

More specifically of interest here, the geographical neighbourhood may therefore potentially act as a social group where health-related social practices are enacted. However, again, the type of neighbourhood may be important in determining how this might happen. If neighbourhoods vary in the strength of their integration and regulation, the effect of the health-related social practices of other residents will also vary. So rather than assuming that health-related social practices observed by residents in different neighbourhoods are equally influential, it may be that characteristics of the neighbourhood could affect the transmission of the practices, with health consequences.

A third factor also needs to be integrated into the above pathways, that of the importance of individual characteristics. While Durkheim focused on societal organization, he asks us to look both ways - to the nature of social life, and to the responses of individuals. Obviously, neighbourhood or social group characteristics cannot provide the full picture in explaining health outcomes: individual and society wide factors are also important. Factors such as gender and psychiatric health status appear to be an important part of understanding the dynamic between neighbourhood and health. In other words, the conceptual model needs to be able to capture how the levels interact with each other.

For Durkheim, the importance of 'psychological' factors lies in the extent to which people might become more vulnerable to the various currents or states around them. He proposed that in order to understand variations in the rate of suicide across time (and perhaps places) we need to consider how the environmental social context might provide a particular imbalance to which some individuals may be more sensitive;

*"If, in a given moral environment, in the same religious faith or in the same body of troops or in the same occupation, certain individuals are affected and certain others not, this is undoubtedly, in great part, because the former's*

mental constitution, as elaborated by nature and events, offers less resistance to the suicidogenetic current." (Durkheim, 1951, p. 323).

Both pathways have been examined in the health and wellbeing literature in recent times, but not necessarily with explicit reference to Durkheim's thesis, or the specific mechanisms discussed above. Despite the many, varied and contradictory ways in which Durkheim's work has been interpreted there is an interesting body of work that has translated the mechanisms discussed above. Semantics aside, I will discuss the literature which uses the mechanisms: firstly the work examining the relationship between integration and regulation and health outcomes (at various scales); and secondly work which specifically looks at the social context of health-related practices.

#### *4.2.1.1 'Integration and Regulation' Mechanism*

Durkheim proposed that either a paucity or excess of integration and regulation would be detrimental to the wellbeing of individuals (Taylor and Ashworth, 1987). The integration and regulation of social groups might be operating on individual health in three ways. Firstly, levels of integration and regulation, or social cohesion in a group have been regarded as a source of 'social support' (Andren and Rosenqvist, 1987), referred to as one of the social capital resources in Bourdieu's terms (Carpiano, 2006). Decreased levels of support have been regarded as detrimental to wellbeing and health. Secondly and closely related, the level of integration and regulation in a group has been seen as a modifier of other stressors. Thirdly, the degree of integration and regulation has been related to some form of monitoring, either formal or informal.

The benefits to membership of social groups have long been discussed in the health literature, notably in the social cohesion and capital body of work. Participation in organizations has been a common measure used in the social capital literature: for

example, measures of volunteering (Blakely et al., 2006). Better health has been associated with participation (either by volunteering or membership) in organizations and social networks (Sundquist et al., 2004, Weitzman and Chen, 2005), although the evidence has sometimes been contradictory with poorer health being associated with membership of social groups (Siahpush and Singh, 1999, Maycock and Howat, 2007).

Research examining social ties between people and within groups has emphasized the need for both strong (bonding) and weak (bridging and linking) ties (Li et al., 2005). Each provides individuals and groups with potential benefits, but also downsides. Cattell (2001) summarized the benefit of strong ties as providing support and a sense of being understood, and the benefit of weak ties as access to resources outside of one's immediate circle. Certainly psychosocial benefits of feeling connected to a group have been observed (Young et al., 2004), as has the benefit of receiving support from groups (Halpern, 1995). Balkundi noted the benefit not only to the individual, but also to the group of a balance between the two (which in turn will benefit the individual) (Balkundi et al., 2007).

To date, much of the social fragmentation literature has focused on mental illness outcomes, specifically suicide and psychiatric outcomes. Physical illness outcomes have also been examined but have provided reasonably consistent evidence against social fragmentation at the neighbourhood level being an important contributory factor. It could be argued that the focus on variations in illness has narrowed the potential for understanding potential mechanisms that might be at play. There may be a clearer pathway between mental health or wellbeing and the 'integration and regulation' mechanism.

Distinguishing between illness and health is problematic. It has been argued that they are two separate, but related constructs (Halpern, 1995). For example, misery or

distress can have different pathways from illness. Halpern concluded that the style of coping mechanisms of an individual were an important means of distinguishing between the two. Pathological or maladaptive processes were more likely to describe illness than misery or distress (Halpern, 1995). Durkheim made the argument that certain currents could create psychological distress (Taylor and Ashworth, 1987), because they failed to balance the competing needs of the collective and the individual. Therefore it is worth considering evidence in the neighbourhoods and health literature for a relationship between health and the specific 'integration and regulation' mechanism proposed above.

There does appear to be some support for the 'integration and regulation' mechanism on mental health in the literature. Halpern (1995) suggested that social groups can provide support against stressful situations such as unemployment (the 'stress buffering' mechanism), as well as being of benefit generally (a 'main effect' mechanism). Mulvaney-Day et al (2007) found that self-reported mental health was predicted by perceived levels of family cohesion. Interestingly, in their study peer support was not a significant predictor once the more proximal family measures were included. Not unexpectedly, neither was the even more distal neighbourhood social setting. While the results failed to demonstrate that neighbourhoods were significant sources of support, their findings demonstrated that social groups such as families were a source of support, even when a number of other individual factors were taken into account (Mulvaney-Day et al., 2007).

Fone et al (2007) investigated the relationship between mental health and social cohesion at the neighbourhood level. They found that their measure of cohesion (aggregated individual perceptions of the neighbourhood) was a statistically significant predictor of mental health (the mental health scale of the Short Form -36 measure), even after controlling for individual cohesion levels (and other socioeconomic confounders) and neighbourhood deprivation (Fone et al., 2007).

Furthermore, they found that neighbourhood cohesion modified the association of neighbourhood income and mental health (but not the more general deprivation measure). Cohesion was protective for mental health against living in an income deprived area (Fone et al., 2007). The authors suggest that high levels of cohesion in a neighbourhood may be supportive via processes such as affective support and regulation over problem behaviours (Fone et al., 2007).

A number of authors have used affiliation with social groups as a way of measuring integration and regulation type mechanisms. The scale of “societies” into which an individual can be integrated varies widely across research, making comparisons difficult. Durkheim himself referred to both “society” at the national or more global level when he referred to the effects of the French Revolution on national level society generally. But he also referred to very small scale societies such as family and marriage. For the sake of clarity and consistency I will refer to the unit of analysis in the research discussed below as the ‘social group’.

A common approach has been to compare rates of affiliation with various social groups across time or space, and assess associations with variation in outcome rates. Commonly the scale of interest in the research is at the broader national societal level. Poppel and Day (1996) built on Durkheim’s original analysis of the rates of suicide across Protestant and Catholic religious groups by examining for potential bias from differences in the way that suicide that could account for the associations observed by Durkheim. An important finding was that there was strong evidence that Catholic deaths were less likely to be reported as suicide. When potentially all external deaths were taken into account, including those that may have been misclassified, there was little difference between the two religions.

One potential interpretation of the results not discussed by the authors is the strength of the norms, or the moral force, that may underlie the differences in the coding of

the suicide mortality records. The authors argued that the strong social and religious sanctions that surrounded suicide in the Catholic population would have led them to take measures to avoid having a relative's death classified as suicide (van Poppel and Day, 1996). However, it is difficult to know the extent to which this was due to differences in the degree of regulation and integration of individuals within the different religious social group (in support of Durkheim), or because of different beliefs and cosmologies around suicide between the two religious groups (in contrast to Durkheim who claimed them not to be the determining factor).

Using data from more recent times, Dervic et al (2004) examined the effect of religious affiliation per se on suicide behaviours. They examined the hypothesis that not being affiliated to a religious social group would result in individuals being relatively poorly integrated and regulated, compared with those who were affiliated. They observed a protective effect of affiliation on both the patients' history for suicide attempts and recent ideation. If we can assume that affiliation is a proxy for participation in a religious social group, their findings lend support to Durkheim's theory that participation is protective against suicide attempts and ideation because the individual benefits from the integration and regulation of the group (Dervic et al., 2004).

As well as looking at the relationship between affiliation and suicide attempts as an test of Durkheim's theory, Dervic et al (2004) were also interested in other differences that may be part of a clustering of factors that potentially mediate the relationship. Firstly, they observed differences in measures of individual aggression and compulsiveness, with unaffiliated patients demonstrating higher levels. While the authors do not link this back to Durkheim's theory, I would argue that the association showed support for his proposal that being integrated and regulated by a group helps to provide normative limits to "uncontrollable passions", in this case aggression and compulsiveness.

Fernquist (2007) examined suicide rates across eight countries and eight time periods and examined their relationship with measures of integration and regulation.

Following Durkheim's example, he makes the case for divorce and unemployment acting as indicators of individuals being affiliated with marital and occupation social groups (and presumably therefore integrated and regulated by them). The author found that rates of suicides in the 64 cases were associated with variations in the aggregations of individual affiliation with various societal level institutions, even after controlling for levels of depression and alcoholism (Fernquist, 2007), suggesting that having a society where members are integrated into either societal institutions or social groups may be protective.

The difficult political transition for the Eastern European countries in recent times has been used by researchers to test some of Durkheim's proposed mechanisms, with the predominant focus being on anomie. Makinen (2000) sought evidence for anomic suicidogenic currents arising from the rapid social change, whereby societal organisation was less able to provide a protective degree of regulation to individuals resulting in increased rates of suicide. While there were changes in the rates of suicide within countries around the transition period, he did not find evidence that this was related to changes in measures of social disorganisation, economic development, the political structures, and so on. While the findings did not support Durkheim's theory, Makinen did point out that this may be due to intermediary structures, such as specific national cultures, that were unable to be measured in his analyses. However, he argued in support of Durkheim's theory that there was strong evidence that context matters for individual suicidal propensity: suicide rates could not be understood by focusing only on individual level factors (Makinen, 2000).

The extent to which integration and regulation of a neighbourhood setting acts as a positive social resource for mental illness is not always clear cut. Reviews of literature on social capital and mental health and illness have generally not been able

to establish a clear pattern of association. In part this was due to multiple conceptualisations and definitions of the exposure and outcomes (Almedom, 2005, De Silva et al., 2005, Whitley and McKenzie, 2005). But it could also be a reflection of the complexity of the relationship.

Individual and other contextual characteristics appear to be an important factor in understanding how integration and regulation might be related to mental health (Almedom, 2005). It has been suggested, for example, life course stage may be an important factor. Almedom argued that the mental well being of children and adolescents may benefit from more cohesive settings because they provide higher levels of informal social control that allows them to feel more secure in their local environment (Almedom, 2005). Gender also appears to be an important factor to understanding the importance of integration and regulation for mental health (Healy et al., 2007, Kavanagh et al., 2006, Matheson et al., 2006, Stafford et al., 2005). Almedom concluded that variations in rates of depression between men and women can be related to the gendered ways that social support is given and received in their social networks (Almedom, 2005).

The role played by integration and regulation in the mental well being of residents may be related to other contextual factors. Whitley and Prince (2005) highlighted the importance of access to community social resources for the mental health of some residents. They found that fear of crime was a major factor in inhibiting particularly older people's participation in the local community, with consequences for their mental health and access to socially supportive networks (Whitley and Prince, 2005). Schulz et al (2006) found that perceived access to social support in part mediated the stresses for women of living in highly disadvantaged neighbourhoods. Furthermore, they found that length of residence appeared to be part of the explanation as it predicted the level of perceived social support, suggesting that the longer residing residents had better established sources of support.



Some authors have discussed the deleterious health consequences for residents of living in homogenous neighbourhoods that don't 'fit' with their individual characteristics. It has variously been termed the 'group density' effect (Halpern, 1995), 'ethnic density' (Neeleman et al., 2001, Moon and Barnett, 2003), or 'person-environment fit' (Fagg et al., 2008). For example, Neeleman et al examined the mental health consequences of ethnic density living in a neighbourhood where an individual's ethnic identity was dissimilar to their fellow neighbours, finding that poorer mental health outcomes were associated with living in a neighbourhood where a high proportion of other residents were of a different ethnicity (Neeleman et al., 2001). The findings offer an interesting insight into the complexity of the pathways between neighbourhood segregation and health, suggesting that both homogeneity and diversity in neighbourhood composition may have positive and negative effects. The authors suggest that the effect of integration and regulation on mental health was therefore modified by factors which may hinder the ability of a neighbourhood and the individual to be a part of the group. Returning to Carpiano's point, as well as examining the social resources that are available, it is also necessary to understand factors such as ethnicity which determine individual access to them (Carpiano, 2006).

