access to tobacco in lower SES families is also an important consequence of the higher prevalence of parental smoking in such families, which is also likely to contribute to adolescent smoking by providing increased opportunities for smoking experimentation.

Deviant behaviour, another common social risk factor seen as contributing to adolescent smoking, is also possibly more prevalent the lower down the social hierarchy a family is situated. As noted in Chapter 2, deviant behaviour is probably related to parental SES, given the SES gradient in adult risk behaviours. Greater tolerance of deviant behaviour may be part of the social class milieu of families from lower SES backgrounds, and may potentially be linked to affiliations with smoking peers (as it has been empirically linked with *deviant* peer affiliations), and to educational achievement. Both are associated with higher rates of adolescent smoking. Yet, at the same time, deviant behaviour, like many health-related behaviours, may to some extent be socially constructed and subject to a middle-class definition of what constitutes appropriate or normal behaviour, for instance, “the diagnoses ‘antisocial personality’ has been called a middle-class psychiatrist’s label for patterns of behaviour normal in the lower class, or at least understandable in the lower class because of pressures of poverty and low prestige” (Robins 1978: 619). What remains to be seen is whether deviant behaviour arises out of primary socialisation processes within the home, or whether secondary socialisation processes associated with the school system have a greater or equal role to play in the creation of deviant behaviour among youth.

The final two social ‘risk’ factors for adolescent smoking that have been identified from the epidemiological literature – affiliation with smoking peers and educational achievement – are mechanisms which occur largely outside the social context of the family. These two factors may be conceptualised as part of the wider secondary (non-familial) socialisation processes, which contribute to the socioeconomic patterning of smoking among adolescents. Previous discussions have considered the manner in which affiliations with smoking peers may be linked empirically with adolescent smoking – via greater access to tobacco in the peer group, which may in turn lead to smoking experimentation and initiation – as well as the possibility of bi-directional influences of peer influence and differential association. On the basis of social-class theory, as conceptualised by Weber and Bourdieu, both differential association and peer influence are likely to play important roles in explaining social class patterns in smoking behaviour amongst adolescents.

The finding reported earlier by Glendinning *et al.* (1994) of the association between social mobility and subsequent smoking behaviour amongst young people is also significant, in that it suggests that, for adolescents, movement up or down the social hierarchy is associated with the uptake of the class-based practices of their social class of destination, rather than that of their origin. It appears to be the case that those who are upwardly or downwardly mobile adopt behaviors of their social class of destination. This may be a means of achieving social acceptance in that group or, alternatively, it may be the reverse (that certain behaviours lead to exclusion from certain social groups, which in turn affects social mobility). Smoking may also be seen by higher SES

adolescents as symbolic of low social status, and may act as a cue for various practices of social closure and exclusion, just as non-smoking in a peer group – where smoking is common – may be seen as acting in a manner inconsistent with ‘people like us’. Much of this may, as Bourdieu suggests, occur largely in an unconscious manner, although there is evidence from qualitative research that adolescents are all too aware of smoking as a symbolic identity marker (Mosbach and Leventhal 1988). They are less likely, however, to be conscious of the class basis to such distinctions (although, in some sociological accounts, “. . . youth cultures have been seen as reflecting class divisions . . . [*serving*] as a means of cultural resistance to middle class authority, at the same time as reproducing class divisions by anticipating future lifestyles and preparing young people for working-class adulthood” (Willis cited in Daykin 1993)).

In summary, the epidemiological and sociological literature together provides ample evidence of the existence of class-based practices and the social and economic differences between classes. Both familial socialisation processes and ‘exposure’ factors are likely to vary according to one’s position in the socioeconomic hierarchy. The mechanisms identified in the previous chapter, and discussed again above, are both theoretically and empirically viable factors implicated in the causal pathway between parental SES and adolescent smoking. Most, or all, of these mechanisms are likely to contribute to social-class gradients in smoking behaviour because they are associated with SES and are well-recognised determinants of adolescent smoking. Certainly, a plausible argument can be made for the view that the way in which society is stratified by social class creates environments in which there is a ‘dose-response’ type relationship between exposure to epidemiological risk factors for smoking and smoking outcomes in early adulthood.

Chapter 4

Methodology

4.1 The Christchurch Health and Development Study

The Christchurch Health and Development Study (CHDS) is a longitudinal study following a birth cohort of children born in the Christchurch urban region of New Zealand during the period 15 April 1997 to August 5, 1977 (Fergusson *et al.* 1978). From the outset, the general aims of study were to “. . . build up, from multiple sources, a running record of the major features of the child’s history of health, education and welfare” from birth onwards (Fergusson *et al.* 1989:304). Respondents in the CHDS were studied annually up to age 16, and again at ages 18 and 21. Early data sources included semi-structured maternal interviews (or paternal interview in cases of sole fathers), health diary information, hospital notes, general practitioner records, teacher questionnaires, and psychometric assessment (Fergusson *et al.* 1989). Later data sources were expanded to include respondent interviews and police records. By age 21 there had been sample loss of 254 individuals the sources of which were; mortality (n=25), out-migration (n=127), refusal to participate (n=95) and, inability to trace some participants (n=7). From the original cohort, 79.9% – 1011 individuals (constituting 90.4% of study participants *alive and resident* in New Zealand) – were still participating in the CHDS at age 21. It is the information collected on this study population and their families that is the data source for this thesis.

4.2 Access to the data

Permission to access selected data from the Christchurch Health and Development Study was granted by the Executive Director of the CHDS after submitting the research proposal, detailing the aims, methodology and proposed methods of statistical analysis, that initiated this thesis. The data was provided on computer disks in (version 6) SAS (Statistical Analysis Software) format which was then converted to Version 8 SAS format for the data analysis. As the array of variables available in the CHDS is exhaustive, the data was provided in stages in between discussions centred around methodological and statistical issues, support that was generously provided by the study convenors and by my supervisor throughout the course of this work.

4.3 Proposed methods of statistical analysis

Given the nature of the research question and the data source, multiple regression techniques were chosen as the most appropriate and efficient method of statistical analysis. Multiple regression techniques, although computer-intensive, allow the researcher to examine the effect of more than one independent variable (hereafter called the explanatory variables) on a dependent variable (hereafter called the outcome variable), while at the same time accounting for the interrelationships between the explanatory variables (Nie *et al.* 1975). The particular multiple regression technique adopted for the forthcoming analysis is linear logistic regression. Elaboration of this

method must however, await explication of, firstly, simple bivariate regression and, secondly, multiple regression methods.

4.3.1 Bivariate regression

Bivariate regression, as its name suggests, involves only two variables (outcome and explanatory), and is represented by the equation of a straight line that has the form:

$$y = a + bx$$

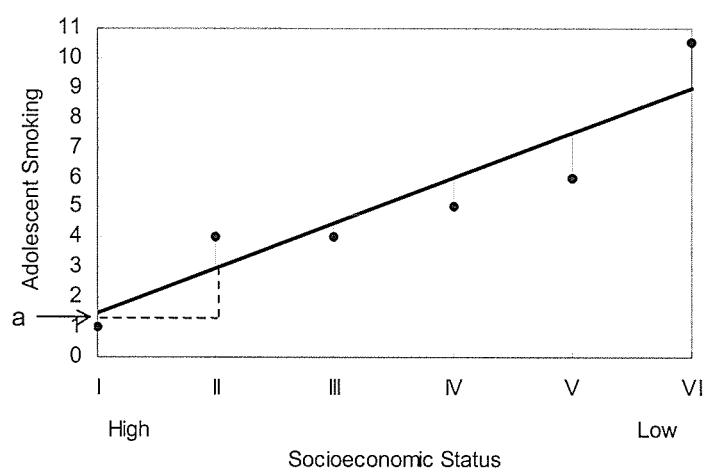
(Fox 1998: 235).

The explanatory variable is always represented by *x*, and *y* denotes the outcome variable (Fox 1998). In this equation *b* is the slope (the estimated effect or coefficient of *x*): a value that reflects the amount by which *y* changes when *x* increases by one unit (Fox 1998). The constant *a* is the intercept, the value of *y* when *x* = 0 (Fox 1998). Denoting the explanatory variable of parental SES by *x* and the outcome variable of adolescent smoking as *y* the relationship between them may be rewritten as:

adolescent smoking = $a + b \times \text{SES} + e$

The term *e* represents the error component, that part of the variability of *y* which is not explained by the relationship with *x*. Using the example of SES and adolescent smoking (with hypothetical data), and using a modified version of an illustration provided by Lewis-Beck (1980), the bivariate linear regression is schematised in Figure 5.

Figure 5: Scatter-plot of the association between SES and adolescent smoking



Source: Modified from Lewis Beck (1980: 12).

In Figure 5, the intercept *a*, does not in itself have any substantive meaning, other than as a baseline from which to measure the effects of the model (Lewis-Beck 1980). It falls at 1.5, and represents the value of *y* (adolescent smoking) when *x* (SES) = 0 (or, as in this case SES group 1, the reference group from which to compare rates of

smoking with other SES groups). The data points above and below the regression line represent the (hypothetical) rates of adolescent smoking for each of the six SES groups. The slope b of the regression line represents the best *estimate* of the association between SES and smoking based on the observed data and describes the average *rate of change* in the outcome variable y as the explanatory variable x changes (Lewis-Beck 1980). Clearly, this estimate does not fit the data points exactly, but rather provides the most parsimonious fit of the data on SES and smoking under the assumptions of a linear association (Fox 1998). The vertical lines between the data points and the regression line – known as residuals, denoted by e (the error term) in the regression equation – show how much the observed data *deviates* from the predicted regression line (Lewis-Beck 1980). In determining the most parsimonious fit of the data, the *least squares method* is utilised to calculate the minimum sum of the squared deviations between the actual outcome variable observations (adolescent smoking) and scores predicted by the regression line (Lewis-Beck 1980). In other words, the regression line is estimated by minimising the sum of the squares of the error (Fox 1998). In Figure 5 we can see that for each unit change in level of SES, the rate of smoking changes by 1.5; thus, for each unit decrease in SES, the average rate of smoking increases by 1.5.

4.3.2 Multivariate regression

When seeking to ascertain the effects of *more* than one explanatory variable (for instance, parental smoking, peer smoking and deviant behaviour), the same logic of the bivariate regression can be extended to the analysis of multiple variables (Lewis-Beck 1980). In multiple regression, the inclusion of additional explanatory variables takes the form of the equation:

$$Y = a_0 + b_1X_1 + b_2X_2 + b_3X_3 + \dots + b_kX_k + e \quad (\text{Lewis-Beck 1980:48}).$$

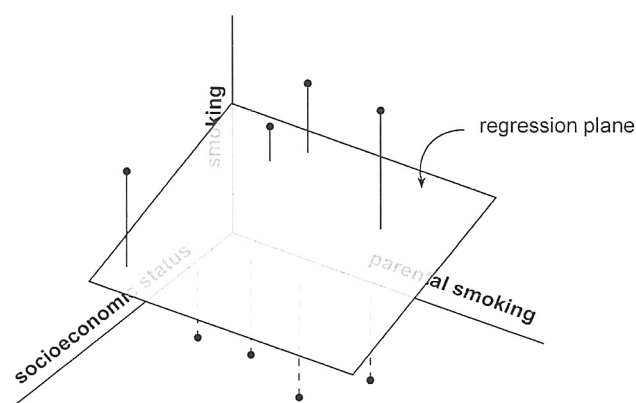
Where: a_0 is the intercept, and e the error term. b_1 is the estimated coefficient (effect) of the first explanatory variable (X_1), and b_2 the estimated effect of the second explanatory variable (X_2), b_3 the effect of the third explanatory variable (X_3) and so on, for all remaining explanatory variables $\dots b_k X_k$. (Lewis-Beck 1980).

Taking up again the example of SES and adolescent smoking, although this time including a third variable, parental smoking, an elementary three-variable form of the above equation can be rewritten as:

$$\text{smoking} = b_0 + b_1 * \text{SES} + b_2 * \text{parental smoking} + e$$

b_1 is the estimated effect of SES (X_1) on adolescent smoking, and b_2 the estimated effect of parental smoking (X_2) on adolescent smoking. Like bivariate regression, multivariate regression uses the method of *least squares* to determine the best fit of the data (Lewis-Beck 1980). However, rather than estimating a regression *line* as depicted by the scatter-plot in Figure 5, multiple regression estimates the most parsimonious fit of the data as regression *plane* (Fox 1998). Schematically this looks something like the illustration presented in Figure 6 – which is a customised version of an illustration by Fox (1998).

Figure 6: Regression plane in a three-dimensional scatter-plot



Source: Modified from Fox (1998: 286).

In Figure 6 the most parsimonious fit of the data showing the effects of the two explanatory variables, SES and parental smoking, on the outcome variable – adolescent smoking – is represented by the flat, two dimensional plane cutting through the three-dimensional space. The actual data points (observations) can be seen falling above and below the plane, and the vertical lines joining them to the plane are representative of the residuals or error – the difference between the actual observations and that predicted by the best-fitting multivariate plane (Fox 1998).

With multiple regression the inclusion of more than one explanatory variables has the effect of simultaneously controlling or holding constant the other explanatory variable/s in the equation (Lewis-Beck 1980). For instance, the estimated effect of SES on adolescent smoking in this example translates upon interpretation to the estimated effect of SES, after taking into account, or statistically controlling for, parental smoking, as well as the reverse – that is, the estimated effect of parental smoking taking into account the effect of SES (Lewis-Beck 1980). The goal in multivariate regression is to find the combination of explanatory variables that best predicts the outcome, taking into consideration what is known and hypothesised about the relationships between variables (Lewis-Beck 1980).

Multivariate regression, while suitable for outcome measures that are continuous in nature (such as the number of cigarettes smoked), is, however, not suited to situations where one has a dichotomous outcome – such as smoking or not smoking. Although an SES gradient for the number of cigarettes smoked amongst adults has been reported, with higher SES adults generally reported as smoking less than their lower SES smoking counterparts (NHC 1998; NZHIS 1995), it is not known whether this is the case amongst adolescents since the outcome has almost always been measured as a dichotomous one (Conrad *et al.* 1992). In addition, there is no standard definition of what constitutes a heavy, moderate or light cigarette smoker. Furthermore, from a public health (and medical) perspective, unlike the case of alcohol consumption, there is no ‘safe level’ of tobacco

consumption (Rabin and Sugarman 1993). Replicating the dichotomous smoker/non-smoker measure most prevalent in the literature is therefore warranted on these grounds.

This decision necessitates using a particular form of multiple regression known as logistic regression designed specifically to cope with a dichotomous outcome. It does this by taking the *log* of the *odds* (or *logit*) of the dependent variable (Hosmer and Lemeshow 1989). With the exception of this difference, logistic regression operates conceptually in the same way as multiple regression, although in place of the *least squares* estimation method, logistic regression utilises the *maximum likelihood* method to estimate the unknown parameters by using an iterative algorithm which maximises the probability of obtaining the observed set of data (Armitage and Colton 1998; Hosmer and Lemeshow 1989).

4.3.3 Logistic regression

The equation for logistic regression has the form:

$$\text{Logit}(Y_i = 1) = B_0 + B_1 X_1 + \sum B_{ji} X_{ji} + e. \quad (\text{Armitage and Colton 1998:2319})$$

Where: $\text{Logit}(Y_i = 1)$ is the log odds of the outcome variable, B_0 is the intercept, $B_1 X_1$ is the estimate (B_1) of the first explanatory variable (X_1), $\sum B_{ji} X_{ji}$ the sum of the estimates for the other explanatory variables, and e the error term.