In particular, there has been some discussion in the literature of living in a closely integrative and regulated neighbourhood that is under strain from other factors such as deprivation. Ross et al (2000) argued that in highly disordered neighbourhoods, strong social ties were not able to mitigate the effect of the neighbourhood disorder on psychological wellbeing. They also suggested that the inability to leave a neighbourhood contributed to stress levels (Ross et al., 2000). Caughy et al (2003) reported a similar relationship between deprivation and parent's attachment to the neighbourhood. They found that the children of families living in deprived neighbourhoods were more likely to have behavioural problems if their mothers reported high levels of neighbourhood attachment than highly attached mothers

living in less deprived neighbourhoods (Caughy et al., 2003). Thus there may be a limited ability of social groups to protect residents from neighbourhood stressors.

On the other hand, other researchers have found that social capital could potentially ameliorate the effects of deprivation. Van der Linden et al (2003), for example found that children's use of mental health services was higher for those living in more deprived neighbourhoods. The association was somewhat modified by the degree of social capital of their neighbourhood, with a higher level of capital reducing the effect of deprivation (van der Linden et al., 2003). Such results highlight the importance of examining interactions between neighbourhood factors as well as their relative or independent contribution to health.

As discussed briefly above, Durkheim did not appear to be suggesting that the currents within a social group were necessarily the cause of mental illness. He explicitly recognised that, in his terms, there will always be a given prevalence of "psychological factors" in a population (Durkheim, 1951). His point was that the individual status made that person more or less susceptible to the currents. Therefore being part of a pathologically imbalanced social group could make matters worse, or may trigger the presentation of illness outcomes in a predisposed person, but they were not causative of the underlying condition. The pathway between mental illness and 'integration and regulation' mechanism may therefore be less direct and harder to observe in the absence of very rich data on individual factors too.

It has been suggested that types of neighbourhoods vary in their ability to regulate patients with mental illness. These studies did not suggest that the type of neighbourhood 'caused' the level of illness, but rather that the help available to residents that were mentally unwell was related to the likelihood of them receiving care. Gleeson (1998) was able to highlight that the provision of mental illness services is not evenly distributed by areas. Local access to services may be an important

means of providing a more formal regulating influence on people with mental illness.

Drukker (2004) highlighted the potential role of surveillance, or informal social control for mental health outcomes. In these studies, variations in use of health services, for example, may occur because some neighbourhoods are less able to provide informal social controls on health behaviours, or provide monitoring of patients' current state of health (Drukker et al., 2004). It may be that in more highly fragmented neighbourhoods, vulnerable residents might be more likely to slip between the cracks. Another possibility is that vulnerable people may selectively drift to live in highly fragmented areas for a number of reasons {Evans, 2004 #1321, for example, the increased anonymity due to decreased surveillance.

Unfortunately, there was insufficient reliable data on mental illness variation and mental health service access or utilization to explore either of the above pathways these avenues for this thesis. Understanding more about how neighbourhoods provide a more or less protective environment for people vulnerable to the suicidogenetic currents would certainly be an interesting test of Durkheim's theories.

The literature discussed above has suggested that the mental health of individuals may be sensitive to the 'integration and regulation' mechanism. Membership of social groups (of which the neighbourhood could be one) appears to be a means of accessing social resources that are important for health. It also appears that the type of social group is important for understanding what kind of social resources may be available to members. Other factors, such as individual characteristics, are important in understanding how important such social resources are to wellbeing. The various ways in which NeighFrag and mental health may be related will be explored empirically in Chapter Six.

#### *4.2.1.2 Transmission of Health-related Social Practices*

As discussed in the introductory chapter, individual smoking data was available for use in analyses for this thesis. The discussion in the first section of the chapter has established a theoretical framework which suggests how a health practice such as smoking might be related to neighbourhood fragmentation. In health research, activities such as smoking are commonly regarded as individual practices that are sensitive to their social context (discussed further below) and could therefore be of use in investigating this mechanism. The degree of transmission of social practices within a neighbourhood can be investigated by observing how smoking practices of the individual are related to those in their immediate neighbourhood environment. Less easy to observe is the richer picture of how the multiple layers of influence interact with the neighbourhood level to affect individual smoking practices. In the absence of rich data sources, the focus here remains on transmission within the neighbourhood and the individual's response to local practices.

Smoking is an individual activity that occurs within a social context {Turner, 2006 #1147; Frohlich, 2002 #297}. The contribution of smoking to morbidity and mortality, and to health inequalities in New Zealand is well documented (Hill et al., 2005, Barnett et al., 2004). It has consistently been demonstrated that individual risk factors such as socioeconomic status, ethnicity, psychological characteristics etc, are significant predictors of tobacco use (Siahpush et al., 2006). However, research has also demonstrated that individuals are nested within multiple layers of influence (Wilcox, 2003), ranging from the proximal, family and peers to more distal factors such as national norms and regulations. Wilcox goes on to suggest that "These contextual effects can impact directly on both group and individual level behaviour (e.g. main effects), and they can also condition the effects of individual-level factors on individual behaviours (e.g. moderating effects)" (Wilcox, 2003, p. 58). Pathways between the environment and individual smoking are complex.

Smoking is influenced by wider layers of context, of which the neighbourhood social environment is one. In an ecological model, the individual response (seen in the centre of Figure 4:3) can be viewed as arising from a number of possible sources, from the individual characteristics to the neighbourhood and occupational groups, to global events. Neighbourhoods may be seen as possible sites of influence for smoking behaviour through a variety of mechanisms such as “social norms, psychosocial stress, and exposure to advertising” (Datta et al., 2006, p. 1053). Smoking patterns tend to show non-random variation across neighbourhoods in many studies reported internationally, suggesting that causal factors go beyond the individual (Wilcox, 2003). The variation remains even after controlling for the individual risk factors for smoking. (Blakely et al., 2004, van Lenthe and Mackenbach, 2006, Wilcox, 2003)

The contribution of area deprivation to individual smoking over and above individual level socioeconomic status has been well researched (Blakely et al., 2004). For example, Stead et al (2001) documented the possible mechanisms by which pervasive neighbourhood disadvantage may influence individual smoking behaviours, such as psychosocial processes of despair and stress, access to tobacco products and so on. Yet there is some evidence suggesting that non-deprivation social group or neighbourhood factors may also be significant predictors of individual smoking (Patterson et al., 2004).

The literature suggests three possible mechanisms for the way in which social characteristics of neighbourhoods might influence smoking in individuals: social control, contagion and social norms. It has been argued that engagement in social networks has a protective effect (Kawachi, 2002, Siahpush et al., 2006). It is suggested that participation in networks increases the social control over individuals who are then more likely to conform to conventional health practices and less likely to practice risky health behaviours (Rasmussen et al., 2005). A link is frequently

suggested in the literature between high levels of neighbourhood social cohesion and capital and the uptake of healthy behaviours (Lundborg, 2005, Patterson et al., 2004). This is often argued to be through the diffusion of positive health messages (Siahpush et al., 2006), but also more directly “by increasing the likelihood that the norms of healthy behaviour are adopted” (Patterson et al., 2004, p. 692). In this instance the health message or norm can be seen as coming from outside the neighbourhood and the neighbourhood acts as a scaffold for the transmission of a wider health norm.

A difficulty with the social control mechanism is that it presupposes a universal or conventional health norm that is transmitted via neighbourhoods. Therefore it fails to account for alternative or non-traditional health norms that may nevertheless be transmitted within and across neighbourhoods (Lundborg, 2005, Stead et al., 2001). Thompson (cited in Wilcox 2003) argued that the result of peer pressure on smoking initiation will be a reflection of the norms around smoking within each group, not simply an effect of integration into the group.

By contrast the contagion model recognizes that norms and values around health behaviour will vary across societies. In this model it is the exposure to the smoking behaviours and attitudes of others that influences individual tobacco use (Wilcox, 2003, Christakis and Fowler, 2008). Thus neighbourhoods can have high rates of smoking, reflecting pro-smoking norms, or low rates, reflecting anti-smoking norms. Exposure may be an important factor in neighbourhoods because of geographical proximity. For example Christakis and Fowler (2008) found that workplace effects on the smoking cessation of study participants were dependent on workplace size, with smaller workplaces demonstrating a stronger effect, presumably because proximity facilitates interaction. It is therefore interesting to note that the authors did not find any effect for geographical distance when looking at the influence of neighbours on cessation (Christakis and Fowler, 2008). However, the authors did not account for the

part played by geography in establishing friendship networks. It is therefore possible that geography is an intermediary between social networks and cessation. Proximity seems to matter, above and beyond the social networks (Skjaeveland et al., 1996, Barnett, 2000)

A number of authors have made a case for participation in smoking practices being used by groups and individuals as a social marker: 'this is what we do'. Stead et al (2001) describe how high smoking rates within disadvantaged neighbourhoods created a pervasive pro-smoking norm. So strong was the "moral force" that non-smokers had to use excuses not to smoke that would be acceptable to their peers, such as health complaints. Smoking was seen as a normal activity that everyone did. As well as the act of smoking, the authors reported that the process of sharing in para-smoking practices such as purchasing and pooling of cigarettes acted to increase the feeling of belonging in the community. The authors also commented on the lack of other local social opportunities for the residents in this study setting to jointly participate in, increasing the importance of smoking as a collective activity (Stead et al., 2001).

Lundberg (2005) suggested that participation in substance abuse may be a means of gaining access to social networks. This mechanism has also been proposed as a way of explaining smoking initiation in youths who use participation in smoking as means of conforming to the social practices of older peers (Fidler et al., 2006). The argument aligns with the Durkheimian model where common social practices give rise to a moral force, which in turn reinforces the need for individuals to enact common social practices, in order to be a part of the group. Again, as highlighted by Wilcox (2003) there is a social risk to individuals of changing 'normal' behaviours in strongly integrated groups.

There is good reason to suggest that smoking behaviours may provide a useful test of the 'transmission' mechanism. The discussion above has established that social groups can provide an influential context for individual smoking practices by establishing norms for members. The prevalence (that is the density or visibility) of smoking practices within a social group, and their impact on individual behaviour will be used to test the mechanism in Chapter Seven.

### **4.3 Conclusion**

Why, and more specifically, how could neighbourhood social fragmentation be related to health? According to the theory developed in this chapter, it is because neighbourhoods may potentially act as social groups. Durkheim argued that social groups were a means of understanding the dynamic relationship between individuals and society. According to his theories, social groups are important intermediaries between the individual and the wider world. They are sites where social practices are enacted, and where the moral force is experienced. An important function of social groups is the role in balancing of the competing collective and individual wills present within the group. It has therefore been proposed that NeighFrag may be capturing types of neighbourhood level social groups, where health-related social practices are enacted and individuals experience levels of integration and regulation, with consequences for health.

Two specific health-related mechanisms have been proposed, with support from the literature. Firstly, the 'integration and regulation' mechanism suggests that living in a highly fragmented neighbourhood would reduce the levels of integration and regulation available to individuals from the local neighbourhood, which would be related to psychological distress and poorer mental health. Secondly, the 'transmission' mechanism suggests that health-related social practices such as smoking would be less easily transmitted within a highly fragmented neighbourhood because of less exposure to the practices of other residents and the



lower strength of the 'moral force'. Therefore we would expect a closer relationship between the smoking practices of fellow neighbours on the smoking practices of individual residents in less fragmented neighbourhoods.

The interaction of non-neighbourhood levels of influence with the type of neighbourhood will be less easily examined, given the available data. Investigating for example, whether societal level smoking norms have more or less influence across the levels of NeighFrag in New Zealand could not be observed or meaningfully interpreted with the data available here. However, it will be possible to investigate the potential for individual characteristics to modify influence of NeighFrag. Therefore the primary focus in the analyses that follow will be on developing tests of the mechanisms that are both observable and interpretable.

The process of turning the theoretical mechanisms into data is achieved in stages over the next three chapters. Chapter five establishes the social epidemiological framework for measuring variation in the health outcomes selected. The mechanisms are translated into observable pathways with exposures and outcomes at multiple levels. The subsequent two chapters focus on the analysis of those pathways using mental health and smoking outcomes. The analytical results are then brought together with the NeighFrag index and theory presented here to consider the evidence for a relationship between neighbourhood fragmentation and individual health.



## Chapter 5

### From Theory to Data: Developing the Methods Using an Epidemiological Framework

The previous chapter has established why and how NeighFrag might theoretically be related to health. Two hypothesized mechanisms were established by which fragmentation of the neighbourhood could affect individual health; 'integration and regulation' and 'transmission'. The next challenge, which is the focus of this chapter, was to design analyses by which the mechanisms could be empirically observed. All of the epidemiological analyses were designed, executed and interpreted by me. The purpose of the analyses was to build up a body of epidemiological evidence for the mechanisms from a variety of data sources, outcomes, and pathways. A social epidemiological framework has been used to empirically assess the association of NeighFrag and three outcomes: mental health, suicide and smoking; according to the mechanisms hypothesized above.

The translation of mechanisms into associations was achieved by defining specific pathways between the neighbourhood exposure and the individual outcomes. The chapter demonstrates the translation of the theory into data, or, in the words of Frohlich and colleagues; "In this context, method may be viewed as the underlying logic that allows researchers to operationalize theory into items." (Frohlich et al., 2007, p. 307). The first part of this chapter describes the process of drawing pathways which determined what the outcome of interest should be, how it might be related to other exposures and therefore how it can be statistically modelled. In epidemiological terms, the pathways represented potential cross-level associations between the neighbourhood and the individual. Two types of epidemiological pathways have been described: main effects and effect modification. The mechanisms

are summarized into four pathways that would allow the mechanisms to be empirically tested.

The chapter goes on to detail the generic methods required to empirically observe the pathways. While each analysis will have a specific methods section, much of the detail is common and so has been described here to avoid repetition. The emphasis in the chapter is on discussing the epidemiological framework and issues pertinent to the analyses. For example, the methodological challenges around deciding how to specify the NeighFrag exposure are discussed. The chapter also establishes the methods of managing and observing confounding, effect modification, and the multilevel regressions.

## **5.1 Epidemiological Strategies: How to Observe the Theory**

The three specific health outcomes (mental health, suicide, and smoking behaviours) are known to have different drivers at both the individual and neighbourhood level and therefore present a useful opportunity to observe differences in their relationship with this neighbourhood exposure. I will also be using regression techniques to measure the effect of neighbourhood fragmentation by modelling the exposure in three ways: as a main effect, as an effect which is modified by other exposures, and also as one which is a modifier of another exposure (where theoretically justified).

### **5.1.1 Multilevel Analytical Strategies**

Achieving the shift from theoretical pathways to associations required careful use of a set of epidemiological strategies. The “integration/regulation” mechanism suggested that neighbourhood fragmentation might be related to health because lower levels of integration and regulation available to individuals in highly fragmented neighbourhoods could adversely affect their wellbeing. In epidemiological terms this would be regarded as a ‘main effect’ of the exposure (NeighFrag) on an outcome (individual wellbeing). In the second pathway, the

“transmission” mechanism suggested NeighFrag might be related to health because fragmentation could affect the level of influence of local norms and practices over individual health-related practices. In other words, NeighFrag could have a ‘modifying effect’ on the relationship between exposure to neighbourhood norms and practices and individual outcomes.

To examine the main and modifying effects of neighbourhood fragmentation on individual health with regressions, the pathways need to be simplified. Blakely and Woodward (2000) used simple causal diagrams to model the various ways in which neighbourhood, or ecological, level exposure variables (X) might “cross-levels” to affect individual outcomes (y), in other words capturing a multilevel process. Upper case has been used to represent ecological level exposure and lower case an individual level exposure. The diagrams were a useful way of examining the complexity of possible pathways, how they crossed levels and operated indirectly through other variables and modifying effects. The simplified pathways between variables and levels have been demonstrated with arrows, with solid lines indicating associations that were observable in this thesis, and dotted lines representing unobservable relationships (due to lack of available data, for example). The vertical line separates the neighbourhood and individual levels.

As well as representing pathways, the diagrams are a good way of indicating what factors need to be taken into account and the inferences that can legitimately be made about cross-level influences. So for example, a relationship between two ecological level variables (X/Y) cannot be used to understand and make inferences about relationships at the individual level (x/y) without committing the ‘ecological level fallacy’ (Diez-Roux, 1998). More common in neighbourhoods and health research is the ‘sociologist fallacy’ (Diez-Roux, 1998) where inferences are made about the multilevel relationship between an ecological level exposure (X) and individual level outcomes (y) in the absence of individual level data. Conversely, relationships at the

individual level (x/y) cannot be used to understand or make inferences about ecological relationships without committing the atomistic fallacy, (Diez-Roux, 1998).

#### 5.1.1.1 Main Effects

Figure 5:1 shows a simple pathway (A) between the ecological variable, neighbourhood fragmentation (X), and the individual outcome (y). If, for example, we thought that mental health may be affected by the degree of integration and regulation provided by the neighbourhood, we would expect to see a main effect association between the NeighFrag at the ecological level that crossed levels to be associated with mental health outcome at the individual level.

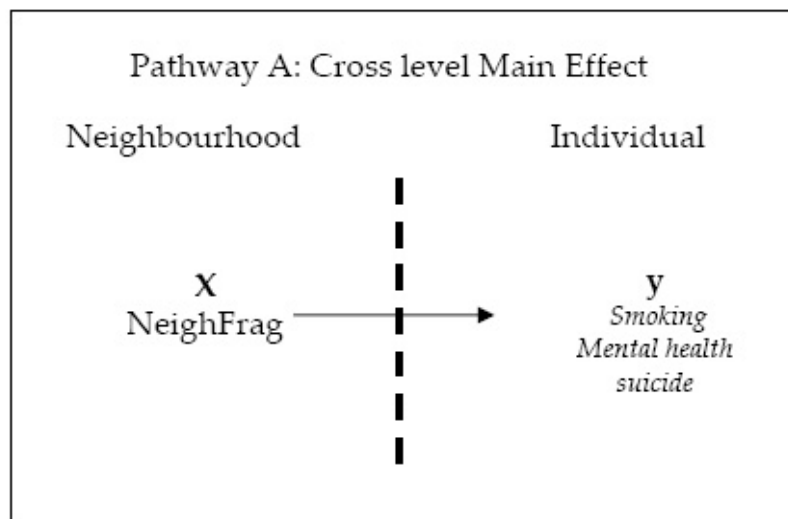


Figure 5:1 Pathway A: Cross-level Main Effect

The diagrams were also useful for exploring other possibilities when planning analyses. For example, how might it be possible for neighbourhood fragmentation to directly affect smoking? As discussed above, according to social control theory, individuals living in highly fragmented neighbourhoods would be more likely to engage in behaviour generally regarded as risky, such as smoking, as they are subject to less social control and monitoring. The lower levels of integration and regulation received from such neighbourhoods could also result in increased stress. Both

pathways could be observed with a cross-level main effect association between fragmentation and smoking.

However neighbourhoods have been theoretically framed in this thesis as just one source of influence. Therefore it was important to empirically observe how other contextual factors or individual characteristics played a part in the relationship between neighbourhood fragmentation and health. A causal diagram can have more explanatory power by taking into account less direct mediating and moderating pathways. In reality, these are likely to be multiple and complex, and beyond the scope of this thesis to either illustrate or observe. Nevertheless, there is still value to be gained from attempting to observe carefully defined aspects of these messy relationships, within the limits of the data.

#### *5.1.1.2 Indirect Effects*

While the main effect in Figure 5:1 is illustrated as a direct effect, in reality it would be better described as a main effect where the indirect pathways are not able to be observed. For example, we know that social capital (as measured by SoCInd) and NeighFrag are related, both theoretically and statistically. It is plausible then, that any relationship between NeighFrag and health might be via the social capital resources of a neighbourhood. In order to observe an indirect, mediating pathway like this we would need first to observe a relationship between NeighFrag and SoCInd. Figure 5:2 illustrates such a pathway where the solid line arrow (1) represents the observed association (the correlation between NeighFrag and SoCInd (the neighbourhood measure of volunteering social capital)) and the dotted arrow represents relationships not able to be empirically observed in this thesis.

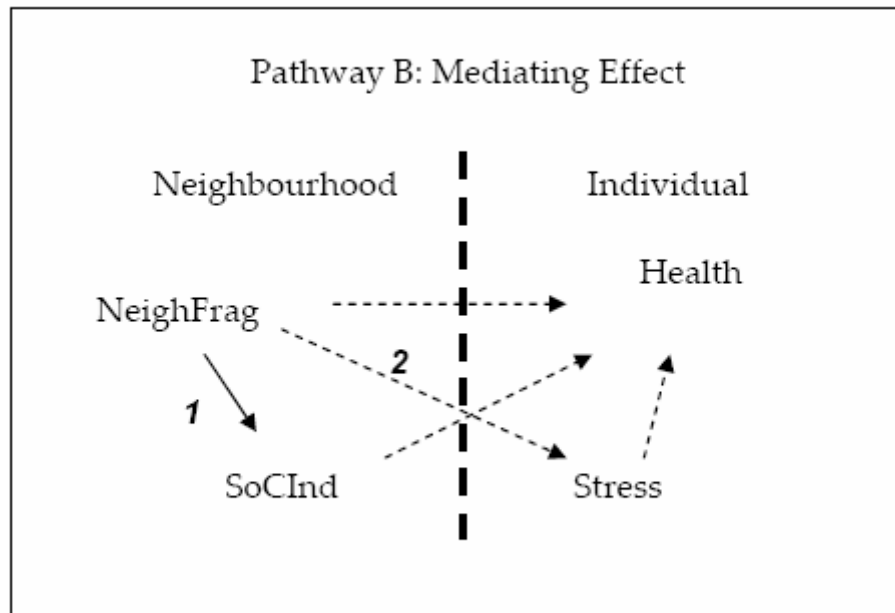


Figure 5:2 Pathway B: Mediating Effect

Indirect, or mediating, effects can also occur when the mediating exposure is at the individual level, as demonstrated by the dotted arrow (2). For example, in the smoking pathway discussed above, level of stress at the individual level can be regarded as a mediating factor between NeighFrag and smoking. However reliable measures of stress were not available to empirically observe the indirect effect. The means for examining indirect effects with cross-sectional data were more limited.

### 5.1.1.3 Effect Measure Modification

Another epidemiological means of seeing beyond direct main effects is effect measure modification (EMM). The modification of an effect, also known as interaction or a moderating effect, does not assume that the effect of an exposure will be the same for all levels of a third variable. Modification can occur across levels (cross-level EMM), or within the same level (within-level EMM).



Variations in the effect of a neighbourhood variable on individual outcomes can occur because of individual level factors, that is, cross-level effect modification. It may be that there are individual characteristics that make the neighbourhood a more or less important influence in a resident's response to their local environment. If we are considering the "integration/regulation" mechanism, we might be interested in how the effect of a highly fragmented neighbourhood might be moderated by an individual's membership of other important social groups.

This cross-level effect modification pathway is illustrated in Figure 5:3. The effect of NeighFrag on mental health is moderated by other individual level exposures. Such a pathway will enable us to observe how some individuals might be more resilient or vulnerable to the effect of living in a certain type of neighbourhood.