As with multiple regression, the logistic regression equation provides parameter estimates (coefficients) for the effects of each of the explanatory variables on the outcome variable, while controlling for the effects of the other explanatory variables in the equation (Green and Carroll 1978). Under logistic regression and *maximum likelihood estimation* the regression plane illustrated in Figure 6, is in effect, modelling the logarithm of the odds of the outcome as a function of the explanatory variables (Hosmer and Lemeshow 1989). The aim under logistic regression, as with other regression techniques, is “to find the most parsimonious, yet biologically [*or theoretically*] reasonable model to describe the relationship between the outcome (dependent or response variables) and a set of independent (predictor or explanatory) variables” (Hosmer and Lemeshow 1989:1).

4.3.4 Log-linear modelling

As with other regression techniques, logistic regression has its limitations. While the logistic regression analyses will estimate the critical explanatory variables in the parental SES and adolescent smoking association, it will not identify the *structural* relationships between these variables. For instance, if parental smoking, peer affiliations, and deviant behaviour were all found to be factors in the SES-smoking association, the regression technique will not help us identify the pathways from SES to smoking, whether this be via parental smoking to peer smoking, or, via deviant behaviour to affiliations with smoking peers, or some alternative route. It is for this reason that this thesis complements the use of logistic regression with log-linear modelling.

Log-linear modelling has been described by Knoke and Burke (1983) as a form of contingency table analysis which uses a mathematical equation (see Agresti 1990; Knoke and Burke 1983) to determine whether the

expected (predicted) model is significantly similar to the observed data. It is ideally suited to the analysis of *multidimensional contingency tables* in terms similar to Analysis of Variance (ANOVA). Specifically, each of the variables used in the cross-tabulation *and* their interactions are tested for statistical significance. The log-linear analysis does this by testing for single-variable (first-order) effects – the significance of a variable relative to all other variables in the specified model, and pair-wise-variable (second-order) effects – the significance of a pair of variables (and their interactions) relative to all other pair-wise-variables (and their interactions) in the specified model (Agresti 1990). A Log-Likelihood Ratio test determines whether the *predicted model* is significantly similar (p value <0.05) or different (p value >0.05), to the observed data, and *significant pair-wise effects* reveal the interrelationships between the variables (Agresti 1990; Knoke and Burke 1983). This clarifies the structure of the data, in this case, the actual pathways between parental SES and smoking.

4.4 Description of the variables

4.4.1 Outcome measure: smoking status at age 21

Age of respondent smoking

Although the definition of adolescence typically refers to the teenage years (13-19), it is the smoking outcomes of the cohort at age 21 that will be analysed in this thesis. Practical limitations ruled out the possibility of analysing smoking behaviour at age nineteen – the cohort participants were not interviewed at this age – and using a measure of smoking at an earlier age could have excluded some adolescents who initiated smoking after the ‘year 18 interview’, but before the age of 20. Strictly speaking, this is not a measure of adolescent smoking but of smoking status on reaching adulthood. It is possible that some respondents in the cohort changed smoking status between the year 18 and 21 interviews, although any such change is not likely to have a significant impact on the analysis.

Cigarette smoking

It has already been established that the measure of cigarette smoking proposed is the dichotomous measure of smoking or not smoking. The actual definition of a cigarette smoker in various studies has, nevertheless, ranged from those who smoke any cigarettes at all to those who smoke daily. In the case of adult smoking, the most common definition of a smoker is a person who smokes one or more cigarettes daily (excluding cigars), a definition that is consistent with that used in the 1996 New Zealand Census and the recent national health survey (MOH 1999). The limitations of such a definition is the exclusion of those who smoke less than daily, often referred to as ‘casual’ smokers. This group may be quite diverse in terms of their attachment to the smoking habit. For instance, they may be in an experimental stage of acquiring the habit, in the process of giving up, or, alternatively, they may be what is often termed ‘social’ smokers – those who smoke only in certain social situations (usually where others are also smoking). A definition of smoking which also includes those who smoke *less than daily* is particularly critical when examining the prevalence of smoking among adolescents and young adults. Becoming a smoker is a *process* involving initiation into smoking and smoking experimentation, followed by more and more frequent use of cigarettes by some and discontinuation by others, leading eventually

to the adult status of smoker or non-smoker (U.S.DHHS 1989). From a preliminary glance at the data from the CHDS cohort on the self-reported smoking status of the study participants at age 21, there were a small number who reported smoking in the past month but who did not report smoking daily. These occasional smokers may differ in their SES origin relative to that of daily smokers. For example, one might expect that occasional smokers would be more likely to come from higher SES backgrounds, given the reported SES gradient in smoking cessation (Pearce *et al.* 1985; Rabin and Sugarman 1993), and in the quantity of cigarettes smoked (NHC 1998; NZHIS 1995). Therefore, these two measures may capture different relationships between SES and adolescent smoking. For these reasons, two measures of smoking are proposed: *daily smoking* (smoking one or more cigarettes per day) for consistency with national data on adult smoking, and *any smoking* (those who reported smoking any cigarettes in the past month combined with those smoking daily) – to capture the so-called ‘casual’ or ‘social’ smokers.

Validity of self-reported smoking

The validity of the measures of any and daily smoking at age 21 is, in the CHDS cohort and in numerous surveys of adult smoking, dependent upon the reliable self-reporting of smoking status. As cigarette smoking has arguably become less socially desirable, self-reports of smoking may have become less reliable and subject to an under-reporting bias. There is some evidence from studies comparing self-reported measures of smoking with biochemical measures, and the bogus pipeline effect (a bogus test) of a tendency for self-reports of smoking to be under-estimated, particularly among younger age groups (Murray *et al.* 1987; Patrick *et al.* 1994). Evidence for the under-reporting of smoking is however, still inconclusive (Murray *et al.* 1987; Patrick *et al.* 1994; Wills and Cleary 1997). Furthermore, even ‘objective’ measures are not without their limitations, since they are of little use for identifying social smokers since they occasionally suffer from biases arising from the misclassification of non-smokers as smokers (for example, carbon monoxide levels can be raised in those who do not use tobacco (Patrick *et al.* 1994)). It has been suggested by Patrick *et al.* (1994) that both question wording and interviewer method may also affect the validity of self-reported smoking. In the CHDS ‘year 21 interview’ the assessment of smoking status was elicited from the questions:

- a. “Over the last month have you smoked a cigarette or cigarettes?”
- b. “If yes, how many cigarettes would you smoke per day?”

Such wording appears relatively unambiguous and is unlikely to be a source of potential bias. Those answering ‘no’ to the first question were classified as non-smokers, and those answering ‘yes’ were asked the second question, with responses coded:

- a. <1 per day,
- b. 1-4,
- c. 5-9,
- d. 10-20, and
- e. 21+ per day.

Those smoking one or more cigarettes per day have been classified for the purposes of the forthcoming analysis as *daily* smokers, and those reporting *any* smoking in the *past month* (<1 per day) have been classified as *any* smokers. In contrast to the self-administered questionnaire method, the ‘year 21 interview’ was administered by an interviewer (in the participant’s home) which, as suggested by Patrick *et al.* (1994), is likely to be superior to the self-administered interview method (because the smoker’s awareness of sensory clues as to their smoking status is likely to encourage a truthful response). Notwithstanding the strength of the data collection method, the possibility of under-reporting cannot entirely be ruled out, but it is likely to have a negligible effect on the overall results of the analysis.

4.4.2 Parental SES

The purpose of this thesis is to explore pathways between *parental* SES and adolescent smoking. Therefore, the essential requirement in assessing parental SES is that it be measured at a stage prior to the assessment of other explanatory variables in the causal pathway. To fit this criterion it was decided to use a measure of parental SES at birth. There are a variety of indicators of SES that could have been chosen from the array of variables recorded on the social and economic circumstances of the CHDS respondents families at birth, but one available measure in particular can be justified on both theoretical and empirical grounds: the Elley-Irving socioeconomic index. While a much more recent measure of SES has been developed by Davis *et al.* (1997), its application to the CHDS data was outside the scope of this thesis because of the time and energy required to extract data of sufficient detail to re-code parental occupations. The Elley-Irving socioeconomic index has a long-standing reputation as a reliable tool for determining SES position in New Zealand and has been used extensively in the fields of education, sociology, medicine, psychology, political science and related disciplines (Elley and Irving 1985)

The CHDS cohort was classified at birth using the 1971 Census-based version of the Elley-Irving socioeconomic index. The index uses 1971 Census data on the education and income profiles of male incumbents, aged 25-44, from 550 occupational groups (Elley and Irving 1972, 1976, 1985). The median educational attainment and income were simply determined for each occupation, standardised to a common scale, and combined to give a summary score. On the basis of these summary scores, six categorical occupational classes were formed so that each socioeconomic group constituted a reasonable proportion of the population in a manner consistent with what was understood about the socioeconomic structure of New Zealand at the time (Elley and Irving 1972). Although the rationale underpinning the development of the Elley-Irving index was to assist researchers in determining the socioeconomic representativeness of survey samples, the methodology from which it was constructed, being based upon education and income profiles of occupations, is also consistent with the Weberian theory of social stratification (as discussed in Chapter 3). Furthermore, occupational socioeconomic measures are indicative not only of income, educational attainment and training, but, perhaps more so than any other measure of SES, reflect the cultural, social, financial, community, and residential resources of different socioeconomic strata as well as “their health-related behaviour, and even the life-course opportunities open to them and their children” (Johnson and Hall 1995: 250). A considerable body of empirical evidence has demonstrated substantial correlations between occupational SES measures and a wide variety of health outcomes (Davey-Smith *et al.* 1998; Davis *et al.* 1999), health-related behaviours (House *et al.* 1990), and other social and economic variables (Hauser and Warren 1997). More crucially, the Elley-Irving occupational index has been

found to be highly correlated with a number of health outcomes, behaviours and other social and economic variables in New Zealand society (Davis *et al.* 1997; Elley and Irving 1985; Fergusson and Horwood 1979).

One important criticism of occupational SES measures is their lack of applicability to those outside the workforce. In the CHDS cohort, three percent of respondents' parents were classified as unemployed. In the absence of information on their previous occupation these parents were placed in Elley-Irving group VI, the lowest SES group. This was justified partly on the grounds that those experiencing unemployment are more likely to come from lower SES groups with lower levels of education, and because preliminary analyses revealed that the smoking characteristics of the unemployed parents matched most closely those of the parents in the lowest Elley-Irving SES group.

4.4.3 Ethnicity

In the CHDS data on ethnicity was recorded at birth and at age 21. While recognising that individuals may identify with more than one ethnic group, the analysis could not reflect such complexities which were beyond the scope of this project. The analysis of ethnicity is further limited to the binary definition of Maori or non-Maori, since the number of non-European and non-Maori participants in the CHDS was too small to be used for statistical purposes. The ethnic data collected at birth defined Maori ethnicity by ancestry according to whether or not either or both natural parents had any Maori ancestry. In contrast, the ethnic data collected at age 21 is derived from the 'Maori ethnic group' definition (used by Statistics New Zealand in the 1996 Census), where those classified as Maori have reported Maori as the only, or one of a number of ethnic groups, to which they belong. While both definitions of Maori will be explored, for they may measure conceptually different constructs, it is ethnicity at birth that is of primary interest because of its causal ordering in relation to parental SES and adolescent smoking.

4.4.4 Parental smoking

The earliest available measure of parental smoking in the CHDS was recorded when study participants were one year old. Where both parents were present at age one data was available on maternal and paternal smoking; and in cases of sole parenthood, the information on the one parent was recorded. Because a significant number of household had sole parents – mostly mothers – it was not possible to examine separately the effects of maternal and paternal smoking on offspring smoking. Therefore, a three level variable was constructed consisting of:

- a. Neither parent smoking at age one (including solo parents who reported no smoking).
- b. One parent smoking at age one (including solo parents who reported smoking).
- c. Both parents smoking at age one.

Ideally, as noted in the Chapter 2, examining the role-modelling effect of parental smoking on offspring smoking, the best measure would be one that captures cumulative exposure to parental smoking prior to initiation in adolescence. However, this would require information on changes to parental smoking status, which in turn would also necessitate information on changes in family structure brought on by parental separation,

divorce or death (as well as the introduction of step-parents to the family unit). While some of this information was available for the CHDS participants, neither of these factors will be considered in the forthcoming analysis, since the focus of the research question is on pathways between parental SES and adolescent smoking, not on the cumulative exposure to parental smoking. Changes in parental smoking status and in family structure are therefore a possible source of bias which may affect the validity of the measurement of parental smoking in this study. For instance, one would expect greater smoking cessation amongst parents from higher SES groups. This could result in an over-estimation of the effect of parental smoking for youth from higher SES groups, and possibly also an inflated estimate of the impact of parental smoking on offspring smoking overall. One might also speculate that changes to family structure may be more prevalent in lower SES families, although it is difficult to speculate what possible impact this may have on exposure to parental smoking.

4.4.5 Parental attitudes to smoking

Since no information on parental knowledge of the health consequences of smoking was collected in the CHDS, the focus here is solely on the attitudinal dimension. The only available measure of parental attitudes to smoking was parental attitudes towards their offspring smoking, as measured by participant report at 15 years of age. It should be noted that this measure represents adolescent *perceptions* of parental attitudes, not direct reports of parental attitude.

In the interviewer-assisted questionnaire possible responses to the question on parental attitudes took the following form:

- a. My parents are very opposed to me smoking.
- b. My parents are opposed to me smoking.
- c. My parents are not opposed to me smoking.
- d. I do not know my parents' attitudes to smoking.

While there were a number of options for dealing with category *d* – those who did not know their parents' attitudes to their smoking, one option would be to include it with either *b* or *c*, another would be to treat it as a missing variable (although when looking at respondent smoking status at age 21 and this variable – category *d* corresponds most closely with the 'opposed' category *b*). It was decided to leave the category unchanged and treat it as a four-level categorical variable in the regression analyses. Treating category *d* as missing would have resulted in a sample loss of 10%, and combining it with category *b* required making a judgement which, although reasonable based on the data, was considered unnecessary.

4.4.6 Early smoking experimentation

The only measure of early smoking experimentation collected during the course of the CHDS is whether respondents *ever* or *never* experimented with cigarettes by ten years of age. This is based upon a combination of parental and child reports. Participants were classified as *ever* having experimented with cigarettes if either the child or their parent reported this occurring, and *never* if both parent and child reported never having experimented with smoking. It is possible, as with any recall data that there may be some bias due to under-reporting of early experimentation. For example, parents may not be aware of whether their child has experimented with cigarette smoking, or they may feel reluctant to disclose such information. It is also possible that in the case of child reports some respondents may not remember whether they have experimented with cigarettes. Alternatively, it is possible that parents or children falsely report early smoking experimentation, although this is less likely (Fergusson *et al.* 1995). If there is any bias, it is likely to be in the direction of under-reporting of smoking experimentation. Given that data on this variable was obtained from both parents and the study participants, the effect of any such bias is likely to be minimal.

4.4.7 Deviant behaviour

The measure of deviant behaviour used in the CHDS was the extent of conduct problems exhibited by the child at age eight years. This was based on a combination of parental and teacher reports. This measure, adapted by the study convenors from a combination of items listed in Rutter's and Conners' behaviour rating scales, has been described in detail elsewhere (Fergusson *et al.* 1991) and has been found to be highly predictive of risk-taking behaviours amongst young people in the CHDS (Fergusson and Horwood 1998).

The scale is a sum of 48 items including, for example, bullying other children, bragging and boasting, temper outbursts, destroying others' belongings, stealing, lying, teasing others, being rude to adults, and displaying negative attitudes towards authority. Teachers and parents were asked to rate each child on each of these 48 items in the following manner:

- a. Does not apply (scored 1).
- b. Applies somewhat (scored 2).
- c. Definitely applies (scored 3).

The possible scores in this composite measure of conduct disorder range from 48, representing absolutely no conduct disorder, to 144 (the greatest possible extent of conduct disorder). As there is no standard cut-off point indicating the presence or absence of conduct disorder, the scores were entered into the regression analyses in their continuous format.

4.4.8 Affiliations with smoking peers

The extent of affiliations with smoking peers was measured in the CHDS when the study participants were age 15. Based upon self-reports of friends' smoking behaviour, the respondents were classified into one of three groups:

- a. No friends smoked.
- b. Some friends smoked.
- c. Most friends smoked.

As with all self-report variables, the validity of this measure is dependent upon the accuracy of reports by the respondents. There seems no good reason why respondents should deliberately falsify the prevalence of smoking amongst their friends, however. What is probably more likely is a tendency among adolescents to over-estimate the prevalence of smoking amongst friends, a phenomenon highlighted by Urberg *et al.* (1991). For the purposes of examining the temporal sequencing of affiliations with smoking peers (to determine whether peer influence or peer selection processes are operating), ideally data on peer affiliations and smoking should be collected at regular intervals. However, as data on self-reported peer smoking behaviours in the CHDS is limited to age 15, it will not be possible to examine the *direction* of the association between affiliations with smoking peers and adolescent smoking. Nonetheless, as the prevalence of smoking amongst adolescents has been found to increase two-fold between the ages of 15 and 18 (McGee and Stanton 1993), it is likely that assessing affiliations with smoking peers at age 15 captures relevant data prior to much of the smoking initiation among adolescents. In fact, Fergusson *et al.* (1995) have found this measure to be highly predictive of later smoking in the CHDS cohort.

4.4.9 Educational achievement

Measures of educational achievement in the CHDS cohort, collected prior to most of the smoking initiation in adolescence include: Intelligence Quotient (IQ) at age eight, Test of Scholastic Abilities (TOSCA) scores at age 13, and Grade Point Average (GPA) at ages 11 and 12. It was decided not to use either the IQ or TOSCA measures since they excluded 25% of the cohort living outside of Christchurch at the time these measures were recorded. Additionally, IQ is more strictly a measure of educational *ability* than *achievement*, and thus its association with adolescent smoking is unclear.

GPA was obtained from teacher reports on the level of respondents' achievement at ages eleven and twelve in five curriculum areas; reading, writing, written expression, spelling and mathematics. To provide an overall measure of educational achievement these rankings were averaged over the five curriculum areas and over the two years. Ratings were made on a scale for each child ranging from 10 – very good (in all curriculum areas on all occasions) – to 50 (very poor). While this variable will be entered in the regressions in its continuous format, initially it will be coded into four categories according to the following range of GPA scores to establish the association between SES and education:

- a. 10-20 Very good.
- b. 21-30 Quite good.
- c. 31-40 Quite poor.
- e. 41-50 Very poor.

4.5 Missing data

As the discussion so far has indicated, the variables selected from the CHDS data set have some limitations which may result in some bias due to the nature of the way the data is collected, or the way it is classified. A final issue worth considering is that of missing data. Aside from parental smoking, where data was available for the 1011 respondents, some data was missing for the remaining five *intervening* variables. For these variables the missing data was as follows:

Early Smoking Experimentation 5% (n=53)
Deviant Behaviour 5% (n=54)
Peer Smoking 9% (n=89)
Education 6% (n=58)
Parental Attitudes 10% (n=102)

The potential for bias exists if the missing data on these variables is in some way associated with the smoking outcomes or parental SES, that is, not missing at random. However, as the proportion of missing data was minimal – no more than 10% – and in absence of a reliable method for determining the randomness of the missing data, this potential source of bias was not examined. As such, the forthcoming interpretations of the linear logistic regression and log-liner analyses rest on the assumption that such data was missing at random. In the initial *bivariate* analyses, however, since data was sometimes available for more than the 1011 respondents whose smoking status at age 21 was recorded, this data has also been included.

Chapter 5

Data analysis

5.1 Smoking prevalence by parental SES

Of the CHDS cohort of 1011 at age 21, 403 (39.9%) reported *any* smoking at age 21, and 368 (36.4%) reported smoking *daily*. The proportion of the cohort smoking daily is slightly higher than the 1996 New Zealand Census national figure of 32.3% for the 20-24 age group (SNZ 1998: 90). Applying a chi-square one-sample test (Siegel and Castellan 1988:42-43) showed this difference to be statistically significant ($p<0.05$). These results suggest that rates of smoking in the CHDS cohort were slightly, but significantly, higher than the national average for 20-24 year-olds. Table 1 shows the proportions of the cohort who reported *any* and *daily* smoking by parental SES (as represented by the six Elley-Irving socioeconomic groups varying from I – the highest to VI – the lowest).

Table 1: Association between parental SES and cigarette smoking at age 21

Elley-Irving Group	N = SES group	% (n) Any Smoking	% (n) Daily Smoking
I	115	31.3 (36)	26.1 (30)
II	96	36.4 (35)	32.3 (31)
III	265	35.0 (93)	32.6 (86)
IV	288	41.3 (119)	37.6 (109)
V	139	50.4 (70)	47.5 (66)
VI	108	46.3 (50)	42.6 (46)
Total	1011	39.9 (403)	36.4 (368)
χ^2_5		15.00	17.19
p value		0.010	0.0042
MH Linear χ^2_1		12.07	14.35
p value		0.0005	0.0002
Non-Linear MH χ^2_4		2.93	2.84
p value		>0.05	>0.05

For both the smoking outcomes, there is an overall trend toward increasing prevalence of reported smoking with declining parental SES. Young adults from the lowest socioeconomic group (VI) are 1.6 times as likely as those from the highest socioeconomic group (I) to report *daily* smoking (42% compared to 26%), and 1.5 times more likely to report *any* smoking (46.3% compared with 31.3%). Table 1 shows three tests of significance: a) the

overall chi-square test of significance, b) the Mantel-Haenzel (MH) test of linearity, and c) the test of non-linear trend. These tests showed the presence of a highly significant linear trend between SES and smoking, but no non-linear trend.

5.2 Smoking prevalence by parental SES – stratified by gender

Recent surveys undertaken in New Zealand have demonstrated that the prevalence of smoking is higher among young women than young men (Borman *et al.* 1999). The prevalence of smoking among females in the CHDS cohort was also higher than for males, although only marginally so. The proportion of respondents reporting any smoking was 40.4% for females and 39.3% for males, and for daily smoking the proportions were 36.9% and 35.89% respectively. Thus, there did not appear to be much of a gender difference in smoking behaviour in the CHDS cohort, despite some evidence in New Zealand to the contrary. There still remains, the possibility, however, that the relationship between SES and smoking varied according to gender (see Chapman-Walsh *et al.* 1995; Rahkonen *et al.* 1995). It was therefore necessary to determine whether the association between parental SES and smoking at age 21 was the same for males and females. The prevalence of smoking by gender and parental SES is shown in Table 2.

Table 2: Percent smoking at age 21 by Elley-Irving SES group and gender

a) Males	Elley-Irving Group	n = males SES group	% (n) Any Smoking		% (n) Daily Smoking	
	I	49	30.6	(15)	24.1	(12)
	II	46	36.1	(17)	30.4	(14)
	III	138	34.8	(138)	32.6	(45)
	IV	133	41.6	(55)	39.1	(52)
	V	72	47.2	(34)	44.4	(32)
	VI	58	44.8	(26)	39.7	(23)
	Total	496	39.3	(195)	35.9	(178)
b) Females	Elley-Irving Group	n = females SES group	% (n) Any Smoking		% (n) Daily Smoking	
	I	66	31.8	(21)	27.7	(18)
	II	50	36.0	(18)	34.0	(17)
	III	127	35.4	(45)	32.3	(41)
	IV	155	41.3	(64)	36.8	(57)
	V	67	53.7	(36)	50.8	(34)
	VI	50	48.0	(24)	46.0	(23)
	Total	515	40.4	(208)	36.9	(190)

Inspection of Table 2 suggests that SES is related to smoking in a similar way for both males and females. To confirm this conclusion the data were analysed using logistic regression models, first modelling the main effects (gender and SES), and then running the analysis a second time with an interaction term added.

The logistic regression models fitted were:

1. $\text{Logit}(Y_i = 1) = B_0 + B_1 X_1 + B_2 X_2$
2. $\text{Logit}(Y_i = 1) = B_0 + B_1 X_1 + B_2 X_2 + B_3 (X_1, X_2)$

Where $\text{Logit}(Y_i = 1)$ was the log odds of (any or daily) smoking, $X_1 = \text{SES}$, $X_2 = \text{gender}$, and (X_1, X_2) = the gender by SES interaction.

The results are given in Table 3.

Table 3: Linear logistic regression for gender-SES interaction: any and daily smoking

Outcome	Model	Factor	df	LR χ^2	p value
<i>Any Smoking</i>	A	Parental SES	1	12.0816	0.0005
		Gender	1	0.2469	0.6193
	B	Gender*SES interaction	1	0.1404	0.7079
<i>Daily Smoking</i>	A	Parental SES	1	14.3187	0.0002
		Gender	1	0.2439	0.6214
	B	Gender*SES interaction	1	0.0884	0.7662

The test of main effects is model A.
The test for interaction is derived by subtracting model A (LR chi-square) effects from model B (LR chi-square) effects.

The results in Table 3 suggested:

- a) Parental SES significantly predicted offspring smoking at 21 ($p<0.001$).
- b) Gender had no significant effect on smoking after controlling for SES ($p>0.05$).
- c) There was no significant gender * parental SES interaction ($p>0.05$).

It was concluded from Table 3 that the association between parental SES and any and daily smoking at age 21 was the same for both males and females.

5.3 Smoking prevalence by parental SES – stratified by ethnicity

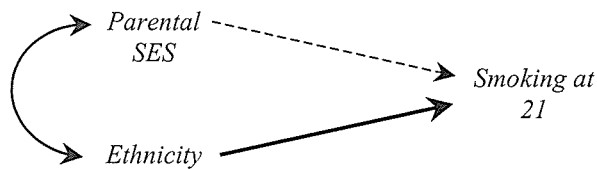
Maori adolescents, like their adult counterparts, have the highest smoking rates of any ethnic group in New Zealand (SNZ 1998). Young Maori, aged 15-24 were 1.8 times as likely (42%) as non-Maori (23%), to report daily smoking in the 1996 Census (SNZ 1998: 90). The definition of ethnicity used here draws on the data collected at birth (those classified as Maori had either or both natural parents with any Maori ancestry and those without Maori ancestry were classified as non-Maori). The prevalence of smoking by ethnicity and SES group is shown in Table 4.

Table 4: Percent smoking at age 21 by Elley-Irving SES group and ethnicity

a) Maori	Elley-Irving Group	n = Maori in SES group	% (n) Any Smoking		% (n) Daily Smoking	
	I	3	0		0	
	II	8	50.0 (4)		50.0 (4)	
	III	17	47.1 (8)		41.9 (7)	
	IV	37	45.1 (17)		45.1 (17)	
	V	16	56.3 (9)		50.0 (8)	
	VI	32	50.0 (16)		46.9 (15)	
	Total	113	47.8 (54)		51.0 (51)	
b) Non-Maori	Elley-Irving Group	n = Non-Maori SES group	% (n) Any Smoking		% (n) Daily Smoking	
	I	112	32.1 (36)		26.8 (30)	
	II	88	35.2 (31)		30.7 (27)	
	III	248	34.3 (85)		31.9 (79)	
	IV	251	40.6 (102)		36.7 (92)	
	V	123	49.6 (61)		47.2 (58)	
	VI	76	44.7 (34)		40.8 (31)	
	Total	898	38.9 (349)		35.3 (317)	

Overall, Table 4 shows that young Maori are 1.4 times as likely to report daily smoking as non-Maori (51% compared to 35% for non-Maori). The difference is less for any smoking. There is also evidence of a much clearer socioeconomic gradient in smoking for non-Maori than for Maori. However, with the exception of Elley-Irving Group I (where no Maori reported smoking at age 21), the prevalence of smoking among Maori is clearly higher than that for non-Maori across all socioeconomic groups. Given both this higher overall prevalence of smoking among Maori, and the over-representation of Maori among lower socioeconomic groups, one contribution to the association between SES and smoking is the empirical linkage between ethnicity and SES. A diagrammatic representation of this hypothesis is depicted in Figure 7.

Figure 7: Ethnicity as a confounder of the SES– smoking association



The curved arrow between parental SES and ethnicity indicates the two are taken as correlated. The bold arrow from ethnicity to smoking at 21 denotes a causal link, while the broken arrow suggests that the association between SES and smoking is a spurious one due to the association between ethnicity and parental SES.

However, it also possible, since ethnicity is associated with both SES and smoking at age 21, that *SES* confounds the association between ethnicity and smoking. This hypothesis is represented in Figure 8.

Figure 8: SES as a confounder of the ethnicity– smoking association

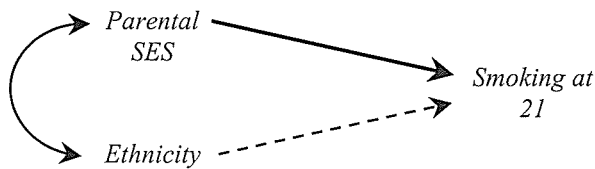


Figure 8 implies that the association between ethnicity and smoking is spurious, being attributable to the over-representation of Maori among lower SES groups.

To test the above models (and to check for possible interaction effects between ethnicity SES and smoking), linear logistic regression models were applied to the data in Table 4.

The logistic regression models fitted were:

1. $\text{Logit}(Y_i = 1) = B_0 + B_1 X_1 + B_2 X_2$
2. $\text{Logit}(Y_i = 1) = B_0 + B_1 X_1 + B_2 X_2 + B_3 (X_1 \cdot X_2)$

Where $\text{Logit}(Y_i = 1)$ was the log odds of (any or daily) smoking, X_1 = SES, and X_2 = ethnicity at birth, and $(X_1 \cdot X_2)$ = the ethnicity by SES interaction.

The results are shown in Table 5.

Table 5: Linear logistic regression with ethnicity and SES: any and daily smoking

Outcome	Model	Factor	df	LR χ^2	<i>p</i> value
<i>Any Smoking</i>	A	Parental SES	1	10.0779	0.0015
		Ethnicity	1	1.3517	0.2450
	B	Ethnicity*SES interaction	1	0.0124	0.9114
<i>Daily Smoking</i>	A	Parental SES	1	11.8980	0.0006
		Ethnicity	1	1.7303	0.1884
	B	Ethnicity*SES interaction	1	0.1395	0.7087

The test of main effects is model A.
The test for interaction is derived by subtracting model A (LR chi-square) effects from model B (LR chi-square) effects.

The fitted model for both smoking outcomes shows:

- a) SES was a significant predictor of smoking ($p < 0.05$).
- b) Ethnicity was not a significant predictor of any or daily smoking after controlling for SES ($p > 0.05$).
- c) There was no significant SES by ethnicity interaction ($p > 0.05$).