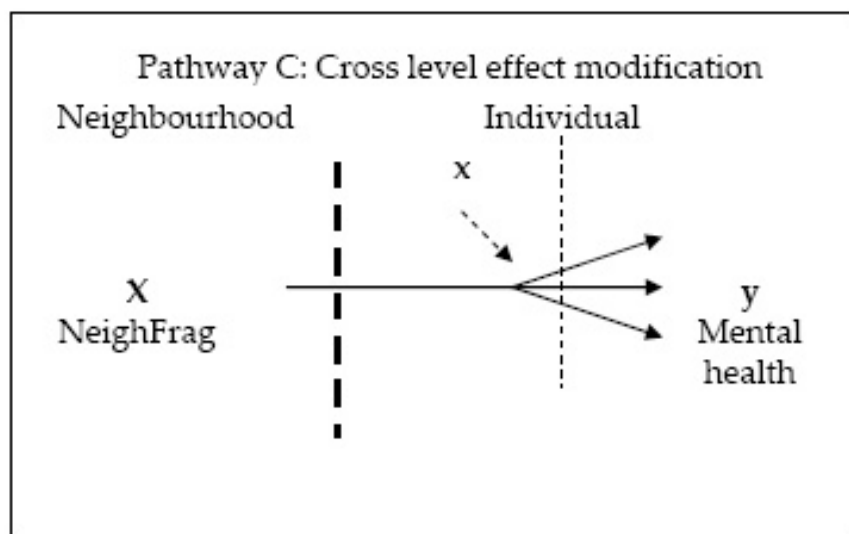


Figure 5:3 Pathway C: Cross-level Effect Modification

Within-level effect modification is illustrated in Figure 5:4. Effect measure modification can also occur within the neighbourhood level and can be used to observe the "transmission" mechanism. To what extent does NeighFrag vary the level of influence neighbourhood health-related norms and practices have on individual residents' practices? How easily are neighbourhood practices transmitted

within the neighbourhood in different types of neighbourhoods? The availability of census data on smoking enabled the testing of the ‘transmission’ mechanism by examining the modifying effect of fragmentation on the transmission of smoking norms and practices of the neighbourhood to the individual resident. In Figure 5:4 the effect of neighbourhood smoking norms X1 on the individual risk of smoking, y, may be modified by NeighFrag, X2

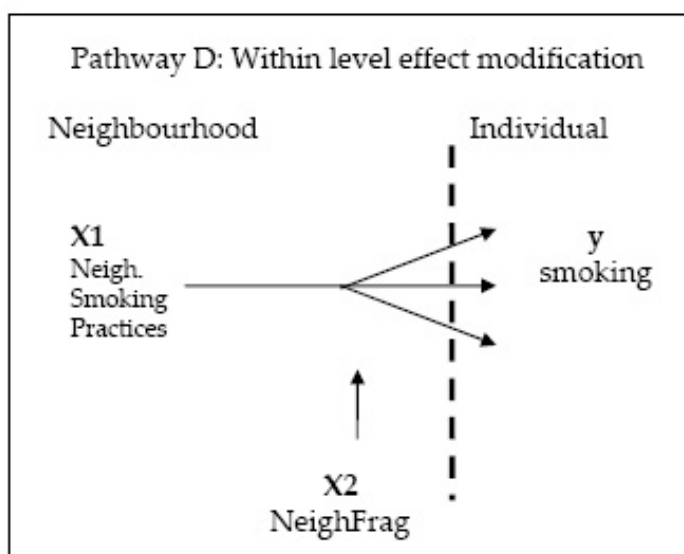


Figure 5:4 Pathway D: Within Level Effect Modification

As discussed previously, the degree of fragmentation of a neighbourhood has been hypothesized to have an effect on the extent of social networks within a neighbourhood. In a neighbourhood with comprehensive networks and high levels of connectivity, it may be that individuals are more exposed to and more affected by the particular smoking norms of their neighbourhood, whether they be low or high. On the other hand, residents in a more fragmented neighbourhood will be less exposed to the behaviours of their neighbours via networks and therefore less affected by them.

There are many alternative pathways that could be observed with these epidemiological strategies, depending on the data available. These four have been

selected because they were able to provide empirical information on some of the mechanisms proposed by Durkheim and discussed above and were thought interpretable from the available data. Together, they were used to provide empirical information on whether, and how, neighbourhood fragmentation might affect individual health.

## **5.2 Analytical Process**

The analytical process and presentation are enhanced when a considered and transparent approach is employed (Elm et al., 2007). Employing the hypothetico-deductive method (Susser, 2001) each analysis has been driven by research questions addressing the proposed theoretical pathways. The pathways were tested with the epidemiological strategies detailed above from which a set of hypothesised associations were specified. The analytical process was then used to provide statistical evidence for the pathways (Kleinbaum et al., 1998, Bhopal, 2002).

In order to examine the pathways, outcomes, exposures and the nature of the relationship between the two have been clearly specified. The primary statistical tools used here to investigate relationships were unadjusted, stratified analyses based on cross tabulations; and adjusted analyses using multivariable, multilevel regressions (Kirkwood et al., 2003). The body of evidence used to test the hypotheses comprised of, in order of priority, the magnitude of the association, and the level of precision. Both were important when deciding whether to support or reject the hypotheses (Kirkwood et al., 2003). As well as examining the individual estimates, patterns of association within each analysis, and across the set of analyses were also sought.

### **5.2.1 Datasets**

Two datasets were available to explore the hypotheses: the 2002/3 New Zealand Health Survey (Ministry of Health, 2004) and the 1996 National Census (Statistics

New Zealand, 2007a). Residential address information for each individual observation allows neighbourhood characteristics to be assigned to individual level data, creating a set of multilevel datasets with information on neighbourhood characteristics and a wide range of individual characteristics for each observation.

In all cases the datasets were provided for research with the linked individual and neighbourhood data completed by the dataset holders. No dataset had information that could identify individuals to their actual residence, thereby maintaining the confidentiality of the participants. The census datasets with individual information could only be accessed at the Statistics NZ secure data laboratory. Information brought out of the laboratory was subject to checking procedures to ensure confidentiality. All counts have been random rounded to a near multiple of 3 and care has been taken that no information is presented that could be used to identify individuals.

The Health Survey provides detailed health data, including standardised health measures, as well as information on individual characteristics. NZCMS provides individual level data from the census dataset that has been probabilistically linked to mortality records. In the 1996 National Census all adult New Zealanders were asked about their smoking habits. This provided a large, national dataset of an important health behaviour that included rich data on individual characteristics. Data on neighbourhood characteristics were appended to individual records in each dataset by the data holders for our use.

### **5.2.2 Outcomes**

Three types of variables were available within the datasets. The mental health outcome of interest in the NZHS was provided as a numerical variable (the Short Form 36 Mental Health scale) and was therefore used in linear regression methods, which come under the General Linear Model umbrella (Rothman and Greenland,

1998). The smoking variable dichotomised participants into current smokers (one or more cigarettes per day) or non-smokers to create a binary variable and was therefore analysed in logistic regressions, a common regression technique for binary outcome from the generalized linear model school (Rothman and Greenland, 1998). The different types of variables allow the opportunity to use both linear and non-linear outcome variables. Further details on each specific outcome are in the relevant analyses section below.

### 5.2.3 Exposures

As discussed in the index chapter, NeighFrag was modelled as a categorical variable with decile rankings of the score. This had several advantages in exploratory analyses. Firstly, nonlinearity could be examined, as an association was calculated for each decile (relative to a reference group), rather than the estimate representing an averaged unit change in outcomes across each NeighFrag decile without first examining for linearity.

Secondly, the original NeighFrag score had a highly skewed distribution. Converting the data into deciles (implying a ranking of neighbourhoods) still allowed for a meaningful interpretation of the score and did not need to be untransformed to be understood (if for example NeighFrag was logged for analyses) (Kirkwood et al., 2003).

However a downside to the use of categorical variables was a reduction in statistical power for each comparison of groups, as well as the increased degrees of freedom required. Therefore consideration was given to various groupings which would balance the need for information on any non-linearity of a NeighFrag – health association, but still allow for adequate precision and simplicity. Using quintiles and tertiles would have provided an even distribution of area units at each level and a relatively small number of categories. However, information would be lost about any

associations at deciles 1 and 10, a problem given the differences observed in their relationship with variables in Chapter Three. Therefore it was decided to categorise NeighFrag into 6 levels; decile 1; deciles 2&3; 4&5; 6&7; 8&9; and decile 10.

Choosing an interpretable reference group for NeighFrag was problematic. Common practice is to use the lowest decile or 'unexposed' group as the reference (Kirkwood et al., 2003). However, in keeping with Durkheim's theories of regulation and integration discussed in Chapter Four the theoretical preference was to model fragmentation as a balance between too little and too much. A statistical issue was the smaller numbers in decile 1, particularly when restricted to the urban population, which could result in imprecise estimates if NF1 were used as the reference category (Rothman and Greenland, 1998). The solution was to select deciles 4&5 as the reference group. Those living in neighbourhoods with moderate levels of fragmentation could then be compared with both ends of the spectrum.

NeighFrag was also modelled as a continuous variable as a means of further reducing the total number of indicator variables and for assessing any linear dose response relationships when categorical treatment of NeighFrag suggested this was appropriate. Rather than return to the (highly skewed) NeighFrag score, two alternatives for a 'continuous' variable were considered. Putting the NeighFrag deciles into the regression model as a continuous variable (that is, not including NeighFrag in the class statement in SAS) would provide an estimate that represents the average change per unit, in this case, per decile.

However, in these analyses, the primary interest was in observing the magnitude of any effect between more and less fragmentation rather than the average difference between the specific deciles. Therefore a variable was created that modelled the average change from the least to most fragmented neighbourhoods, where the unit of change was now the full range of fragmentation. Of course some information would

be lost on, for example any non-linear pattern of association, however the compromise could be managed with careful interpretation of the estimates with regard to the categorical results. This modelling could also be used to assess the statistical significance of any dose response between extremes of fragmentation. The use of the continuous variable in the Health Survey analyses also offered a partial sensitivity test of the categorical approach offered above. Agreement in the patterns and magnitude of the estimates between the two ways of modelling the index provided assurance that the results were not simply due to statistical artefact from the modelling process.

#### **5.2.4 Covariates**

While the focus was on the relationship between the primary exposure and outcome, other exposures must also be taken into account. Covariate exposures may be included in an analysis because they alter the 'true' relationship between the primary exposure of interest and the outcome in some way. Covariates can be divided into mediators, confounders, and effect modifiers (Rothman and Greenland, 1998). In practice, the distinction between the three effects is much less obvious (Diez Roux, 2002b).

The emphasis in a regression analysis is on assessing the relationship between a primary exposure and outcome. In reality, the association will be a reflection of indirect effects. This is particularly so in studies of contextual effects. Blakely and Subramanian (2006) used the example of unequal income distribution across neighbourhoods to highlight potential mediators between the neighbourhood exposure and individual outcomes. They argued that the causal pathway would need to include factors such as individual access to material and social resources, psychosocial stress and so on (Blakely and Subramanian, 2006).

The process of working through the causal diagrams can be a useful way of considering how such factors might be related. The term 'pathway' implies direction or movement from one factor to another. While the cross-sectional data here can only reveal associations and cannot give a definitive indication of causal direction, regressions can help to assess the presence of a pathway – is there an observable association (which could go either way)? Further, the analyses have attempted to unpack the components of the pathway by exploring how much of the relationship between neighbourhood fragmentation and individual outcomes can be explained by known individual risk factors and how much by the neighbourhood context. Then the onus is on the researcher to determine firstly if important factors on the pathways can be observed and secondly the implications of their not being included. There was limited scope available in the cross-sectional observational study designs employed here to properly examine mediating variables and indirect effects. It is also important to remember that the presence of mediators need not threaten the internal validity of an association. Therefore rather than focusing on illuminating the full causal pathway, the emphasis in this thesis has been to concentrate on using covariates to reduce sources of error.

While mediating variables are a topic of substantive enquiry, confounding variables are a potential source of error that requires careful management (Rothman and Greenland, 1998). Confounding of an association can occur when a third variable is common to both the exposure and outcome. The presence of such a factor can lead to error in the estimation of effect and possible spurious findings of association (or no association in the case of negative confounding) (Rothman and Greenland, 1998). As discussed in the literature review, adjusting for confounders is a standard statistical process used to address the exchangeability assumptions of regression analyses. In the analyses undertaken in this thesis, controlling for confounding has been used to better compare individuals living in neighbourhoods with varying degrees of



fragmentation, by making them more exchangeable. However the limitations of the process have been well recognised and remain contentious (Oakes, 2004)

Therefore the challenge for regression based epidemiological analyses reliant on cross-sectional observational data is to aim for adequate control of confounders (Rothman and Greenland, 1998). As Rothman and Greenland point out the focus should be "...to ensure that there are enough well-measured confounders in Z so that the no-confounding assumption is at least plausible" (Rothman and Greenland, 1998, p. 365). The interpretation of the association can then be cognizant of the degree to which confounding has not been managed, and the ensuing implications.

Finally, another factor could be modifying the primary relationship. While confounding is generally treated as a source of bias and therefore "managed", modification is of substantive interest, (Rothman and Greenland, 1998). Fortunately, modification was more readily observable with the data and techniques available here.

### 1) *Confounders*

In order to be considered a confounder, a distorting factor must fulfil three properties (Rothman and Greenland, 1998). Figure 5:5 illustrates the relationship between the substantive exposure of interest, the confounding exposure and the outcome. The arrows illustrate the confounding properties. Firstly, the factor must be associated with the outcome as a risk factor, independently of the substantive exposure (Rothman and Greenland, 1998). The direction of the arrowhead indicates that the confounder needs to predict the outcome in a causal fashion, but with recognition that the confounder (for example, age) may be a proxy for other more causal processes.

Secondly, the confounder must also be associated with the exposure (Rothman and Greenland, 1998). Here the lack of arrowheads indicates that the association does not need to be demonstrated as causal. It may well be that the association reflects a ‘backdoor’ causal pathway that cannot be fully illustrated in the data.

However the final third property is also important. The exposure should not cause the “confounder” (indicated by crossed arrow) as it would then be effectively on the causal pathway as an intermediary, or mediating, variable and therefore be of substantive interest (Rothman and Greenland, 1998).