The results suggested that data in the CHDS cohort are consistent with the hypothesis depicted in Figure 8. In other words, the higher prevalence of smoking amongst Maori is accounted for by their over-representation among lower SES groups. Yet, with a larger sample it might be possible to detect an ethnic effect on smoking that is not attributable to socioeconomic status. A replication of the above analysis to test for interaction effects and potential confounding, using *self-identified* (by respondent) ethnic group at *age 21*, was also undertaken. In this analysis of the relationship between ethnicity, SES and smoking, the results were exactly the same as those in Table 5; in other words, the effect of parental SES on smoking at age 21 was the same for Maori and non-Maori. Consistent with the results presented above, the higher prevalence of smoking among Maori was again found to be due to their greater concentration among lower socioeconomic groups.

5.4 Baseline model – of SES and smoking outcomes at 21

To provide a *baseline model* of the relationship between parental SES and any and daily smoking at age 21, a linear logistic regression model was fitted to the data in Table 1.

The logistic regression model fitted was:

$$\text{Logit}(Y_i = 1) = B_0 + B_1 X_1$$

Where $\text{Logit}(Y_i = 1)$ was the log odds of (any or daily) smoking and, $X_1 = \text{SES}$.

The results of this model are outlined in Table 6. The table shows: a) estimates of the model parameters (B_0 ; B_1) and their standard errors (SE), b) the log likelihood ratio chi-square test of significance of SES as a predictor of smoking (LR χ^2), and c) the p value test for statistical significance.

Table 6: Baseline Model – Linear logistic regression with smoking outcomes and parental SES

Outcome	Factor	<i>B</i>	SE	LR χ^2	<i>p</i> value
<i>Any Smoking</i>	Intercept	-0.9801	0.1782	30.2674	<0.0001
	SES	0.1584	0.0458	11.9594	<0.0005
<i>Daily Smoking</i>	Intercept	-1.1942	0.1834	42.4037	<0.0001
	SES	0.1764	0.0468	14.1894	0.0002

The results displayed in Table 6 show that parental SES was significantly ($p<0.001$) related to any and daily smoking at age 21: a decline in parental SES was associated with an increase in level of smoking. To provide further interpretation of the model parameter B , it was converted into odds ratios. Using this approach it can be shown that the odds of smoking for the k^{th} level of SES measure relative to SES group I (the reference category) is given by:

$$e^{B \times (k-1)}$$

where k is the level of SES, and e the natural logarithm. The confidence interval for the estimate is given by:

$$e^{(B - 1.96 \times SE) \times (k-1)}$$

(lower confidence limit)

$$e^{(B + 1.96 \times SE) \times (k-1)}$$

(upper confidence limit)

Table 7 displays estimates of the odds ratios for parental SES and any and daily smoking at age 21, with their confidence intervals in brackets.

Table 7: Odds ratios of any and daily smoking at age 21 by parental SES

Elley-Irving Group	Odds Ratios (95% CL) of Any Smoking at 21	Odds Ratios (95% CL) of Daily Smoking at 21
I	1.0	1.0
II	1.17 (1.07 - 1.28)	1.19 (1.09 - 1.31)
III	1.37 (1.15 - 1.64)	1.42 (1.30 - 1.56)
IV	1.60 (1.23 - 2.11)	1.70 (1.55 - 1.86)
V	1.88 (1.32 - 2.70)	2.03 (1.85 - 2.22)
VI	2.20 (1.41 - 3.46)	2.42 (2.20 - 2.65)

Table 7 shows that the odds of smoking increases with decreasing socioeconomic status, with those from SES group VI having odds of any smoking 2.2 times higher than those from SES group I, and 2.4 times higher for daily smoking.

5.5 Intervening variables

The preceding results indicate that the association between SES and respondent smoking was the same for both sexes and ethnic groups, and was not confounded by ethnicity. This suggested a possible causal link between parental SES and offspring smoking at age 21. This conclusion, in turn, required a consideration of the intervening mechanisms that might link parental SES to smoking at age 21.

This section of the analysis reports on the role of the following mediating variables found to be *significant* in explaining the association between SES and smoking outcomes at age 21:

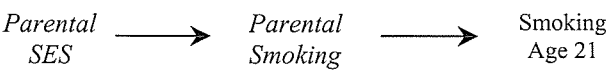
- Parental smoking
- Early conduct problems (deviant behaviour)
- Educational achievement
- Affiliations with smoking peers

Other factors also investigated as initially intended, but subsequently excluded, were: early smoking experimentation, and parental attitudes towards smoking. Preliminary analyses undertaken ruled out the role of parental attitudes as a significant explanation of the association between SES and smoking at 21. Additionally, the results from exploratory analyses of early smoking experimentation are also excluded because it was considered to be too closely similar to the outcome measure. As discussed in Chapter 2, early smoking experimentation should be seen as a stage in adolescent smoking, rather than as a predictor.

5.5.1 Parental smoking as an intervening variable (Model 1)

One of the possible pathways between parental SES and adolescent smoking, noted by a number of authors (Conrad *et al.* 1992; Tyas and Pederson 1998; Bauman *et al.* 1990; Green *et al.* 1991), is through parental smoking. The simplest explanation of the relationship between parental SES and smoking at age 21 is shown in Figure 9. In this model, there is no direct association between parental SES and smoking at age 21, but an indirect effect of parental SES on smoking at age 21 via the effects of parental smoking.

Figure 9: Model 1 – Parental smoking as an intervening variable



For model 1, depicted in Figure 9, to be plausible, parental smoking would need to be associated with parental SES. To determine whether this was the case, the association between parental SES and parental smoking. The results are presented in Table 8:.

Table 8: Association between parental smoking and parental SES

Elley-Irving Group	n = SES group	% (n) Neither parent ever a smoker		% (n) One parent ever a smoker		% (n) Both Parents ever smokers	
I	131	77.1	(101)	15.3	(20)	7.6	(10)
II	114	66.7	(76)	22.8	(26)	10.5	(12)
III	293	57.7	(169)	35.2	(103)	7.2	(21)
IV	332	42.2	(140)	36.8	(122)	21.1	(70)
V	167	34.7	(58)	36.5	(61)	28.7	(48)
VI	143	33.6	(48)	38.5	(55)	27.1	(40)
Total	1180	50.2	(592)	32.8	(387)	17.0	(201)
<hr/>							
χ^2_{10}		121.43					
<i>p</i> value		<0.0001					

Table 8 shows that parental non-smoking – that is, neither parent ever having smoked – increases with higher parental SES. Those from the highest socioeconomic group are 2.3 times as likely as those from the lowest socioeconomic group to have neither of their parents smoking at age one. In contrast, the likelihood of having one or both parents smoking at age one, increases with decreasing parental SES, with those in the lowest socioeconomic group 2.5 times more likely to have one parent smoking, and 3.7 times more likely to have both parents smoking than those from the highest socioeconomic group. The association between parental smoking and parental SES was also highly significant ($p<0.001$).

To determine the effects of parental smoking, on any and daily smoking at age 21, a linear logistic regression model was applied to the data on parental smoking, parental SES and smoking outcomes at age 21. The logistic regression model fitted was:

$$\text{Logit}(Y_i = 1) = B_0 + B_1 X_1 + B_2 X_2$$

Where Logit ($Y_i = 1$) was the log odds of (any or daily) smoking, X_1 = SES, and X_2 = parental smoking.

The results are presented in Table 9.

Table 9: Model 1 – Linear logistic regression results for parental smoking and SES

Outcome	Factor	<i>B</i>	SE	LR χ^2	<i>p</i> value
Any Smoking	Intercept	-1.0121	0.1809	31.3111	<0.0001
	SES	0.1112	0.0488	5.1871	0.0228
	Parental Smoking	0.2912	0.0914	10.1550	0.0014
Daily Smoking	Intercept	-1.2627	0.1869	45.6259	<0.0001
	SES	0.1350	0.0499	7.3071	0.0069
	Parental Smoking	0.3174	0.0926	11.7535	0.0006

As indicated by the *p* values in Table 9, both parental SES and parental smoking had statistically significant effects on any and daily smoking at age 21. The model estimates suggest that controlling for parental smoking had some effect on the association between SES and smoking at age 21. Before the adjustment for parental smoking, the estimate for any smoking was 0.1584 and for daily smoking 0.1764 (see Table 6). After adjustment the estimates were 0.1112 and 0.1350, for any and daily smoking, respectively.

To show the effects of the adjustment for parental smoking on smoking at age 21, Table 10 gives the adjusted and unadjusted odds ratios for each of the SES groups.

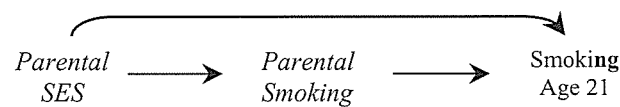
Table 10: Unadjusted and adjusted (for parental smoking) odds ratios for SES and smoking at age 21

Outcome	Elley-Irving Group	Unadjusted Odds Ratios (95% CL)	Adjusted Odds Ratios (95% CL)
<i>Any Smoking</i>	I	1.0	1.0
	II	1.17 (1.07-1.28)	1.12 (1.02-1.23)
	III	2.37 (1.15-1.64)	1.25 (1.03-1.51)
	IV	1.60 (1.23-2.11)	1.40 (1.05-1.86)
	V	1.88 (1.32-2.70)	1.56 (1.06-2.29)
	VI	2.20 (1.41-3.46)	1.74 (1.08-2.81)
<i>Daily Smoking</i>	I	1.0	1.0
	II	1.19 (1.09 - 1.31)	1.14 (1.04-1.26)
	III	1.42 (1.30 - 1.56)	1.31 (1.08-1.59)
	IV	1.70 (1.55 - 1.86)	1.50 (1.12-2.01)
	V	2.03 (1.85 - 2.22)	1.71 (1.16-2.54)
	VI	2.42 (2.20 - 2.65)	1.96 (1.20-3.20)

A comparison of the unadjusted and adjusted odds ratios of Elley-Irving group VI, with Elley-Irving Group I as the reference group, revealed that the odds of *any* smoking reduced after adjustment for parental smoking, from 2.20 to 1.74. The odds of *daily* smoking for those in the lowest socioeconomic group had also reduced after adjustment for parental smoking, from 2.42 to 1.96.

These findings suggest that the association between parental SES and smoking at 21 is in part explained by the causal chain model illustrated in Figure 10.

Figure 10: Relationship between parental SES, parental smoking and smoking at 21



The model in Figure 10 shows that parental SES is linked to parental smoking, which in turn partially explains why those from lower SES groups are more likely to smoke. The arrow linking parental SES to smoking at 21 shows the *still significant association between parental SES and smoking*, after accounting for the effects of parental smoking (a result also reported by Green *et al.* 1991). Therefore, an additional factor or factors are implicated in the pathway between parental SES and smoking at age 21.

The results reported in Table 10 showed the effects on smoking at 21 of a simple adjustment for parental smoking. Below, the analysis is extended to consider additional intervening variables; specifically, conduct disorder, educational achievement, and affiliations with smoking peers. The analysis begins by examining the linkages of each of these factors with parental SES, and then fits a series of linear logistic regression models to examine the intervening role of these variables in the association between parental SES and smoking at 21.

5.5.2 Early conduct disorder as an intervening variable

As discussed in Chapter 2, one potential explanation for the association between SES and adolescent smoking is that young people from lower SES backgrounds are more likely to engage in risk-taking or rebellious behaviour, and, in turn, also more likely to take up smoking (a finding that has been documented by Burt *et al.* 2000; Tyas and Pederson 1998; Conrad *et al.* 1992; Collins *et al.* 1987; and Chassin and Presson 1986). The measure of risk-taking and rebelliousness utilised was the extent of conduct problems exhibited by the child at the age of 8, based on a combination of parental and teacher reports (obtained using items from the Rutter and Conner’s behaviour rating scales). This measure was scored in a continuous rather than a categorical manner. In order to adapt the variable for the correct mode of analysis, it was dichotomised (the top 10% of the cohort with the highest scores on the conduct disorder scale was calculated and compared to the remaining 90% of the cohort). The association between parental SES and conduct disorder is explored in Table 11.

Table 11: Association between conduct disorder and parental SES

Elley-Irving Group	n = SES group	Conduct Disorder	
		% (n) Top percentile	% (n) Rest of cohort
I	118	4 (5)	96 (113)
II	102	6 (6)	94 (96)
III	273	6 (16)	94 (257)
IV	315	13 (40)	87 (275)
V	150	13 (19)	87 (131)
VI	125	18 (23)	82 (102)
Total	1083	10 (109)	90 (974)
χ^2_5		24.86	
<i>p</i> value		0.0001	

The association between parental SES at birth and childhood conduct disorder is highly significant ($p<0.001$). Compared to those from Elley-Irving group I, those from Elley-Irving Group VI are 4.5 times as likely to be in the top 10% of high scores on the measure of conduct disorder.

5.5.3 Educational achievement as an intervening variable

The association between low educational achievement and adolescent smoking has been reported in numerous studies (Conrad *et al.* 1992; Glendinning *et al.* 1994; Tyas and Pederson 1998; U.S.DHHS 1994). Also frequently documented in sociological literature is the association between parental SES and educational achievement, with children from higher SES families consistently found to have higher levels of educational attainment (Lauder and Hughes 1990; Lauder *et al.* 1992; Lauder *et al.* 1985; Robinson 1982). Table 12 outlines the association between parental SES and educational achievement at ages 11 and 12 as measured by teacher reports (see Chapter 4).

Table 12: Association between educational achievement and parental SES

Elley-Irving Group	n = SES group	Educational Achievement			
		% (n) Very good	% (n) Quite good	% (n) Quite poor	% (n) Very poor
I	113	55.8 (63)	32.7 (37)	11.5 (13)	0
II	99	40.4 (40)	40.4 (40)	18.9 (18)	1.0 (1)
III	264	35.2 (93)	41.3 (109)	18.1 (50)	4.6 (12)
IV	303	22.4 (68)	43.6 (132)	30.0 (91)	4.0 (12)
V	141	25.5 (36)	41.8 (59)	24.1 (34)	8.5 (12)
VI	122	16.4 (20)	41.8 (51)	32.8 (40)	9.0 (11)
Total	1042	30.7 (320)	41.1 (428)	23.6 (246)	4.2 (48)
χ^2_{15}		84.82			
<i>p</i> value		<0.0001			

There is a clear socioeconomic gradient between parental SES and educational achievement apparent in the table. The proportion of those classified as *very good* across the five curriculum areas, for instance, ranged from just 16% of those in the lowest SES group to over 55% for those in the highest SES group. The reverse is evident for those classified as *very poor*, with none of the respondents from the highest SES group in this category (compared to 9% from the lowest SES group). In statistical terms, the association between parental SES and educational achievement was highly significant ($p<0.001$).

5.5.4 Affiliations with smoking peers as an intervening variable

Another potential pathway between parental SES and adolescent smoking, and one well documented in the literature examining predictors of adolescent smoking, is via affiliation with peers who smoke (Conrad *et al.* 1992; Fergusson *et al.* 1995; Tyas and Pederson 1998). The extent to which affiliations with smoking peers contributes to adolescent smoking behaviour has been found to vary considerably depending upon other covariates examined; in particular, whether or not a measure of parental smoking has been included in the analysis. In terms of a causal pathway between parental SES and smoking, it is possible that affiliations with smoking peers is more prevalent amongst young people from lower than higher SES backgrounds, because of the greater social acceptability of smoking among lower SES groups. To determine whether there was an association between parental SES and affiliations with smoking peers, using the measure of peer smoking at age 15 (as defined in Chapter 4), the relationship between affiliations with smoking peers and parental SES group is presented in Table 13.

Table 13: Association between affiliations with smoking peers and parental SES

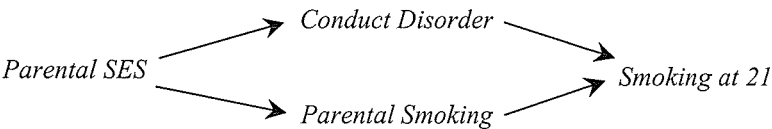
Elley-Irving Group	n = SES group	Affiliations with Smoking Peers		
		% (n) No friends smoked	% (n) Some friends smoked	% (n) Most friends smoked
I	107	53.3 (57)	36.5 (39)	10.3 (11)
II	90	45.6 (41)	43.3 (39)	11.1 (10)
III	248	43.1 (109)	44.4 (110)	11.7 (29)
IV	279	45.2 (126)	36.6 (105)	17.2 (48)
V	134	37.3 (50)	43.3 (58)	19.4 (26)
VI	107	32.7 (35)	46.7 (50)	20.6 (22)
Total	965	43.3 (418)	41.6 (401)	15.1 (146)
χ^2_{10}		18.77		
<i>p</i> value		0.0433		

Table 13 shows that the association between affiliations with smoking peers and parental SES is statistically significant ($p<0.05$). Although there does not appear to be a socioeconomic gradient in the proportion of those reporting having *some* friends who smoked at age 15, there is an SES gradient apparent for the proportion of those reporting having no friends who smoke (ranging from 53.3% for SES group I down to 32.7% for SES group VI). An SES gradient is also evident for respondent reports of *most* friends smoking with 10.3% in SES group I compared to 20.6% in SES group VI.

Linear logistic regression models for the remaining intervening variables

The previous tables have revealed that in all cases there was a highly significant association between SES and the intervening variables of conduct disorder, educational achievement and affiliations with smoking peers. However, so far only *Model 1* (parental smoking) has been applied to the data revealing that parental smoking explains some, but not all, of the association between SES and the smoking outcomes. The remaining three models to be fitted to the data are schematised below.

Figure 11: Model 2 – Including conduct disorder in the model

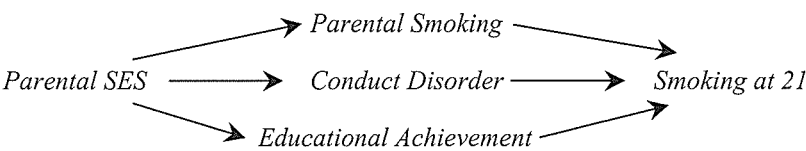


To determine the explanatory power of parental smoking and early conduct disorder in the association between parental SES and smoking at age 21, the following logistic regression model was applied to the data illustrating the association between parental SES and parental smoking, and parental SES and conduct disorder, for the two smoking outcomes:

$$\text{Logit} (Y_i = 1) = B_0 + B_1 X_1 + B_2 X_2 + B_3 X_3$$

Where Logit ($Y_i = 1$) was the log odds of (any or daily) smoking, X_1 = parental SES, X_2 = parental smoking, X_3 = early conduct disorder (continuous measure). *Model 2* implies: $B_1 = 0$; $B_2, B_3 \neq 0$ (meaning no effect for parental SES, but an effect for both conduct disorder and parental smoking).

Figure 12: Model 3 – Including educational achievement in the model

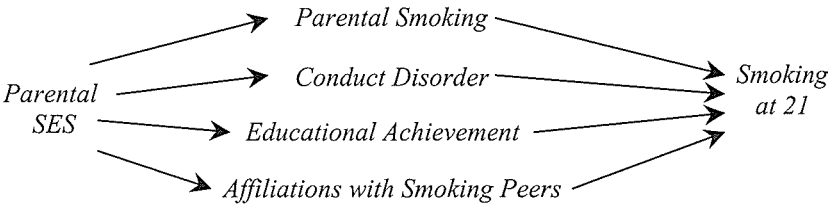


To test the hypothesis of *Model 3* the following logistic regression model was applied to the data:

$$\text{Logit} (Y_i = 1) = B_0 + B_1 X_1 + B_2 X_2 + B_3 X_3 + B_4 X_4$$

Where Logit ($Y_i = 1$) was the log odds of (any or daily) smoking, X_1 = parental SES, X_2 = parental smoking, X_3 = early conduct disorder (continuous measure) and X_4 = educational achievement (continuous measure). *Model 3* implies: $B_1 = 0$; $B_2, B_3, B_4 \neq 0$ (meaning no effect for parental SES, but a significant effect for parental smoking, conduct disorder, and educational achievement).

Figure 13: Model 4 – Including affiliations with smoking peers in the model



To test for the effects of all the above four intervening variables on the association between parental SES and the smoking outcomes, the following logistic regression model was fitted to the data:

$$\text{Logit} (Y_i = 1) = B_0 + B_1 X_1 + B_2 X_2 + B_3 X_3 + B_4 X_4 + B_5 X_5$$

Where Logit ($Y_i = 1$) was the log odds of (any or daily) smoking, X_1 = parental SES, X_2 = parental smoking, X_3 = early conduct disorder (continuous measure), X_4 = educational achievement (continuous measure), X_5 = affiliations with smoking peers. *Model 4* implies: $B_1 = 0$; $B_2, B_3, B_4, B_5 \neq 0$ (meaning no effect for parental SES, but a significant effect for parental smoking, conduct disorder, educational achievement, and affiliations with smoking peers).

5.6 Results of linear logistic regression models for intervening variables

The results for the above three linear logistic regression models, together with those from the baseline model of parental SES and smoking and Model 1, are presented in Tables 14 and 15, for any and daily smoking, respectively.

Table 14: Linear logistic regression results for intervening variables, SES and any smoking at age 21

Factor	Baseline Model SES		Model 1 SES, PS		Model 2 SES, PS, CD		Model 3 SES, PS, CD, E		Model 4 SES, PS, CD, E, ASP	
	unadj <i>B</i>	<i>p</i> value	adj <i>B</i>	<i>p</i> value	adj <i>B</i>	<i>p</i> value	adj <i>B</i>	<i>p</i> value	adj <i>B</i>	<i>p</i> value
SES	0.1584	<0.0005	0.1112	0.0228	0.0640	0.2077	0.0242	0.6468	-0.0008	0.9887
PS			0.2912	0.0014	0.2850	0.0027	0.2847	0.0031	0.1911	0.0628
CD					0.0356	<0.0001	0.0276	0.0026	0.0159	0.1067
E							0.0295	0.0007	0.0344	0.0003
ASP									0.7920	<0.0001
† OR	2.20		1.74		1.38		1.13		1.00	
‡ CL	1.41-3.46		1.08-2.81		0.84-2.27		0.67-1.90		0.57-1.73	

PS = parental smoking; CD = conduct disorder; E = educational achievement; ASP = affiliation with smoking peers.

† Odds Ratios for Elley-Irving group VI relative to Elley-Irving group I.

‡ Confidence Limits for Elley-Irving group VI.

The outcome from the *baseline* linear logistic regression model reported earlier, and those from *Model 1* (parental smoking), are replicated in Table 14 alongside results from the additional regressions (which includes the variables specified in Models 2, 3 and 4). As noted previously, the results of *Model 1*, fitted with just parental SES and parental smoking, showed that both factors had statistically significant effects on any smoking at age 21. This was reflected in the odds of any smoking for those from the lowest Elley-Irving group being reduced from 2.2 to 1.74 after adjustment for parental smoking.

The inclusion of conduct disorder into the regression under *Model 2* shows that both parental smoking and conduct disorder are significant factors in the association between SES and any smoking. While these two variables are statistically important, the effect of SES has been reduced to non-significance. Nonetheless, although there is no longer a significant SES effect after accounting for early conduct disorder and parental smoking, these two intervening variables explain only 0.0944 of the original 0.1584 association between SES and any smoking (model 2 SES effect subtracted from the baseline model SES effect). A comparison of the odds ratios for the models show that those from the lowest SES group had odds of any smoking that were 1.38 times higher than those from the highest SES group after adjustment for the effects of parental smoking and conduct disorder, compared to 1.74 for *Model 1* and 2.2 for the *Baseline Model*.

The results from *Model 3*, in which educational achievement was introduced into the equation, show all three intervening variables as significant. The effect of SES is substantially reduced from the baseline figure of

0.1584 to just 0.0242 after accounting for parental smoking, conduct disorder and educational achievement. These three factors have also reduced the odds ratio for Elley-Irving group VI further, to 1.13.

Under *Model 4*, incorporating affiliations with smoking peers into the equation, the SES effect has been removed entirely, with only educational achievement and affiliations with smoking peers remaining statistically significant explanations of the association between SES and any smoking. It is notable that the *statistically* significant ($p<0.05$) effect of parental smoking has been largely eroded with the inclusion of affiliations with smoking peers. *Model 4* has also reduced the odds of any smoking at age 21 for the lowest SES group to 1.0. Thus, after accounting for parental smoking, conduct disorder, educational achievement and affiliations with smoking peers, the odds of any smoking at age 21 for those from the lowest socioeconomic group were equal to that of the highest Elley-Irving group.

The results for *daily* smoking are given in Table 15.

Table 15: Linear logistic regression results for intervening variables, SES and *daily* smoking at age 21

Factor	Baseline Model SES		Model 1 SES, PS		Model 2 SES, PS, CD		Model 3 SES, PS, CD, E		Model 4 SES, PS, CD, E, ASP	
	unadj <i>B</i>	<i>p</i> value	adj <i>B</i>	<i>p</i> value	adj <i>B</i>	<i>p</i> value	adj <i>B</i>	<i>p</i> value	adj <i>B</i>	<i>p</i> value
SES	0.1764	<0.0002	0.1350	0.0069	0.0872	0.0935	0.0518	0.3385	0.0271	0.6361
PS			0.3174	0.0006	0.3178	0.0010	0.3197	0.0010	0.2307	0.0267
CD					0.0341	<0.0001	0.0268	0.0033	0.0148	0.1339
E							0.0273	0.0021	0.0320	0.0009
ASP									0.8045	<0.0001
† OR	2.42		1.96		1.55		1.30		1.15	
‡ CL	2.20-2.65		1.20-3.20		0.93-2.57		0.76-2.20		0.65-2.01	

PS = parental smoking; CD = conduct disorder; E = educational achievement; ASP = affiliation with smoking peers.

† Odds Ratios for Elley-Irving group VI relative to Elley-Irving group I.

‡ Confidence Limits for Elley-Irving group VI.

As shown in Table 15, the odds ratio for *daily* smoking at age 21 for SES group VI was 2.42 under the *Baseline Model*. This reduced to 1.96 after including parental smoking in *Model 1*. As in the case for any smoking, the results from *Model 2* show that both conduct disorder and parental smoking are significant factors in the association between SES and daily smoking and that, as a result of their impact, the effect of SES has been reduced to non-significance. The odds of daily smoking for the lowest Elley-Irving group have also reduced to 1.55. Conduct disorder and parental smoking explain approximately half (0.0892) of the baseline 0.1764 association between SES and daily smoking.

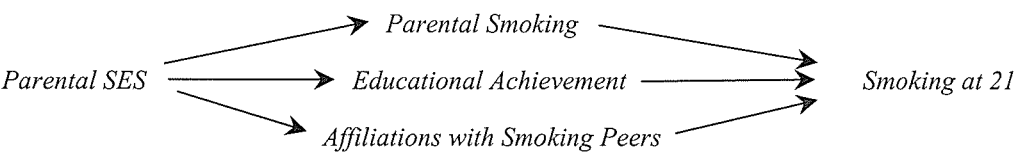
Under *Model 3*, the addition of educational achievement to the regression has reduced the effect of parental SES to approximately a third (0.0518) of its original size (0.1764). As was found to be the case for any smoking, parental smoking, conduct disorder and educational achievement are all significant factors in the association between SES and daily smoking, and the odds ratio for SES group VI has reduced further to 1.30.

In the final regression under *Model 4*, the inclusion of affiliations with smoking peers results in the erosion of the conduct disorder effect to non-significance. Three intervening variables are significant under this model: parental smoking, educational achievement, and affiliations with smoking peers. The odds of daily smoking for the lowest SES group have reduced to 1.15.

Conclusion of linear logistic regression analyses

Comparing the linear logistic regression results of *Model 4*, for any and daily smoking, two variables; educational achievement and affiliations with smoking peers are statistically significant explanations of *any* smoking, while three variables; educational achievement, affiliations with smoking peers, and parental smoking, are statistically significant explanations of *daily* smoking. However, in the case of *any* smoking, the *p* value for parental smoking, at 0.0628, is *marginal* by the <0.05 convention. As such, it was considered *substantively* significant in terms of an *explanation* of the association between parental SES and any smoking at age 21. This explanation of the association between parental SES and *any* smoking can therefore be considered as consistent with that of daily smoking; that is, the relationship between parental SES and smoking at 21 can be accounted for by parental smoking, educational achievement, and affiliations with smoking peers. This conclusion is illustrated in Figure 14.

Figure 14: Significant explanations of the association between parental SES and any and daily smoking at 21



5.7 Log-linear modelling

The final fitted model (Model 4) in Tables 14 and 15 suggests that the association between SES and any and daily smoking at 21 is mediated via three intervening variables: parental smoking, educational achievement, and affiliations with smoking peers. However, a limitation of this analysis is that it does not display the *structural* relationship between the intervening variables in the model. In other words, it is not clear what relationships exist both among the intervening variables, and with the key predictor and outcome variables. For example, it is not clear whether SES has a direct influence on affiliations with smoking peers (which in turn leads to young people smoking), or whether SES leads to educational achievement which, in turn, leads to affiliations with smoking peers and finally to smoking at 21. Other pathways from SES to smoking are also possible.

To provide a more comprehensive picture of the structure of the data, a log-linear model was fitted to the joint distribution of SES, parental smoking, conduct disorder, educational achievement, affiliations with smoking peers, and both the smoking outcomes. To reduce problems associated with sparse data, all variables entered into the log-linear model were dichotomised in the manner outlined in Table 16.

Table16: Method for dichotomising variables for log-linear modelling

Variable	1	Negative attributes	0	Positive attributes
Parental SES	Low	E-I groups 4-6	High	E-I groups 1-3
Parental smoking	Yes	One or both smoked	No	No parents smoked
Conduct disorder	High	Top 10%	Low	Remaining 90% cohort
Educational achievement	Low	Very and quite poor	High	Very and quite good
Affiliations smoking peers	Yes	Some and most friends smoked	No	No friends smoked
Daily smoking at 21	Yes		No	
Any smoking at 21	Yes		No	

A consistent scoring system was adopted in Table 16 such that all the ‘positive’ attributes were given a value of 0, while all the ‘negative’ attributes were scored 1.

Tables 17 and 18 show the response profiles of these variables and the corresponding frequency distributions for any (Table 17), and daily (Table 18) smoking at age 21. The variables in these two tables are as dichotomised in Table 16. In all, there are 64 possible profiles for the above variables (2⁶ contingency table).