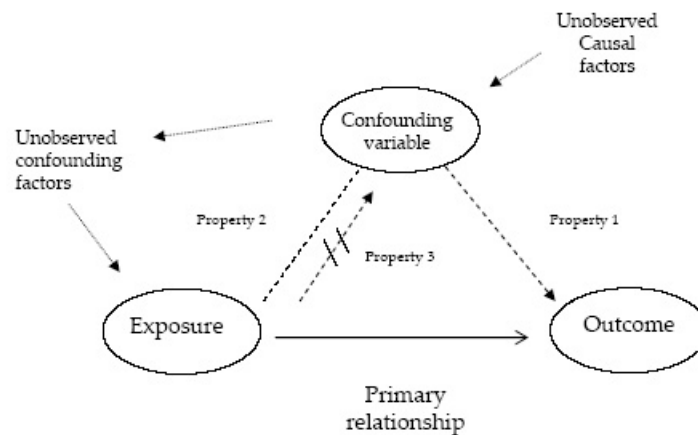


Figure 5:5 The Confounding Triangle

Potential confounding covariates were included in the analyses after consideration of prior research into risk factors for the outcome and assessment in the dataset (property 1). Chapter Three has established the associations between NeighFrag and potential covariates (property 2). Prior research and logic was used to determine the presence of property 3. The confounding triangle process has been detailed in each analysis to select confounding exposures.

To include confounding factors in a regression they must first have been available in the dataset as variables, secondly be measurable in a reliable way, and thirdly be a valid approximation of the confounding construct. Operationalizing confounding

factors was limited in this study by the data available. Therefore it would not be possible to fully control for all possible distorting effects (either known or unknown), leading to unmeasured or residual confounding. It was also important to remember that the confounders that are used are only as good as the proxy measures available in the datasets. Poor measurement can occur because the confounder is not recognized as a confounder and therefore not considered (Fewell et al., 2007); it cannot be operationalized with the available data, or that it is only partially measured (for example when SES is measured with a single variable). Measurement error can also occur when the confounder is mismeasured. Both errors in the measurement of the confounding process can lead to biased estimates (Fewell et al., 2007).

Given the recognised constraints of the regression modelling process (Fewell et al., 2007, Diez Roux et al., 2007) it was clear that the social world could not be fully captured with the data and methods available here. This was particularly so because the processes of selection into neighbourhoods would be difficult to take into account. As highlighted in the literature review and raised by Blakely and Subramanian (2006), it was unlikely that the cumulative effect of life course processes would be accurately captured. What can be useful however, is to take the process of the imperfect regression modelling "...to provide a simplified but useful picture of reality" (Kirkwood et al., 2003, p. 466). The picture will still be murky with residual and unmeasured confounding but by adding our proxies carefully into the model it may be made it a little clearer (Diez Roux et al., 2007).

Taking this approach was particularly important because of the secondary nature of the datasets. That is to say, they had not been developed to examine the hypothetical relationships and so the measures of the confounding constructs could only ever be proxies. It was also important to keep in mind that the analytical focus was on the effect of neighbourhood fragmentation, rather than seeking to accurately determine

the association of other risk factors for each outcome. With all these caveats in mind, the emphasis in these analyses will be to take a judicious approach (Galea and Ahern, 2006), observe the effect of confounding over a set of models, and interpret carefully.

## 2) *Effect Modifiers*

Other variables may be included in the modelling not because they need to be adjusted for but because they modify the primary relationship in some interesting way (illustrated in Figure 5:6). In other words, understanding the effect of the primary exposure was dependent on the presence of the modifier (Kirkwood et al., 2003). Effect modification can be used to examine more complex mechanisms between variables.

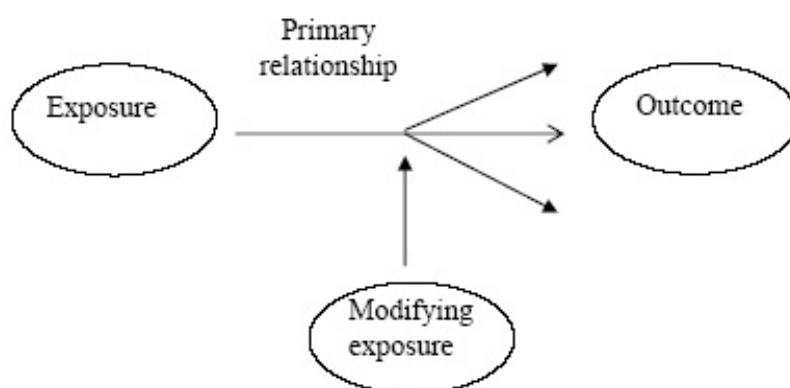


Figure 5:6 Effect Modification

Effect modification can take place between confounders, between confounder and primary exposure, and between exposures of interest. According to Kirkwood et al (2003), in most analyses it is the latter that is of primary interest and can be examined with stratified analyses and interaction terms. Effect modifiers for each analysis were

selected from the hypothesized pathways and mechanisms specified in the previous section. Evidence was sought of effect modification using two methods. Firstly, analyses were stratified by levels, or strata of the potential effect modifier. The analyses were examined for evidence of heterogeneity in the effect of interest across the strata (Figure 5:7). Secondly, a test of statistical interaction was included in the regression model. The estimates of the interaction term were examined for magnitude and precision to see if they indicated that any interaction had taken place (Kleinbaum et al., 1998).

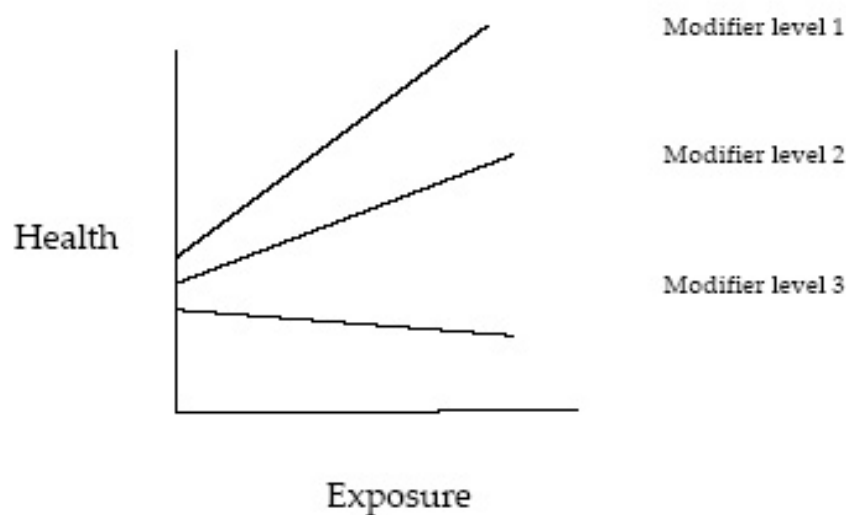


Figure 5:7 Heterogeneity of Effect by Stratum

Statistical support for interaction terms is often constrained by a lack of power and should therefore not be relied upon as the sole source of evidence (Kirkwood et al., 2003). In order to look for consistency between the two methods a check was made using the degree of heterogeneity observed in the stratification method and the size of the interaction term provided by the statistical method. The resulting interaction term estimates were used to back estimate the NeighFrag – health association by

strata of each of potentially modified variable. The results from the initial stratified analysis and the back calculated analysis were then compared for similarity of magnitude, allowing consistency checks of the two methods and an aid in the interpretation of the results.

As noted above, distinguishing between a mediator confounder and a modifier can be highly problematic in neighbourhoods research, particularly with cross-sectional data. Variables such as education could be regarded as either a mediator or a confounder (Blakely and Subramanian, 2006). Unfortunately adjusting for a mediator could result in a 'true' association being explained away, but if education was acting as a confounder, failure to adjust for it would result in a spurious association. To complicate things further, a single variable could be both confounding of one exposure and a mediator or modifier of another exposure in the same regression. Because there is no way of establishing which process was the more important in cross-sectional data such as this, Kirkwood and Sterne (2003) recommended that it is preferable to base judgments on a theoretical understanding of the possible pathways between the exposure, outcome and covariates.

## **5.2.5 Associations**

### ***5.2.5.1 Unadjusted Analyses***

Crude analyses were used to describe the distribution of the outcome by the exposures of interest unadjusted for any other covariates. They contributed to the body of evidence with comparison of the estimates between the unadjusted and adjusted analyses which provided information on the size of confounding effects (Kirkwood et al., 2003). The observed relationships in the crude analyses provided a useful check of the regression results, that the estimates obtained were due to the data rather than the modelling process (Rothman and Greenland, 1998). They were also used to develop the dataset for regressions, restricting the dataset to the required observations, selecting covariates, and modelling variables.

### 5.2.5.2 *Adjusted Analyses*

Multivariable regressions examined the effect of exposure(s) on the outcome, whilst simultaneously adjusting for covariates. A hierarchical model building approach was used to examine the association of NeighFrag with health while adjusting for covariates (Victora et al., 1997). Models started with an 'empty' model consisting of NeighFrag and the outcome. They were then built up with the addition of covariates based on a priori understanding. The most proximal, individual confounders were added first, followed by more distal confounders, such as other neighbourhood characteristics. A table detailing this process is presented for each analysis.

The inclusion of confounders at each stage was dependent on a priori understandings rather than, for example, statistical stepwise selection processes (Kirkwood et al., 2003). While it is preferable in linear regression to reduce the standard errors by including more rather than fewer covariates (provided they are not mediating variables), this is not the case with logistic regressions where the addition of 'extra' covariates decreases the precision of the estimates (Kirkwood et al., 2003). As discussed above, with both types of regression there was a need to reduce bias where possible and increase the internal validity of the associations. In particular, attempting to include some confounders could potentially introduce selection bias. This was because it would have required restricting the datasets to observations with complete information, which might exclude observations with a different exposure-outcome relationship.

The model building process provided statistical evidence of the contribution of NeighFrag to health outcomes. The magnitude and precision of the exposure/outcome association was observed at each stage. Any change in the magnitude of the estimates after adjusting for confounders was noted as evidence of the magnitude of confounding (Rothman and Greenland, 1998). Changes in the precision of estimates between models also contributed to the examination of

collinearity between exposures, where a substantial increase in the standard errors or confidence intervals could result from two highly collinear exposures being included in a model (Rothman and Greenland, 1998). Finally, regressions were used in an exploratory way to trial modelling of covariates and dataset restrictions.

### **5.2.6 Multilevel Statistical Modelling**

Because the interest in this thesis was to predict individual outcomes arising from both neighbourhood and individual level exposures, random effects models were employed. Random effects, or multilevel models, are now routinely used where the data is clustered in some way that means that the individual observations are no longer independent of each other, but are grouped within some hierarchical structure (Kirkwood et al., 2003). The clustering may be of substantive interest, as in neighbourhood, school effects, or repeated measures studies, or simply where the lack of independence challenges the assumptions in the statistical models employed. Employing a multilevel analysis allows for the examination of the effect of neighbourhood characteristics (at level 2) on an individual outcome (at level 1), whilst taking into account other important factors at both individual and neighbourhood levels (Merlo et al., 2005) On the flip side, it also sheds light on the part that individual characteristics play in outcomes even when controlling for an individual's contextual environment.

Another key question in Neighbourhood and Health research is examining how residents in geographical communities may be more like their neighbours in some outcome than those in other neighbourhoods. While this research question goes back at least as far as Durkheim it is only comparatively recently that the appropriate research tools have been developed to empirically examine neighbourhood homogeneity. A multilevel approach allows us to ask whether a degree of homogeneity exists at the neighbourhood level. That is, are the outcomes of individuals within neighbourhoods more alike, or correlated with each other, than



with the general population (Merlo, 2003)? Much harder to empirically assess and interpret is the part played by other levels of influence on any observed heterogeneity within neighbourhoods. It is unfortunately outside the scope of the data available here to assess how other settings such as media or policy influences may be related to any heterogeneity in health outcomes within neighbourhoods.

The inherently clustered nature of the data in neighbourhood research raises complex statistical issues that cannot be ignored if regression analyses are to be used. If data is clustered it violates the statistical assumption of independence between observations. A consequence of this is negatively biased standard errors, with confidence intervals that are too small, therefore affecting the interpretability of the results (Kirkwood et al., 2003).

The datasets employed here allow for a two level structure, with individuals at level 1 and neighbourhood (census area unit) at level 2. Figures 5:1-4 have demonstrated the two level data structure required to examine cross-level effects, whilst taking into account the clustered nature of the data.

### **5.2.7 Regression Equations**

The techniques used here come under the umbrella of General Linear Models (GLM) which aims to determine if the exposure variable(s), in some way, have a linear effect on the outcome variable (Foster et al., 2006). Two forms of the GLM were used in the analyses. Firstly, mixed models (regarded as a special case of multiple regression) were used to measure variance in the outcome means between groups, by a number of exposures and across levels. This approach was used for the continuous Mental Health outcome. Secondly, generalized linear models were used to examine categorical and dichotomous outcomes. Logistic regressions are related to a linear regression but instead use a transformed outcome variable (Foster et al., 2006). Logistic uses the log of the odds of the dichotomous outcome, a logit linking

function. It was used for the smoking outcomes. In SAS, a random effects logistic model is referred to as a Glimmix, that is, a combined generalized linear model using a logit link that has mixed fixed and random effects.

In these analyses a random intercepts model was used, allowing the intercept to vary between neighbourhoods, thus controlling for the non-independence of observations (Note random slopes were not able to be reliably calculated for the mixed models because of weighting problems. For more details refer to appendix 1. Further, statistical power for random slope models (especially for logit) is often limited). The random intercepts model calculates the usual fixed effects within each group, the level 1 regression equation (in this case the individual level). It also calculates the level 2 fixed effects (in this case the neighbourhood). These two levels are simultaneously calculated, hence the common term “mixed model”, capturing the mixture of effects across levels.

The basic equation for a multilevel regression used here is

$$y_{ij} = \beta_0 + \beta_1 NeighFrag_j + \beta_2 confounder_{ij} + (u_{0j} + e_{0ij})$$

Here,  $y_{ij}$  is the outcome for individual  $i$  in neighbourhood  $j$ . The individual exposure values are then represented by  $NeighFrag_j + confounder_{ij}$ . Other neighbourhood level variables included in the model would be represented by  $\beta_3 confounder_j$

The final part of the equation  $(u_{0j} + e_{0ij})$  represents the crucial random part where  $u_{0j}$  represents the neighbourhood random error and  $e_{0ij}$  represents the individual random error

The “random” aspect of the equation is captured in the calculation of the covariance. That is, it calculates the extent to which the predicted variation can be allocated to

within, and also between neighbourhoods, rather than assuming it is the same between neighbourhoods or individuals (Cohen and Cohen, 2003). The extent to which the variation observed in the outcomes can be attributed to the neighbourhood level can then be calculated in linear mixed models as the intraclass correlation (ICC), using the following formula.

$$\text{ICC} = \frac{u_{0j}}{(u_{0j} + e_{0ij})}$$

An ICC of 0 would indicate that there is no homogeneity at the neighbourhood level and that all of the variation in the outcomes can be attributed to individual level differences (Merlo, 2003). On the other hand, a non-zero ICC would indicate that there is a degree of homogeneity within neighbourhoods and that therefore some of the variation in the outcomes can be attributed to the neighbourhood. It is generally recognized that ICC's will be relatively small for neighbourhood effects but still be of substantive interest. In fact Merlo (2003) and Blakely and Subramanian (2006) argued that important ecological effects can be observed even when there is no statistically significant ICC. Furthermore the interpretation of between neighbourhood variation is particularly problematic when non-linear outcomes are used (for example, the smoking data used here) (Blakely and Subramanian, 2006, Diez-Roux, 2000).

### **5.2.8 Sources of Error**

The analyses have been concluded with a discussion focusing on the potential for random and/or systematic error in the associations. To avoid repetition the discussion of internal validity and accuracy has been conducted by dataset. That is, the discussion of all the analyses using mental health as an outcome from the NZHS have been discussed together, as have the smoking outcome analyses using the census dataset. A more comprehensive discussion on the external validity has been undertaken in the discussion chapter. The discussion chapter has then focused on the generalisability of the associations. That is, the extent to which the associations have

offered support for the theoretical hypotheses (Rothman and Greenland, 1998). The discussion has therefore concentrated on exploring alternative explanations for the observed associations between the neighbourhood fragmentation and health.

### **5.2.9 Software**

SAS was used for all statistical processes. Proc Mixed was used for the multilevel linear regressions from the health Survey, using version 9. The SAS GLIMMIX macro was invoked (utilizing Proc Mixed) for the logistic regressions in the Statistics NZ data laboratory using version 8.2.

### **5.2.10 Conclusion**

This chapter has detailed the methods for moving from theory to data, using a social epidemiological framework to examine variations in health outcomes by type of neighbourhood. It has described the process of developing multilevel analyses from the secondary data sources available. The criteria for defining the outcomes of interest, the substantive exposures and other covariate exposures such as confounding and modifying exposures has been described. The modelling of the variables in crude analyses and adjusted multilevel regression analyses has been carefully defined to allow a body of information to be developed.

Four pathways were established to capture the cross-level associations between the neighbourhood exposure and individual health outcomes, each capturing an aspect of the hypothesized mechanisms. The relationship between neighbourhood fragmentation and health could be observed in four pathways: as a cross-level main effect between NeighFrag and health; as a cross-level modified effect, where the effect of NeighFrag on health was modified by individual factors; where NeighFrag modifies another neighbourhood level exposure, that is a within-level modification; and finally, where the effect of NeighFrag is via another factor, an indirect cross-level effect. The last pathway is less easily observed with the available data and was therefore not examined further.

The following chapters present the analyses for the first three pathways. The chapters present the issues and methods specific to each set of analyses, with the detailed results, and a brief discussion focusing on the accuracy and internal validity of the associations. A more in depth discussion of the results as a whole focusing on the extent to which the observed associations provide evidence for the theorized mechanisms is given in Chapter Eight.

The analyses are grouped by health outcomes. Chapter six presents the analyses designed to test the 'integration/regulation' mechanism. It is tested with two pathways, a cross-level main effect between NeighFrag and mental health, and cross-level effect modification, where the effect of NeighFrag is tested for effect modification by individual factors.

The smoking outcome in chapter seven was used to test two pathways. To avoid confusion the chapter has been split into two sections. The first section used a variation of the 'integration /regulation' mechanism in an exploratory analysis to examine a cross-level main effect between NeighFrag and smoking. The second section tested the 'transmission' mechanism by using a within-level effect modification pathway, asking if NeighFrag modified the cross-level main effect of another neighbourhood exposure on the individual smoking risk.

The analyses also present the opportunity to observe how neighbourhood fragmentation is related to other important covariates. The information added to the body of evidence for fragmentation and also shifted the focus from a single causal factor to considering multiple aspects together (Diez Roux, 2007). Increasing our understanding of fragmentation by examining its relative contribution to health alongside other neighbourhood factors, particularly deprivation, has already been established as an important research question (Congdon, 1996b, Whitley et al., 1999, Corcoran et al., 2007). A number of individual variables are also well recognized as

important risk factors for the outcomes examined here. However, in this thesis the focus will be instead on the part they play in explaining the relationship between fragmentation and health. These issues have been addressed within the two chapters.

## Chapter 6

### Measuring the NeighFrag - Mental Health Relationship

The analyses presented in this chapter have been designed to empirically test the hypothesised mechanism of 'integration/regulation' using mental health outcomes. Theory and research have both suggested that the type of neighbourhood social setting (as measured by NeighFrag) could be related to individual mental health. The previous chapter has established a means of empirically observing the mechanism using epidemiological methods. According to the proposed cross-level pathway between NeighFrag and health, exposure to NeighFrag would provide varying levels of integration and regulation for residents, with associated differences in their individual mental wellbeing.

Therefore the first research question was:

- 1) *What was the association of NeighFrag with Mental health?*

*And more specifically,*

- *Was there a cross-level main effect of NeighFrag on mental health, controlling for individual and neighbourhood confounders?*
- *What was the strength and precision of the association of NeighFrag and mental health, after controlling for confounders?*
- *What was the direction of the association? Were increasing levels of neighbourhood fragmentation associated with poorer mental health in individuals, as hypothesised by the integration/regulation mechanism?*
- *Was the association between NeighFrag and health specific to mental health outcomes?*

As established earlier, the thesis has also examined the interrelationships between neighbourhood and individual characteristics, not simply the main effect of a single risk factor. In particular, it is proposed that the effect of fragmentation could be modified by membership of non-neighbourhood social groups because they offer alternative sources of integration and regulation. Alternatively, there may be other social factors which were important in understanding how influential neighbourhood social groups may be, for example, gender. In epidemiological terms, this meant examining cross-level effect modification: how individual factors might modify any observed association between neighbourhood-level NeighFrag and mental health.

The second research question was:

- 2) *Was the main effect of NF on Mental Health modified by individual level characteristics, specifically those which could be proxies for membership of other social groups (for example, labour force status), or which might affect the way individuals respond to the social environment (gender and age).*

*And more specifically,*

- *Did membership of other social groups reduce the magnitude of the association of NeighFrag with Mental Health?*

The analysis offered the opportunity to explore some secondary questions on the association between NeighFrag, NZDep, and health; and a comparison between the two measures of fragmentation: NeighFrag and Congdon(NZ).

- 3) *Exploring the relationship between NZDep & NeighFrag*
  - *Did any association between NeighFrag and health remain after adding NZDep into the model; that is, were fragmentation and deprivation independently associated with health?*



- *Was there evidence of any interaction between NeighFrag and NZDep in the association with mental health?*

*What were the statistical implications of the moderate correlation between NeighFrag and NZDep for use in regression models?*

- 4) *Was there any difference in the association of the two measures of neighbourhood fragmentation (NeighFrag and Congdon(NZ)) with mental health?*

Evidence for the above questions has been examined below in unadjusted and adjusted multilevel regression analyses using the New Zealand Health Survey (2002/3). The analyses are reported in four steps, following the four research questions stated above.

In the first question, the primary outcome of interest was the Mental Health Scale of SF36. The results demonstrate that exposure to NeighFrag was associated with variation in self-reported mental health, which remained statistically significant even when controlling for known individual factors, and when neighbourhood deprivation was included in the model. The direction of the association was as hypothesised with increasing fragmentation predicting poorer mental health. The specificity of the association with mental health was a further test of the mechanism. The association of NeighFrag and health was found to be relatively specific to mental health. While self-reported general health was also associated with NeighFrag, deprivation was a stronger factor. There was less consistent evidence of any association with other SF36 scales, particularly those which captured physical health status.

Secondly, individual factors modified the association of NeighFrag with mental health, suggesting cross-level effect modification. While men's mental health status varied by individual factors and neighbourhood deprivation, there was no suggestion in the data that NeighFrag was a predictor of mental health. Conversely, NeighFrag was associated with women's mental health. Labour Force Status was

used as a proxy of membership of alternative social groups. Stratified and pooled analyses demonstrated that the association of NeighFrag with mental health was considerably worse for unemployed women compared to, for example, employed women.

The third research question examined the association of NeighFrag with mental health which was found to be independent of the association of NZDep with mental health. It appears that neighbourhood fragmentation was a stronger factor for mental health. However, collinearity between the two measures posed problems with sparse data, particularly at lower levels of fragmentation. There was no evidence that the statistical assumptions of the regression modelling were overly compromised. There was limited evidence of an interaction between deprivation and fragmentation for mental health.

Finally, the association of fragmentation and mental health was specific to the NeighFrag index. There was no consistent evidence of an association between mental health and the Congdon(NZ) index.

## **6.1 Methods**

### **6.1.1 Dataset**

A multilevel data set was created from the 2002/3 New Zealand National Health Survey (NZHS) (Ministry of Health, 2004) by linking data on neighbourhood characteristics with individual data. The survey is part of the national health monitoring programme. It employed a clustered, stratified sampling technique. The total sample size of 12,529 was restricted to those 15 years and over and usually resident in permanent private dwellings. A two stage over sampling process was employed to increase the numbers of respondents in key ethnic groups, Māori, Pacific People or Asian. Weights were provided with the dataset to make the dataset

comparable to the whole population. Further details are available elsewhere (Ministry of Health, 2004).

Unfortunately the complicated weighting structures employed lead to problems using the provided weights for multilevel analyses. It was not possible to statistically allow for: all of the weights; stratified survey design; and neighbourhood random effects. Subsequent investigations (by others) resulted in a peer reviewed 'work-around' (see Appendix:1) using design variables as covariates, negating the need to use weights (further details of the design variables are below). As a consequence random intercepts were able to be reliably calculated but not random slopes (see Appendix:1). The increased number of parameters required by the use of categorical design variables also placed some study power limits on possible analyses and required parsimonious modelling.

The dataset was restricted to those with complete data. As the sampling only included permanent private dwellings no further restriction to the type of dwelling was required. The differences in the NeighFrag distribution of the population by urban/rural settings (described in Chapter Three) posed some concerns. Preliminary analyses (not shown) indicated that there was an association of NeighFrag with mental health independent of urbanicity, confirming that NeighFrag was not simply capturing the degree of urbanicity. However, when the analysis was stratified between the urban and rural populations, the results for the rural population were un-interpretable due to the very wide confidence intervals.