Table 17: Frequency distribution of response profiles: *any* smoking at 21

Profile	SES	Parental Smoking	Conduct Disorder	Educational Achievement	Peer Smoking	Smoking at 21	Number responses
1	0	0	0	0	0	0	97
2	0	0	0	0	0	1	19
3	0	0	0	0	1	0	65
4	0	0	0	0	1	1	34
5	0	0	0	1	0	0	10
6	0	0	0	1	0	1	3
7	0	0	0	1	1	0	10
8	0	0	0	1	1	1	17
9	0	0	1	0	0	0	2
10	0	0	1	0	0	1	2
11	0	0	1	0	1	0	2
12	0	0	1	0	1	1	2
13	0	0	1	1	0	0	1
14	0	0	1	1	0	1	0
15	0	0	1	1	1	0	5
16	0	0	1	1	1	1	1
17	0	1	0	0	0	0	27
18	0	1	0	0	0	1	17
19	0	1	0	0	1	0	34
20	0	1	0	0	1	1	26
21	0	1	0	1	0	0	8
22	0	1	0	1	0	1	7
23	0	1	0	1	1	0	5
24	0	1	0	1	1	1	7
25	0	1	1	0	0	0	0
26	0	1	1	0	0	1	1
27	0	1	1	0	1	0	3
28	0	1	1	0	1	1	1
29	0	1	1	1	0	0	0
30	0	1	1	1	0	1	1
31	0	1	1	1	1	0	0
32	0	1	1	1	1	1	0
33	1	0	0	0	0	0	49
34	1	0	0	0	0	1	15
35	1	0	0	0	1	0	21
36	1	0	0	0	1	1	30
37	1	0	0	1	0	0	18
38	1	0	0	1	0	1	5
39	1	0	0	1	1	0	11
40	1	0	0	1	1	1	15
41	1	0	1	0	0	0	1
42	1	0	1	0	0	1	1
43	1	0	1	0	1	0	1
44	1	0	1	0	1	1	0
45	1	0	1	1	0	0	2
46	1	0	1	1	0	1	2
47	1	0	1	1	1	0	4
48	1	0	1	1	1	1	5
49	1	1	0	0	0	0	52
50	1	1	0	0	0	1	18
51	1	1	0	0	1	0	61
52	1	1	0	0	1	1	50
53	1	1	0	1	0	0	10
54	1	1	0	1	0	1	8
55	1	1	0	1	1	0	18
56	1	1	0	1	1	1	38
57	1	1	1	0	0	0	2
58	1	1	1	0	0	1	2
59	1	1	1	0	1	0	4
60	1	1	1	0	1	1	5
61	1	1	1	1	0	0	5
62	1	1	1	1	0	1	8
63	1	1	1	1	1	0	4
64	1	1	1	1	1	1	10
Total							882

Table 18: Frequency distribution of response profiles: *daily* smoking at 21

Profile	SES	Parental Smoking	Conduct Disorder	Educational Achievement	Peer Smoking	Smoking at 21	Number responses
1	0	0	0	0	0	0	100
2	0	0	0	0	0	1	16
3	0	0	0	0	1	0	72
4	0	0	0	0	1	1	27
5	0	0	0	1	0	0	10
6	0	0	0	1	0	1	3
7	0	0	0	1	1	0	12
8	0	0	0	1	1	1	15
9	0	0	1	0	0	0	3
10	0	0	1	0	0	1	1
11	0	0	1	0	1	0	2
12	0	0	1	0	1	1	2
13	0	0	1	1	0	0	1
14	0	0	1	1	0	1	0
15	0	0	1	1	1	0	5
16	0	0	1	1	1	1	1
17	0	1	0	0	0	0	28
18	0	1	0	0	0	1	16
19	0	1	0	0	1	0	34
20	0	1	0	0	1	1	26
21	0	1	0	1	0	0	9
22	0	1	0	1	0	1	6
23	0	1	0	1	1	0	5
24	0	1	0	1	1	1	7
25	0	1	1	0	0	0	0
26	0	1	1	0	0	1	1
27	0	1	1	0	1	0	3
28	0	1	1	0	1	1	1
29	0	1	1	1	0	0	0
30	0	1	1	1	0	1	1
31	0	1	1	1	1	0	0
32	0	1	1	1	1	1	0
33	1	0	0	0	0	0	52
34	1	0	0	0	0	1	12
35	1	0	0	0	1	0	23
36	1	0	0	0	1	1	28
37	1	0	0	1	0	0	19
38	1	0	0	1	0	1	4
39	1	0	0	1	1	0	14
40	1	0	0	1	1	1	12
41	1	0	1	0	0	0	1
42	1	0	1	0	0	1	1
43	1	0	1	0	1	0	1
44	1	0	1	0	1	1	0
45	1	0	1	1	0	0	2
46	1	0	1	1	0	1	2
47	1	0	1	1	1	0	4
48	1	0	1	1	1	1	5
49	1	1	0	0	0	0	55
50	1	1	0	0	0	1	15
51	1	1	0	0	1	0	62
52	1	1	0	0	1	1	49
53	1	1	0	1	0	0	11
54	1	1	0	1	0	1	7
55	1	1	0	1	1	0	20
56	1	1	0	1	1	1	36
57	1	1	1	0	0	0	2
58	1	1	1	0	0	1	2
59	1	1	1	0	1	0	4
60	1	1	1	0	1	1	5
61	1	1	1	1	0	0	5
62	1	1	1	1	0	1	8
63	1	1	1	1	1	0	4
64	1	1	1	1	1	1	10
Total							882

Tables 17 and 18 show that the total number of responses, where data is available on all six variables, has reduced to 882 from the original sample size of 1011 respondents. There are still, however, similar proportions of daily smokers at 36.1% (n=369), and any smokers at 39.7% (n=350) in this sub-sample. As an example of interpreting the data in these two tables, it can be seen that profile 56, in Table 18, refers to 36 respondents who had low SES, one or both parents smoking, no conduct disorder, low educational achievement, some or most peers smoking at age 15, and were smoking at age 21.

Using the CATMOD function in SAS, the data in the 2⁶ contingency tables of the variables in Tables 17 and 18 were then analysed in a log-linear model. The fundamental assumption of this approach is that the contingency data table is adequately summarised by a model, which includes all first-order (single-variable effects) and second-order (pair-wise effects) pathways. This model may be summarised (see Figure 16) as AB, AC, AD, AE, AF, BC, BD, BE, BF, CD, CE, CF, DE, DF, EF where:

- A = parental SES
- B = parental smoking
- C = conduct disorder
- D = educational achievement
- E = affiliations with smoking peers
- F = smoking at 21

Figure 15: Possible pair-wise associations between the variables

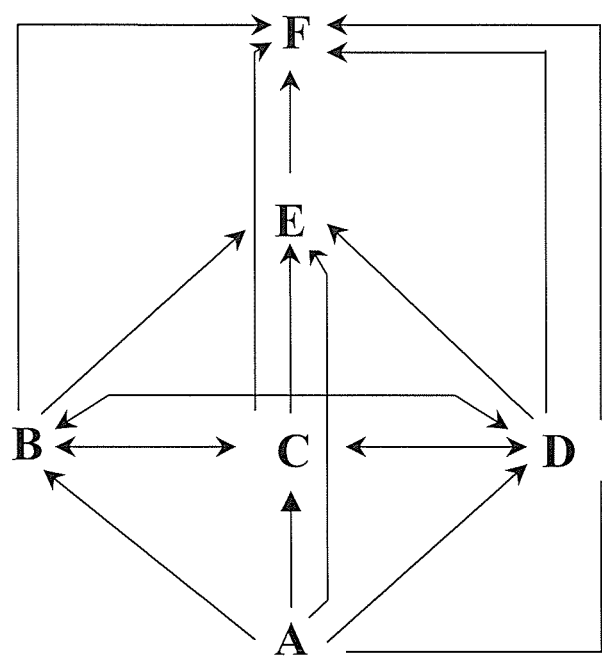


Figure 16 shows all the possible pair-wise (second-order) associations between the variables. Given the results from the final regression analyses reported in Tables 15 and 16, one would expect the pair-wise associations between conduct disorder and smoking at 21 {CF} to be weak (or non-significant), and the associations between both smoking outcomes and parental smoking {BF}, between educational achievement and smoking at 21 {DF}, and affiliations with smoking peers and smoking at 21 {EF}, all to be significant. Also expected to be non-significant is the pair-wise association between parental SES and smoking at 21 {AF}. The results from the log-linear analysis are given in Table 19 for any (a) and daily (b) smoking.

Table 19: Log-linear results for (a) any and (b) daily smoking at age 21

Outcome	Factor	df	χ^2	p value
a) Any Smoking				
<i>First order associations</i>	SES	1	6.26	0.0123
	Parental smoking	1	0.46	0.4955
	Conduct disorder	1	234.24	<0.0001
	Educational achievement	1	7.82	0.0052
	Peer smoking	1	8.10	0.0044
	Smoking at 21	1	3.54	0.0598
<i>Second order associations</i>	SES * parental smoking	1	53.59	<0.0001
	SES * conduct disorder	1	0.27	0.6015
	SES * educational achievement	1	13.68	0.0002
	SES * peer smoking	1	0.08	0.7774
	SES * smoking at 21	1	2.21	0.1387
	Parental smoking * peer smoking	1	6.9	0.0086
	Parental smoking * conduct disorder	1	1092	0.1658
	Parental smoking * education	1	0.02	0.8928
	Parental smoking * smoking at 21	1	4.54	0.0331
	Conduct disorder * peer smoking	1	0.03	0.8528
	Conduct disorder * education	1	37.60	<0.0001
	Conduct disorder * smoking at 21	1	1.29	0.2567
	Education * peer smoking	1	2.37	0.1239
	Education * smoking at 21	1	13.96	0.0002
	Peer smoking * smoking at 21	1	33.93	<0.0001
Log-Likelihood Ratio		36	44.50	0.1564
b) Daily Smoking				
<i>First order associations</i>	SES	1	6.48	0.0109
	Parental smoking	1	0.73	0.3943
	Conduct disorder	1	229.75	<0.0001
	Educational achievement	1	7.31	0.0069
	Peer smoking	1	9.38	0.0022
	Smoking at 21	1	8.24	0.0041
<i>Second order associations</i>	SES * parental smoking	1	52.66	<0.0001
	SES * conduct disorder	1	0.26	0.6071
	SES * educational achievement	1	14.01	0.0002
	SES * peer smoking	1	0.08	0.7732
	SES * smoking at 21	1	1.92	0.1659
	Parental smoking * peer smoking	1	5.84	0.0156
	Parental smoking * conduct disorder	1	1.74	0.1872
	Parental smoking * education	1	0.05	0.8232
	Parental smoking * smoking at 21	1	9.40	0.0022
	Conduct disorder * peer smoking	1	0.08	0.7796
	Conduct disorder * education	1	37.18	<0.0001
	Conduct disorder * smoking at 21	1	2.53	0.1120
	Education * peer smoking	1	2.57	0.1089
	Education * smoking at 21	1	11.67	0.0006
	Peer smoking * smoking at 21	1	34.75	<0.0001
Log-Likelihood Ratio		36	43.92	0.1710

The significant pair-wise associations are given in bold type.

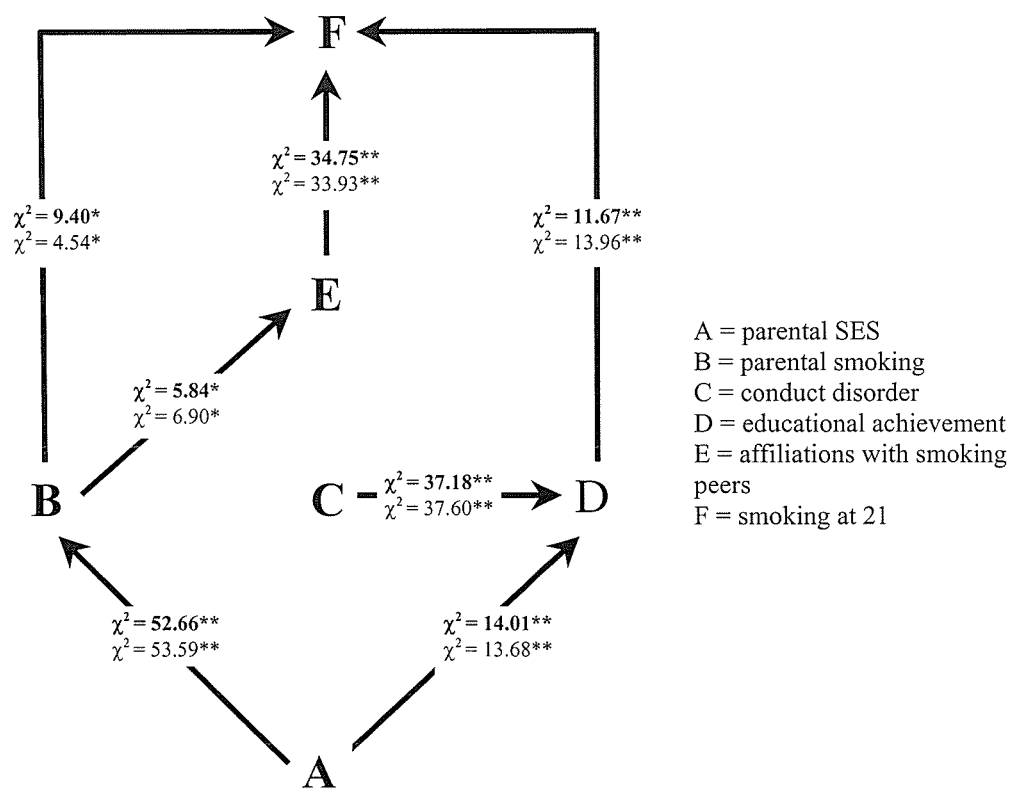
The Log-Likelihood Ratio test of model fit in Table 19 shows that for both any and daily smoking the predicted model fitted to the variables was *not significantly different* from the observed data (both *p* values were >0.05). For both smoking outcomes, there were seven significant pair-wise associations. These were from:

1. SES to parental smoking,
2. SES to educational achievement,
3. Parental smoking to peer smoking,
4. Parental smoking to smoking at 21,
5. Conduct disorder to educational achievement,
6. Educational achievement to smoking at 21 and,
7. Peer smoking to smoking at 21.

No other pair-wise associations were statistically significant on this criteria.

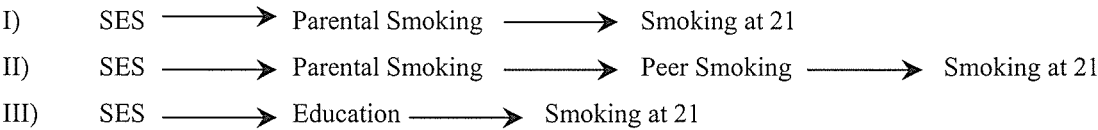
Figure 16 depicts the results from Table 19 as a path diagram with the arrows indicating the significant pathways between parental SES and smoking at 21. The chi-square results given in bold type are the results for daily smoking, and those given in normal type are for any smoking. The significance levels corresponding to the chi-square results are represented by * for statistically significant at the <0.05 convention, and by ** for highly significant at the <0.001 convention.

Figure 16: Structure of pathways between parental SES and any and daily smoking at 21



The following conclusions can be drawn from the fitted models:

- 1. The same structural model described both any and daily smoking at age 21.
- 2. The key intervening variables were: parental smoking, educational achievement, and affiliations with smoking peers.
- 3. The model confirms why conduct disorder was no longer a predictor of any and daily smoking after accounting for educational achievement and affiliations with smoking peers. In particular, conduct disorder is highly correlated with educational achievement ($\chi^2>37$ for any and daily smoking, $p<0.001$), which in turn is linked to smoking at 21. This suggests that the link between conduct disorder and smoking is largely via educational achievement.
- 4. Using this analytical approach, three major pathways account for the linkages between parental SES at birth and smoking at age 21. These are:



These pathways are discussed in detail in Chapter 6.