There was concern that any relationship between neighbourhood fragmentation and mental health could vary across urban and rural settings. It was noted in Chapter Three that there was concern that CAU's may not capture 'neighbourhoods' to the same degree as more urban settings. Therefore high levels of fragmentation in a rural neighbourhood may mean something different than in a more urban setting. Earlier

research has also found that different factors have been found to predict poor mental health in women across the two settings (Romans-Clarkson et al., 1990). Because of the smaller numbers living in rural settings, particularly in highly fragmented areas, it was not possible to stratify the results. Therefore the decision was made to restrict the analyses to the population for whom reliable, interpretable estimates could be obtained. The down side of this was the inability to examine the potential modification by another important contextual factor. Also, the numbers in NF decile 1 were then reduced by 50%, decreasing the precision of the estimates for this exposure group. Finally, any results would only be interpretable to the urban population.

### **6.1.2 Outcomes**

Among other measures of health and health-related outcomes, the Health Survey recorded self-reported health using the standardized measurement tool, the Medical Outcomes Study Short Form 36 (SF-36) (Ware et al., 2000). The Australian and New Zealand version was used here and has been validated for the New Zealand population (Scott et al., 1999).

The SF-36 has been designed to capture both clinical and population level differences in the means of the individual scales (Ware et al., 2000). Self-reported health provides a subjective measure of health, focusing more on wellbeing and quality of life than on more objective measures of health status (Ministry of Health, 2004). The SF36 asked participants to rate their health across eight aspects; general health (GH), mental health (MH), vitality (VT), physical functioning (PF), role physical (RP), bodily pain (BP), social functioning (SF), and role emotional (RE).

The dataset was provided with scores calculated from the responses to 36 items, ranging from 1-100 (Ware et al., 2000). A higher score represented better self-reported health. General health, mental health, and vitality were bipolar scales, capturing both

negative and positive health, while all other scores were unipolar, with a maximum score representing no limitation (Ministry of Health, 2004).

The individual scales can also be combined with Principle Components Analysis to represent two related, but separate constructs: general and mental health. The component scores have been validated in a number of Western European populations and are commonly used (Scott et al., 2000). In an investigation into the validity of the component scores in the New Zealand population, a two factor structure was observed in the NZ European population, in keeping with other studies (Scott et al., 2000). The MH, RE, VT and SF scales all loaded onto one factor (Mental Health construct), and PF, RP, and BP loaded onto another (General Health construct). The GH scale was moderately related to both constructs in this population, as expected (Scott et al., 2000).

However, the factor structure of the scales for other ethnic groups in New Zealand was not so clear cut (Scott et al., 2000). In the younger Māori population, a two factor structure was observed, in keeping with the NZ European population. But in the older Māori population the scales loaded onto a single factor, suggesting that mental and physical health could not be so readily separated (Scott et al., 2000). No clear, reliable factor structure emerged for the Pacific population (Scott et al., 2000). The authors suggested that the cultural differences in the meaning of health and wellbeing may underlie participants' interpretation of the questions and therefore their responses (Scott et al., 2000).

Because of the concerns raised over the validity of the component scores, the decision was made to use the individual scales. The primary focus of the analysis was on the relationship between the Mental Health scale and NeighFrag. The scale captures how participants perceive their current mental health, ranging from "feelings of

nervousness and depression all of the time” to “feels peaceful, happy, and calm all of the time” (Ware et al., 2000).

The Mental Health Scale asks participants to rate their responses to five questions;

In the last 4 weeks have you:

- Been a very nervous person.
- Felt so down in the dumps that nothing could cheer you up.
- Felt calm & peaceful.
- Felt down.
- Been a happy person.

Participants were asked to rate how much of the time in the last four weeks they had felt that way; “all the time”; “most of the time”; “a good bit of the time”; “some of the time”; “a little of the time”; or “none of the time” (Ware et al., 2000).

The individual scales have been designed to capture wellbeing or health, rather than illness (Ware et al., 2000). Halpern (1995) distinguished between mental health and illness, with the former being described as “...the condition of being free from psychiatric symptoms and having a subjective feeling of well-being.” (Halpern, 1995, p. 2). However, he did not regard mental illness as the opposite of health (Halpern, 1995). In the same way, participants reporting poor mental health in the SF-36 could not necessarily be regarded as being mentally ill. Nevertheless the two are not unrelated and the MH scale has been used as a screening tool for psychiatric disorders and assessing psychiatric interventions (Ware et al., 2000).

A theoretically plausible relationship between NeighFrag and other SF36 scales was less clear. However, they provided an opportunity to conduct sensitivity analyses to better understand the specificity of the pathway between NeighFrag and mental

health suggested in earlier research. The General Health scale has been demonstrated to be related to the wider mental health construct and could therefore also be an indicator of a generally stressful environment. It was used in analyses here to provide a comparison to the Mental Health scale. The General Health scale captures states ranging from “personal health as poor and believes it is likely to get worse” to “Evaluates personal health as excellent” (Ware et al., 2000).

Participants were asked to rate their health in general;

- How would you say your health is? with the options of “excellent”, “very good”, “good”, “fair” or “poor”

They were then asked to use these responses - “definitely true”, “mostly true”, “don’t know”, “mostly false” or “definitely false” to rate how true the following statements;

- I seem to get sick a little easier than other people
- I am as healthy as other people I know
- I expect my health to get worse
- My health is excellent

While it is possible that there may be a pathway between the health constructs measured by the other scales and NeighFrag, there was little in the literature and in the theory proposed in Chapter Four that suggested such pathways. Estimates for the other six scales (Physical Functioning, Role Physical, Bodily Pain, Vitality, Social Functioning, Role-Emotional, and Health Transition) were calculated for the unadjusted and fully adjusted models, with only the fully adjusted reported for comparison with the General and Mental health results.

If a pathway were present it is hypothesised that any mechanism would likely be less direct and therefore a weaker association would be expected. This provides the opportunity to use the scales as a specificity test of the effect of NeighFrag on self-reported Mental Health. It is expected that if there is an association between NeighFrag and self-reported health it will be stronger in mental health scores compared to other scale scores, with the possible exception of General Health.

#### ***6.1.2.1 Outcome Specification***

The scales were provided in the dataset as continuous measures. Mental and General Health both showed a highly skewed distribution. Further details of the univariate distributions of all the scales in the New Zealand population are available in (Ministry of Health, 2004).

The scales were specified in the regression analyses as untransformed continuous variables. Because of the non-normal distribution of the outcome variable, careful consideration was given to various transformations such as log transformation or categorical ranking. However the added statistical precision gained comes at a cost of interpretability of the estimates in the case of, for example: log transformations (Rothman and Greenland, 1998); potential loss of information if, for example, the scale was ranked or dichotomized (Kirkwood et al., 2003); and when the effect of more than one exposure is being examined (Kirkwood et al., 2003). Using the SF36 scales as a continuous variable in a linear regression also provided the opportunity to examine between-neighbourhood variation with the intraclass correlations (ICC's).

Further, a number of authors have argued that with large sample sizes such as the NZHS a skewed distribution of the outcome does not necessarily pose a threat to the statistical assumptions of the regression model. This is because central limit theorem implies that a large sample size will still have a normally distributed sampling distribution, and that therefore the distribution of the residual sum of squares (RSS)



in a regression will be normal (Gujarati, 1999, Kirkwood et al., 2003). Analyses on a previous 1996/7 Health Survey which used the SF36 also found a highly skewed distribution, but concluded that the sampling distribution was sufficiently normal to meet parametric method assumptions (Scott et al., 1999).

In mixed linear models the F statistics can be used to check the effect of central limit theorem on the distribution of the residuals and therefore the degree to which the statistical assumptions were met. F statistics are used to compare variation in the means between groups by comparing between-group variation and within-group variation of the mean. A value close to 1 would indicate that there was little difference between groups (Kirkwood et al., 2003). Because the calculation is obtained using the RSS (the denominator is obtained by dividing the RSS by the degrees of freedom (Kirkwood et al., 2003)) a statistically significant F value would be reliant on a normally distributed RSS. Therefore a rule of thumb is that when significant p values were obtained for the F statistics it can be taken as evidence that the model assumptions have been met.

SAS calculates the partial F statistic for each exposure variable in a multiple regression. The statistic was therefore used to provide information of the statistically significant contribution of an individual variable to the prediction of the outcome variable, after accounting for the contribution of the other variables (Kleinbaum et al., 1998).

#### ***6.1.2.2 What Does it Mean for Health? Interpretation of Regression Estimates***

The betas calculated in the regressions represent the differences between the reference group's predicted mean score (captured as the intercept) and each other level of the exposure. So, for example, the predicted mean mental health score of non-Māori, Non-Pacific, non-Asian (described in the tables as 'other'), 45-64 year olds, employed people with post school qualifications and living in a moderately

fragmented, least deprived neighbourhood might have a predicted health score of 90. A negative association between NeighFrag and mental health would mean that people with the same individual and NZDep characteristics but living in the most fragmented type of neighbourhood would have a predicted mean mental health score that was 3 points lower (for example).

Thus, evidence for an association can be obtained from the magnitude and precision of the estimates, as given by the size of the beta and its confidence intervals. The magnitude of the association of each exposure has been captured in the size of the difference, as calculated by the beta. The precision of the estimates has been indicated by the standard errors and hence the width of the confidence intervals. It has been well established that small differences in the scale scores (between 3 and 5 points) can be significant for social and clinical definitions of well being, particularly when making cross-sectional population level comparisons (Scott et al., 1999, Ware et al., 2000).

Therefore when considering the evidence for an association of neighbourhood fragmentation on mental health, two aspects can be considered. Firstly, did the difference in scores across NeighFrag levels represent important differences in well-being (as represented by the magnitude and precision of the estimates)? And secondly, what is the magnitude of the association compared to the magnitude of other known risk factors?

### **6.1.3 Exposures**

#### ***6.1.3.1 Neighbourhood Fragmentation***

The two fragmentation measures were modelled as both categorical and continuous variables. When the categorical modelling was used, the reference group was set as NF decile group 4,5 to allow comparison between moderate levels of fragmentation, and more and less fragmentation. The use of the measures as a continuous variable

increased statistical power where needed, allowed examination with the F values of the contribution of the exposures to variation in the outcome, and enabled the further assessment of any possible dose response relationships observed in the categorical modelling of NeighFrag. The NeighFrag (continuous) and Congdon(NZ) (continuous) variables were created by ranking the deciles and assigning a value between 0.05 and 0.95 to each decile. The resulting estimates represented the average predicted change, or the slope, in the SF-36 score, from the hypothetical person living in the most fragmented neighbourhood with the person living in the least fragmented neighbourhood.

#### ***6.1.3.2 Selection of the Covariates***

Potential confounders were selected using the confounding triangle process described in Chapter Five. Triangles were developed for variables known from the literature to be risk factors for mental health and from crude analyses in the Health Survey (property 1). The associations between the potential confounders and NeighFrag (property 2) have been established in Chapter Three. For the third property, the confounding triangle process will be used to discuss the rationale for each factor and explore whether the third property may be met using *a priori* understanding and logic.

Potential effect modifiers were selected on the basis of their potential to capture an alternative social group membership, or as a factor which might be related to how individuals relate to their neighbourhood.

#### ***6.1.3.3 Individual Confounders***

Age predicted mental health status in the HS population, with increasing age predicting improved health (Ministry of Health, 2004), fulfilling property 1 (Figure 6:1). It has also shown to be related to NeighFrag, with the neighbourhood mean proportion 15-34 year olds (for example) increasing as fragmentation increased

(Figure 3:9a-d), fulfilling property 2. It is not logical that NeighFrag can causally influence age, fulfilling property 3. Age was therefore included as a potential confounder.