Chapter 6

Discussion

6.1 Smoking prevalence in the CHDS

The initial stages of the data analysis revealed that smoking amongst young adults in the CHDS was marginally, but statistically significantly, higher than that reported in the New Zealand Census. Part of this difference may be accounted for by the difference in ages for which smoking prevalence has been determined (for all those aged 21 in the CHDS, compared to the wider age band of 20-24 in the Census). It is also possible that in the CHDS cohort some of those who reported smoking at age 21 may become non-smokers by age 24, in which case the smoking prevalence recorded by both data sets may still be correct. Alternatively, some individuals may initiate smoking after 21 years of age (although the actual numbers who do so are likely to be small given that smoking behaviour is typically established during adolescence (Glendinning *et al.* 1994; Townsend 1995). The census data cited also refers to an earlier time period (1996) than that collected via the 'year 21 interview' of the CHDS cohort (1998), in which case the higher prevalence of smoking in the CHDS cohort may be a result of a recent increase in smoking rates for this age group.

6.2 Association of parental SES with smoking at age 21

The association between parental SES at birth and smoking at age 21 was statistically significant, with those in the lowest SES group having *odds* of any and daily smoking that were between 2.2 and 2.4 times that of those from the highest SES group. This association between parental SES and young peoples' smoking behaviour is consistent with findings from other studies conducted in New Zealand (Ree 1986; Stanhope 1975; Stanton and Silva 1991; Stanton *et al.* 1989). It may also help to clarify the inconsistent findings on the parental SES and adolescent smoking association noted by Tyas and Pederson (1998), Conrad *et al.* (1992), and Glendinning *et al.* (1994). Unlike the current investigation, many of the studies examined by Tyas and Pederson (1998) and Conrad *et al.* (1992) were, based upon school-age populations, which are likely to be subject to bias resulting from the under-representation of lower SES students (who are known to leave school earlier and likely to have higher rates of smoking). This may partially account for discrepant findings in many studies examining the association between SES and adolescent smoking.

Attempts to compare the findings from the preceding analysis of the CHDS with those reported in the few studies examining the parental SES and offspring smoking association is also somewhat hampered by other methodological differences, such as the specific measure of parental SES and the point in time at which it was measured. Most cross-sectional, and some longitudinal, studies have measured parental SES during adolescence rather than at birth. However, an essential requirement for determining a causal association is that the exposure (parental SES) must precede the outcome in question. While parental SES could have been reassessed at a point in time immediately prior to the mechanisms linking parental SES to offspring smoking behaviour, this was not a feature of the study methodology. Furthermore, parental occupational data was not collected for

cohort members after birth and prior to the age at which the potential intervening variables were assessed for the current analysis.

6.3 Socioeconomic pattern of smoking by gender and ethnicity

6.3.1 Gender

The prevalence of female smoking at age 21 was only about one percent higher than that reported for males, whereas the 1996 Census reports that the smoking prevalence amongst females aged 20-24 was two percent higher than that reported by males. Again, the difference in age categories and years for which smoking prevalence has been measured in the CHDS cohort and the census means that they are not directly comparable. Nonetheless, the gender difference in smoking prevalence is slight, a finding that is consistent with the international literature from 'Western' countries suggesting that gender is not a significant predictor of adolescent smoking (Chapman-Walsh *et al.* 1995; Tyas and Pederson 1998). Furthermore, the association between parental SES and smoking at age 21 was found to be the same for both males and females, indicating that the New Zealand situation is similar to that reported for adolescents in the U.K. (Lucas and Lloyd 1999).

6.3.2 Ethnicity

As expected, the prevalence of daily smoking amongst Maori respondents in the CHDS cohort was considerably higher than that reported by non-Maori respondents. Young Maori were 1.4 times as likely to report daily smoking (51%) as non-Maori (35%). In addition, the prevalence of smoking amongst young Maori was clearly higher than that of non-Maori across all socioeconomic groups, with the SES gradient in Maori smoking less evident than that observed for non-Maori. This is consistent with the New Zealand research by Mitchell (1983) and Ree (1986).

Nonetheless, logistic regression results to test for interaction effects revealed that, there were no multiplicative effects (interactions) of ethnicity and SES on smoking prevalence. Thus, it was concluded that the association between parental SES and smoking was the same for Maori and non-Maori in the CHDS cohort; in other words, the higher prevalence of smoking amongst Maori was explained by their over-representation among lower SES groups. This does not rule out the possibility that the association between SES and smoking may be shown to vary in a much larger sample. As was found to be the case by (Mitchell 1983), in the CHDS cohort there were very low numbers of Maori in the highest Elley-Irving SES group. The findings from the previous data analysis certainly highlight the need to examine both ethnic and socioeconomic contributions to smoking prevalence since these have important policy implications.

6.4 Intervening variables and linear logistic regression analyses

Both early smoking experimentation and parental attitudes were excluded after preliminary analyses revealed that, although statistically significantly associated with parental SES, they added little to the explanation of the association between parental SES and young adult smoking behaviour (particularly after accounting for the effects of parental smoking). As noted in Chapters 2 and 5, the use of early smoking experimentation as an explanation of later smoking is tautological; the fact that prior behaviour predicts later behaviour is not helpful for understanding causal processes, unless we can examine antecedents to that earlier behaviour. The finding that parental attitudes towards smoking explained very little of the SES and adolescent smoking association is consistent with the general conclusion reached by Conrad *et al.* (1992), in which measures of parental attitudes were found to be predictive of adolescent smoking in less than half the studies including such measures. Although, as discussed earlier, the measure of parental attitudes in the CHDS data was less than ideal – since it captured adolescent *perceptions* of parental attitudes rather than actual parental attitudes towards smoking. Additionally, although not reported in Chapter 5, parental attitudes, as measured by adolescent perceptions, were statistically significantly associated with parental smoking behaviour ($p < 0.0001$). Therefore a possible explanation for the finding in a number of studies that parental attitudes were predictive of adolescent smoking, is that this linkage is better understood as a function of the empirical linkages between parental attitudes and parental smoking behaviour.

The remaining intervening variables examined at the bivariate level were also found to be significantly associated with parental SES. This accorded with expectations since most of these associations have been reported in the studies reviewed in Chapter 2. However, the association between deviant behaviour and parental SES had not been documented in any of the literature reviewed in this thesis. Nevertheless, it was noted previously that such an association was likely, given that risk-taking behaviour among adults is commonly reported to be associated with SES; it might therefore be expected that a similar association would exist among their offspring. The statistically significant associations between parental SES and parental smoking, conduct disorder, affiliations with smoking peers and educational achievement also suggests that these variables were all plausible mechanisms in the pathway between parental SES and young adult smoking behaviour.

Turning now to the first intervening mechanism in the causal pathway, parental smoking, the results from the logistic regression analyses revealed that this mechanism explained a considerable proportion of the association between parental SES and young adult smoking outcomes at age 21. The odds of smoking at age 21 for those from the lowest SES group in comparison to those from the highest SES group reduced from 2.2 to 1.74 for ‘any smoking’, and from 2.42 to 1.96 for ‘daily smoking’. Furthermore, with only parental SES and parental smoking as explanatory variables under Model 1, it was apparent that *both parental SES and parental smoking* had *independent* effects on young adult smoking behaviour. This finding is consistent with that of Green and colleagues (1991).

Including conduct disorder under Model 2 greatly reduced the SES effect for both ‘any’ and ‘daily smoking’. Thus, deviant behaviour is not only a predictor of adolescent smoking in general, as was found to be the case in numerous studies (Burt *et al.* 2000; Chassin and Presson 1986; Collins *et al.* 1987; Conrad *et al.* 1992; Tyas and Pederson 1998), but it also helps account for the association between parental SES and adolescent smoking.

This effect was somewhat reduced, but still statistically significant, even after the inclusion of educational achievement under Model 3. Upon inclusion of affiliations with smoking peers under Model 4, however, the explanatory power of conduct disorder disappeared. The final results from the logistic regression analyses suggested that three variables in particular accounted for the association between parental SES and adolescent smoking: parental smoking at age one, educational achievement at ages eleven and twelve, and affiliations with smoking peers at age 15. The results from the log-linear analysis confirmed this explanation, as well as clarifying the structure of these pathways.

6.5 Pathways between parental SES and smoking at 21

6.5.1 Pathway one: via parental smoking

The first of the three pathways between parental SES at birth and young adult smoking was via parental smoking. Thus, the socioeconomic gradient in *parental* smoking creates an environment in which adolescents are differentially exposed to parental cigarette smoking according to socioeconomic background. This finding is generally consistent with much of the research indicating that parental smoking predicts adolescent smoking (Conrad *et al.* 1992; Tyas and Pederson 1998). As previously suggested (in Chapter 2), the mechanisms linking parental smoking to later smoking are likely to be access to tobacco (an important factor likely to contribute to higher rates of early smoking experimentation among lower SES youth), and the role-modelling effects of parental smoking (which make smoking more normative in lower SES families). Parental attitudes were not included in the log-linear model since preliminary regression analyses suggested that they added little to the explanation of the parental SES and adolescent smoking association. Nonetheless, they may also constitute part of the social class milieu that makes tobacco smoking more acceptable to youth growing up in lower SES families. Additionally, while only *parental* smoking was considered in the analysis, the smoking status of other family members, such as siblings and extended family (and possibly that of significant family friends), may also constitute part of the social class context that contributes to the SES gradient in adolescent smoking.

An important qualification to the above finding concerns the measure of parental smoking itself. Specifically, the measure reflects the smoking status of parents when the cohort participants were one year of age. For a number of reasons already discussed in Chapter 4 – to do with the temporal sequencing of intervening mechanisms and changes to family constitution over time – changes in parental smoking status were not explored. Thus, the analysis cannot assess the impact of cumulative exposure to parental smoking on adolescent smoking, nor assess the possibility that parental smoking cessation might effect the smoking behaviour of their children. A more comprehensive understanding of the effects of parental smoking on adolescent smoking would be gained by further research in this area. Such research would be of benefit to future health promotion and intervention programmes aimed at reducing smoking amongst youth. However, Linzer (cited in Bauman 1990) reported that the smoking behaviour of parents at birth was predictive of adolescent smoking, regardless of the fact that some of these parents had later given up smoking. This suggests that the measure used in the analysis of the CHDS data, that is, parental smoking at age one, is unlikely to have contributed to any major bias in the results.

6.5.2 Pathway two: via parental smoking to affiliations with smoking peers

The finding in this analysis, that parental smoking leads to adolescent smoking via the mechanism of affiliations with smoking peers is a result that has not been reported previously except by Fergusson (1995). Both parental smoking and affiliations with smoking peers have been conceptualised within the social epidemiology of adolescent smoking as two independent determinants of adolescent smoking (Bauman *et al.* 2001; Chassin and Presson 1986; Glendinning *et al.* 1994; Green *et al.* 1991). Such research, limited as it is, has mostly concentrated on which of these two factors is the more important determinant of adolescent smoking. Notably, Chassin *et al.* (1986), in their comparison of cross-sectional and longitudinal data for the purpose of determining the relative effects of parental and peer smoking on adolescent smoking, concluded that the cross-sectional study design produced results consistent with an increase in the influence of peer smoking with age, and a corresponding decrease in the influence of parental smoking. The longitudinal study design, by contrast, produced results suggesting that parental and peer smoking were of equal influence. While this may be pertinent in identifying determinants of smoking, the analysis in this thesis of the *structure* of pathways between parental SES and adolescent smoking suggested a causal pathway in which parental smoking contributes to affiliations being formed with smoking peers, which, in turn, raises the likelihood of smoking at age 21. While it is not immediately apparent how parental smoking leads to affiliations with smoking peers, one possible explanation may be that parents who smoke also socialise with other adults who smoke, and this in turn contributes to the association of like-minded and susceptible offspring. Another possible explanation may be that parental smoking leads to offspring smoking prior to age 15, which, in turn, leads to affiliations with smoking peers at age 15.

The extent to which peer influence, or differential association, contributes to the relationship between parental smoking, affiliations with smoking peers, and subsequent smoking, remains unclear. In theoretical terms, however, both mechanisms are plausible. Although this issue remains unanswered in the preceding analysis, in the case of *determinants* of adolescent smoking *rather* than the SES gradient in adolescent smoking, earlier analyses of the CHDS has shown both peer influence and differential association to contribute to adolescent smoking (Fergusson *et al.* 1995).

The log-linear analysis further revealed that deviant behaviour, as measured by conduct disorder, was not implicated as a factor contributing to affiliations with smoking peers, despite the suggestion of this as a potential pathway in Chapter 2. Nor was there any evidence of a direct path from deviant behaviour to smoking at age 21, contrary to much of the empirical evidence in this area (Burt *et al.* 2000; Collins *et al.* 1987; Conrad *et al.* 1992; Tyas and Pederson 1998). In other words, this often reported empirical linkage does not appear to account for the parental SES and adolescent smoking association in the CHDS cohort.

A slightly unexpected outcome was the lack of a significant pathway between parental SES and deviant behaviour (conduct disorder), given that the initial bivariate analysis revealed that the two were associated. The absence of any statistically significant link between parental SES and affiliations with smoking peers also revealed that this was not a direct pathway. Thus, parental social class itself does not lead to affiliations with smoking peers. Rather, it is through the mechanism of parental smoking that young people from lower SES groups come to affiliate with smoking peers in adolescence.

These findings are subject to the validity of the measures of parental smoking and of affiliations with smoking peers. The possible limitations of the measure of parental smoking were discussed previously. The measure of affiliations with smoking peers at age 15 may also have some limitations, as noted in Chapter 4, in that young people tend to overestimate the prevalence of smoking amongst their peers. Even if this is the case, it is unlikely that this will have substantially altered the results, unless the overestimation of peer smoking prevalence is associated in some way with parental SES.

6.5.3 Pathway three: via educational achievement

The mechanism of educational achievement as a pathway between parental SES and smoking at age 21 is somewhat more complex. The chi-square results for the pair-wise associations between parental SES and educational achievement, and between educational achievement and smoking at age 21, were highly significant, suggesting that parental SES determines educational achievement – which in turn leads to smoking at age 21.

The association between parental SES and educational achievement is consistent with previous research internationally and within New Zealand (Cockerham *et al.* 1997; Lauder and Hughes 1990; Lauder *et al.* 1992; Lauder *et al.* 1985; Robinson 1982). Sociological literature has suggested various mechanisms linking parental social class to the educational achievement of children, many of which have been discussed in more detail in Chapter 2. Briefly, parental and childhood educational aspirations and expectations, together with the middle-class focus of the school system and curriculum and potential class bias by teachers, help to reproduce class inequalities in educational achievement (Ball 1986; Hurrell 1995; Mitchell and McManus 1982; Robinson 1982).

The significant pair-wise association between educational achievement and smoking at age 21 is also consistent with previous research in this area (Conrad *et al.* 1992; Glendinning *et al.* 1994; Tyas and Pederson 1998; U.S.DHHS 1994, 1998). Potential mechanisms linking educational achievement to adolescent smoking noted earlier include: the extent of the knowledge held by young people of the health consequences of smoking (which may well vary according to educational achievement); the possible effects of educational achievement on peer selection; and the possible effects of educational achievement on deviant behaviour, or conversely, the effects of deviance on educational achievement. Absence of a measure of respondents' knowledge of the health effects of smoking precluded analyses of the relationship between this variable and educational achievement (and thus its possible contribution to later smoking). In any case, a large number of studies have suggested that knowledge of the health effects of smoking is not an important determinant of adolescent smoking (Miller and Slap 1989; U.S.DHHS 1989), despite some evidence that such knowledge varies according to educational level (Brownson *et al.* 1992). The second potential mechanism, the possible impact of educational achievement on affiliations with smoking peers, must be ruled out in the CHDS as there were no significant pair-wise associations between educational achievement and affiliations with smoking peers. This is not to say that educational achievement is not a basis for friendship formation amongst adolescents, only that it is not a basis for affiliations with peers of the same smoking status.

The log-linear analyses also revealed the role of deviant behaviour in the SES and smoking association. Both the associations – between parental SES and deviant behaviour, and between deviant behaviour and smoking at

21 – were *non-significant*. Therefore, the association between deviant behaviour and young adult smoking, as an explanation of the relationship of parental SES to adolescent smoking, appears to be spurious and due to the empirical linkages between deviant behaviour and educational achievement. Theoretically, as suggested in Chapter 2, the causal direction of the association between educational achievement and deviant behaviour may go either way. With deviant behaviour measured at age eight, and educational achievement at ages eleven and twelve, the results from the CHDS suggest that the *direction* of this association is one where deviant behaviour leads to lower educational achievement. Miech *et al.* (1999), in their analysis of longitudinal data, found that conduct disorder did in fact predict educational failure. Nonetheless, it is still possible, had the analysis included a measure of education prior to age eight, or a measure of deviant behaviour after age twelve, that the converse situation may have been confirmed (that is, lower educational achievement leading to deviant behaviour).

The finding that education is an important mechanism linking parental SES to young adult smoking outcomes is also significant in light of the results reported by Glendinning *et al.* (1994), in which adolescent smoking was found to be more closely related to adolescent social class of destination (as indicated by adolescent educational attainment) than to their social class of origin (parental social class). While the relative strength of the associations between educational achievement and parental social class with respondents' smoking were not assessed in this thesis, the log-linear analyses did reveal educational achievement and parental social class to be causally related. Parental social class was found to significantly contribute to educational achievement, which in turn, was linked to smoking at age 21. Lynch and colleagues have noted that “the association between adult SES and health behaviour exists, at least in part, because adult SES destinations depend on childhood SES origins” (Lynch *et al.* 1997:816). As to whether educational achievement has a moderating influence on other risk factors for smoking – as was found to be the case in the research by Glendinning *et al.* (1994) – in the CHDS this does not appear to be the case, since there was no significant pair-wise association between educational achievement and peer affiliations or parental smoking. The empirical links between education and smoking at 21, and between education and social class, therefore, suggest that education may be an important mechanism through which class-based health-related behaviours are transmitted from one generation to the next. Add to this the influence of parental smoking on affiliations with smoking peers, and one has considerable insight into how the socioeconomic gradient in smoking behaviour comes to be reproduced in the next generation.

The above conclusions concerning the educational pathway to smoking amongst young adults in the CHDS are necessarily subject to the validity of the educational measure utilised. Although there is no reason to doubt the adequacy of the GPA measure, an analysis of the degree of correlation between the GPA and the other measures of educational achievement available in the CHDS would clarify the nature of this variable.

6.6 Theoretical implications

The association of the investigated social-epidemiological risk factors for adolescent smoking with parental social class supports the notion that young peoples' smoking is partly a result of social class variation in exposure to particular risk factors. Specifically, the social class gradient in parental smoking explains much of the SES gradient in adolescent smoking, and much of this is likely to be due to the role modelling influence of parental behaviour on their offspring. The empirical linkage between parental smoking and affiliations with smoking peers suggests that processes much more complex than those typically conceptualised by current perspectives in social epidemiology may be operating to reproduce social class patterns in health-related behaviour. A more straightforward explanation of the role of peer affiliations in the pathways between parental SES and adolescent smoking, for instance, would have been one where peer affiliations were determined by parental SES. However, as previously noted, affiliations with smoking peers at age 15 may be partly a consequence of earlier smoking initiation, which might explain why parental smoking is linked with affiliations with smoking peers.

The role of educational achievement in contributing to young adult smoking also suggests that education is an important mechanism for the reproduction of class-based behaviours. If educational achievement can be taken as indicative of social class of destination, then it appears that young people in the CHDS, like those in the studies conducted by Glendinning *et al.* (1994) and Karvonen *et al.* (1999), adopt behaviours in a manner consistent with their social class of destination. However, the empirical evidence from the CHDS and other national and international literature has shown that educational attainment itself is significantly determined by social class of origin (Cockerham *et al.* 1997; Lauder and Hughes 1990; Lauder *et al.* 1992; Lauder *et al.* 1985; Robinson 1982). It is in this manner that one can begin to see, as the theory suggests, how class-related behaviours are both a consequence of, and a means to ensure, the reproduction of class hierarchies.

The extent to which young people can be thought of as freely exercising individual choice in their decision to initiate or refrain from smoking must be seen in light of their relative exposure to the social-epidemiological risk factors for cigarette smoking. Certainly, with parental smoking and educational achievement as the primary mechanisms accounting for the higher prevalence of smoking amongst youth from lower SES backgrounds, one can begin to understand how factors associated with the social structure and beyond the young person's control exert important influences on behavioural 'choices'. According to Lynch and colleagues:

One model of health behaviour is strongly based on the premise that adult health behaviours are largely individual phenomenon which reflect some process involving free choice. This approach views unhealthy behaviours as the consequence of poor lifestyle management Epidemiology confirmed that many poor health outcomes were associated with the daily conduct of peoples' lives The other model of health behaviour agrees that while individuals make choices about how they act, those choices are situated within economic, historical, family, cultural and political contexts. According to this view, these contexts exert important influences on both the processes of choice and the types of behavioural options which are available and, indeed appropriate. A number of authors have argued that decontextualising behaviour from this real world setting obscures its socioeconomic production and encourages blaming the victims of inequality for their unhealthy lifestyles. According to this approach, evidence that health behaviours are differentially distributed by SES should be viewed in a lifecourse perspective, as the cumulative responses of different classes of people to conditions imposed by social structure (Lynch *et al.* 1997: 810).

6.7 Strengths and limitations of data and analysis

The cohort of young people in the CHDS may not necessarily be representative of similarly-aged young people in the New Zealand population. As only children born in the Christchurch urban region during 1977 were eligible for participation in the CHDS, children born in rural hospitals were excluded from the outset. If those born in rural areas are different to urban dwellers in terms of their socioeconomic or smoking characteristics, then the generalisability of the results may be compromised to a degree. There is some anecdotal evidence to suggest that rural dwellers have lower rates of smoking than their urban counterparts (Hay and Foster 1984) and this could be partly due to a greater proportion of higher SES families in rural areas.

Furthermore, the recruitment of families into the CHDS did not occur over a full year, and if children born to families during particular months (specifically those born between April and August) are different in terms of their socioeconomic characteristics to children born in other months of the year, then this may also compromise the generalisability of the findings. One method for assessing whether the sample population is representative of the wider population is to compare baseline demographic characteristics in the sample with those of the wider population. While such a comparison was not possible in this thesis, it would have been useful for determining whether the findings from the CHDS can be extrapolated to the wider New Zealand population. It is also important to remember that the results from the analysis in this thesis pertain to a specific cohort, those born in Christchurch in 1977, therefore the findings may not be indicative of smoking acquisition and socioeconomic processes in subsequent (or earlier) cohorts.

Another limitation of the preceding analysis concerns the potential for bias resulting from missing data on some of the key intervening variables. As noted in Chapter 4, 10% of data on parental attitudes was missing, and between 5-9% of data was missing for the variables of early smoking experimentation, deviant behaviour, peer smoking and educational achievement. Bias may result to the extent that such missing information was not randomly distributed. However, in absence of a reliable method for assessing the randomness of missing data, this potential source of bias could not be determined.

Aside from the limitations already identified, there is one final limitation specific to the data analysis adopted in this thesis which, although minor, deserves consideration. The assessment of statistical significance in this thesis has relied heavily on the use of *p* values – defined at the conventional significance level of <0.05 . While acknowledging both that this may be considered by some as a rather a crude method for determining substantive significance (Goodman 2001), and that other methods have been proposed (Poole 2001; Weinberg 2001), a consideration of alternative methods for determining substantive and statistical significance was outside the scope of this thesis.

Despite the above-mentioned limitations, the data source and analysis used in this thesis has considerable strengths. The greatest of these lies in the longitudinal nature of the data. Compared to cross-sectional studies – limited by design to the analysis of correlation between factors – the longitudinal design of the CHDS has facilitated the time-dependent causal modelling approach adopted in this thesis. This approach has been informed by both the empirical evidence from the social epidemiology of adolescent smoking and the theoretical reasoning underpinning sociological theories on social stratification. Furthermore, the statistical methods

utilised in the analysis, in particular the use of log-linear modelling to determine the structure of the pathways between parental SES and young adult smoking outcomes, has added substantive value to the analysis. Finally, adequate sample size and low rates of sample attrition suggest that the results are likely to be reasonably robust and applicable to similarly aged cohorts in urban centres in New Zealand.

Chapter 7

Public health implications and conclusion

7.1 Introduction

Smoking is a critical health risk behaviour amenable to change. Its association with SES in both adults and their offspring explains a considerable proportion of SES inequalities in health observed in later life. An estimate based upon mortality data from men aged 21-59, suggests that as much as 28% of the social class gradient in mortality in New Zealand was attributable to cigarette smoking (Zhang *et al.* 2001). Such an estimate does indicate the potential benefit of addressing the SES gradient in smoking. Additionally, reducing the SES gradient in smoking will also substantially lower the overall rates of premature death and disability in the total population. Furthermore, for Maori, addressing the SES differential in smoking is likely to reduce their higher death rates from smoking attributable diseases to something closer to that of non-Maori New Zealanders.

7.2 Potential points for intervention

To date, very little is understood about the underlying processes which cause the socioeconomic differential in cigarette smoking. This hampers health promotion and public health efforts to reduce smoking prevalence in the total population. The analysis presented in this thesis, particularly the identification of causal pathways between parental SES and offspring smoking, suggests specific points for intervention, the most critical of these being parental smoking and educational achievement. Affiliations with smoking peers is another potential point for intervention, although being further along the pathway it is perhaps less important.

7.2.1 Parental smoking

In the temporal sequencing of events along the causal pathway from parental SES to adolescent smoking, parental smoking is the first mechanism encountered. Health intervention and promotion programmes need to acknowledge the influential role of parental smoking on the smoking acquisition process of young people. Reducing parental smoking, although a difficult public health goal, will result in a reduction in young peoples' access to tobacco – and thus limit their opportunities for experimentation, reduce the social acceptability of smoking, and provide more positive health-behaviour role models for youth. Additionally, exposure to environmental tobacco smoke will be further limited as a result of any decrease in parental smoking. Given also the empirical linkage between parental smoking and affiliations with smoking peers in adolescence, a reduction in parental smoking is also likely to result in a reduction in affiliations with smoking peers.

Furthermore, if the number of years of exposure to parental smoking has a dose-response effect on the chances of their offspring smoking, interventions targeted at parents of younger children will result in a greater public health benefit than those targeted at parents of older children. Of course, this needs to be balanced against the

potential benefits to parents themselves of the earliest possible smoking cessation, given the dose response association between smoking and adverse health outcomes. Ideally, programmes targeted to young adults, especially those of lower SES, prior to family formation, will have the most health benefit for their own and subsequent generations.

7.2.2 Educational achievement

In the first instance, low levels of educational achievement amongst youth may help health promoters identify those most at risk of tobacco addiction, especially if their parents also smoke. As noted earlier, exactly why education is linked with smoking is not entirely clear, although it is unlikely to be accounted for by differences in knowledge of the health effects of smoking. The results from this thesis provide some support for the social mobility explanation for the adoption of health-related behaviours – namely that young people adopt behaviours consistent with their future position in the social hierarchy. Although, it is important to note, that lower educational achievement appears to be largely a consequence of low socioeconomic status.

Given that low education is associated with a wide range of health-related behaviours, a strong argument exists for a greater focus on, and resource allocation to, under-achieving youth. If, as suspected, deviant behaviour has a particularly disqualifying impact on educational achievement, then interventions to address deviant behaviour are also indicated.

7.3 Reducing socioeconomic inequalities

Nevertheless, while the above-mentioned avenues for intervention may be considered important “... a concern with mechanisms in health inequalities research can lead to a focus on technical interventions along causal pathways, with the roots of health inequalities, wider social inequalities, being ignored” (Nazroo 1998). Although some perspectives do hold that one’s socioeconomic position is largely a consequence of individual effort, ability or personal traits, the low rates of social mobility between social classes from one generation to the next, and the association between parental social class and educational achievement, highlight the presence of structural factors in determining one’s position in the socioeconomic order. Thus, broader social and economic policies to improve the socioeconomic position of those at the bottom relative to those at the top of the social hierarchy may be the most viable solution to changing health-related behaviours associated with socioeconomic status. Moreover, given that the higher rates of smoking among Maori were found to be attributable to their over-representation among lower SES groups, broad social and economic policies to improve the socioeconomic position of Maori are also indicated.

7.4 Conclusion

This thesis examined smoking behaviour not only because it is an amenable health risk behaviour – associated with higher morbidity and mortality from a wide range of diseases – but because of its contribution to socioeconomic inequalities in health outcomes. The association of adult cigarette smoking with socioeconomic status, although a relatively recent feature of ‘Western’ countries, has been reported in numerous studies. However, considerably less is known about the causal processes underlying this phenomenon and potential points for intervention. Given that cigarette smoking is a habit primarily established during adolescence, untangling the determinants of adolescent smoking has become an important area for research in social epidemiology. Of particular interest to both our understanding of socioeconomic inequalities in health, and the contribution of health risk behaviours (such as smoking) to such inequalities, was whether the socioeconomic gradient in adult smoking behaviour has been replicated in the next generation of young people. This thesis found that this was indeed the case – the socioeconomic pattern in adolescent smoking mirrors that found amongst the adult population, and this pattern is similar for both males and females and Maori and non-Maori. Notably, for Maori the higher prevalence of smoking was found to be due to their over-representation among lower SES groups.

Upon examining the key potential mechanisms implicated in the causal pathway between parental SES and adolescent smoking – specifically, parental smoking, parental attitudes towards smoking, early smoking experimentation, affiliations with smoking peers, deviant behaviour, and educational achievement – only three were found to account for the association between parental SES and their offspring smoking. These were: parental smoking, educational achievement, and affiliations with smoking peers. Thus, the socioeconomic gradient in young adult smoking prevalence appears to be due to a socioeconomic gradient in educational achievement and in parental smoking, which itself, is directly and indirectly (via affiliations with smoking peers) linked to smoking behaviour at age 21. These pathways suggest that parental smoking and educational achievement in particular, and affiliations with smoking peers to a lesser extent, are critical areas for intervention.

Successful intervention in these areas are likely to have a positive impact on reducing smoking-attributable socioeconomic inequalities in health outcomes in later life. Such interventions may be somewhat limited, however, as mechanisms in the causal pathways between parental SES and young adult smoking ultimately stem from the underlying socioeconomic inequalities themselves. Much broader social and economic policies to reduce these social inequalities may therefore hold the most promise as avenues through which to address health behaviours associated with socioeconomic status.

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