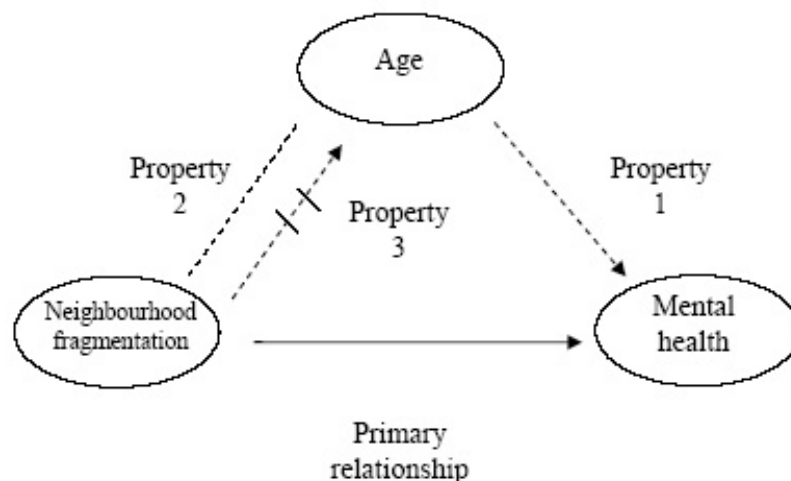


Figure 6:1 Age Confounding Triangle

While ethnicity needed to be included in the regression models as a design variable, its confounding role was also of interest in its own right (Figure 6:2). Ethnicity has been established as a risk factor for mental health in the HS population with, for example, those who identify as Asian reporting slightly better mental health than non-Asians (Ministry of Health, 2004) (property 1). As established in Chapter Three, ethnicity was also associated with NeighFrag (Table 3:5), fulfilling property 2. It is plausible that the process of identifying one's ethnicity may be related to the strength of local norms around ethnic identification, suggesting that neighbourhood fragmentation could in some way be causally related to ethnicity. However it is unlikely that NeighFrag would be a strong factor in most residents' ethnic identification. For the purposes of these analyses it is therefore argued that it is unlikely that neighbourhood fragmentation would have a sufficiently causal effect on the process of identifying one's ethnicity in the census, fulfilling property 3. Ethnicity was therefore included as potential confounder.

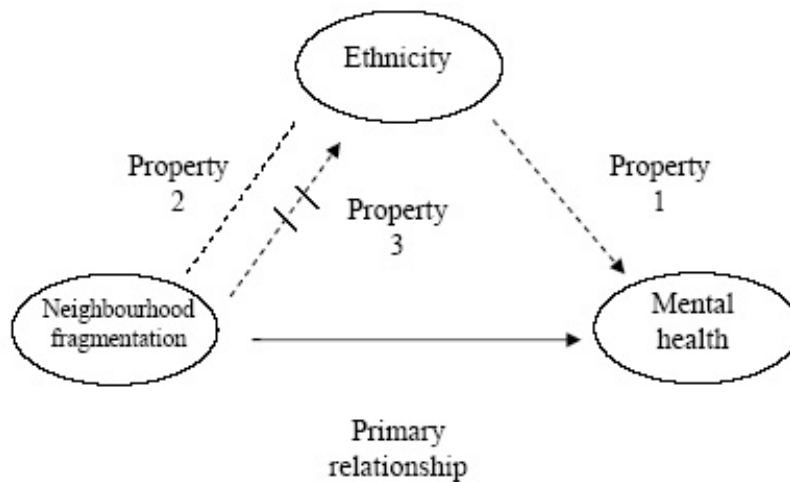


Figure 6:2 Ethnicity Confounding Triangle

Participation in education is commonly recognized as a risk factor for mental health (Gary et al., 2007, Harpham et al., 2004). Cross tabulations of the HS data have also demonstrated that post school education was associated with better mental health scores (property 1) (Figure 6:3). Individual levels of education qualifications have been shown to be predicted by NeighFrag (property 2), where those with higher levels of education qualifications are more likely to live in neighbourhoods with higher levels of NeighFrag (Table 3:5). The strength of local norms around education could potentially affect the likelihood of people gaining qualifications. However, it was considered unlikely that NeighFrag would have been a sufficiently strong causal influence on an individual's level of qualification to have violated property 3. Therefore levels of education qualifications were included as a possible individual level confounder.

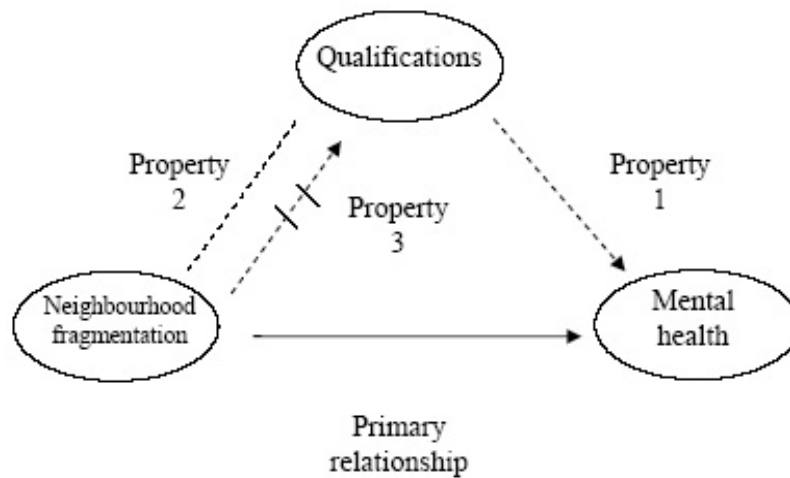


Figure 6:3 Education Confounding Triangle

Household Tenure is commonly used as a confounder in analyses of mental health outcomes (Phongsavan et al., 2006) suggesting that it could fulfil the first property (Figure 6:4). An association has already been demonstrated between tenure and NeighFrag with lower levels of homeownership in highly fragmented neighbourhoods (Table 3:5), fulfilling property 2. However, it can be argued that NeighFrag could have an effect on the likelihood of a house being rented; therefore any association between NeighFrag and mental health may be via household tenure, challenging the third property of a confounder. For this reason household tenure was not included as a potential confounder.

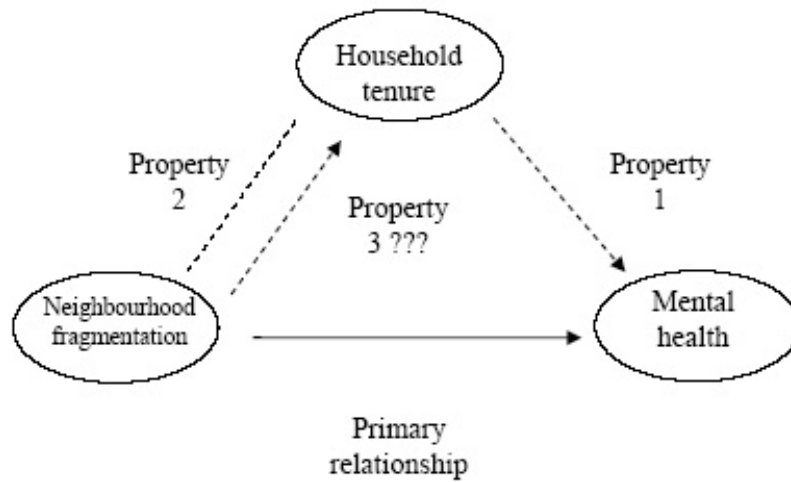


Figure 6:4 Household Tenure Confounding Triangle

Income is another marker of SES, and is commonly used as a confounder in analyses of mental health outcomes (Gary et al., 2007, Phongsavan et al., 2006), again suggesting that property 1 may be met (Figure 6:5). However, there was limited evidence for a relationship between income and NeighFrag (Table 3:5), challenging the second property. Therefore it was not included as a potential confounder in these analyses.

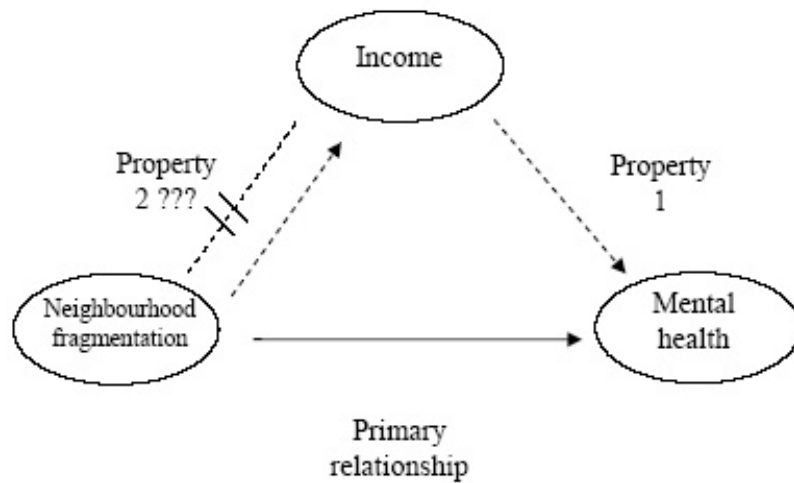


Figure 6:5 Income Confounding Triangle

The association between labour force status and mental health is well established in the literature and has commonly been used as a confounder in analyses of mental health outcomes (Phongsavan et al., 2006, Harpham et al., 2004) (property 1) (Figure 6:6). However, there is little evidence of an association between NeighFrag and labour force status in the 15-64 year old age group for whom labour force status questions are relevant (property 2) (Figure 3:11a-f). Therefore it was not included as a confounder.



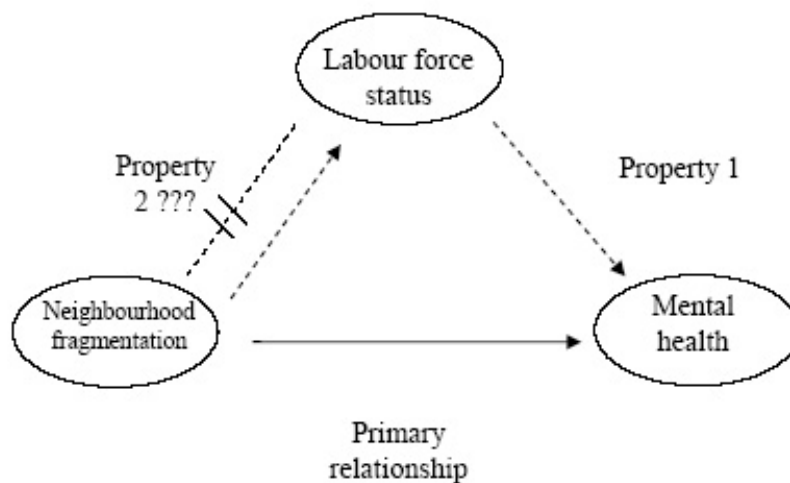


Figure 6:6 Labour Force Status Confounding Triangle

However the labour force status variable did provide an opportunity to consider if membership of a place of employment, for example, acts as an alternative social group, and therefore as a potential modifier to the effect of fragmentation.

Living alone was also considered as another potential indicator of social group membership. However, as the design variables already control for adjustment of the number of adults in the household it would not be sensible to include both variables in a single model. Using the 'adults in the household' as an effect modifier was also considered. Unfortunately, it did not take into account the number of children in the household and would therefore not be able to accurately distinguish between those living alone and those with another potential source of integration and regulation from their household. Another constraining factor was the high number of categories which meant that the stratum were too small to obtain reliable estimates.

The relationship between neighbourhood deprivation and fragmentation was unclear. As discussed above, area deprivation was a well established risk factor for individual mental health (Ministry of Health, 2004), fulfilling property 1 (Figure 6:7). As established earlier, NeighFrag and NZDep are moderately correlated (see above), the second property. However, it is not known to what extent area fragmentation may influence the level of deprivation in a neighbourhood, for example via historical processes. If this were so it would be acting as a mediator and including it in a regression could underestimate any association of NeighFrag with mental health. On the other hand if it was a confounder, the association of NeighFrag and mental health could be spurious were it not included. Taking a hierarchical modelling building approach allowed for careful examination of the independent contribution of both neighbourhood factors to the outcome. Therefore it will be included in the analyses as a potential confounder and modifier (to test for interaction between the two factors), with close observation of the results.

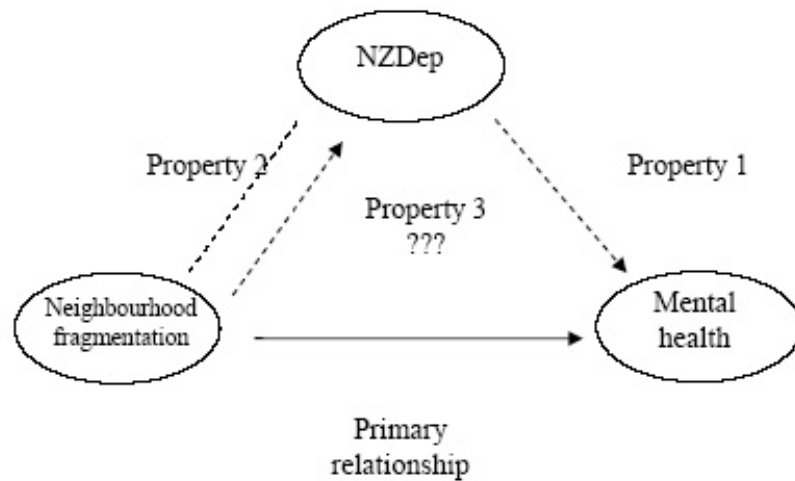


Figure 6:7 NZDep Confounding Triangle

Life-cycle age groups and sex were also considered as potential modifiers because of possible differences in the way different age and sex groups interact with the neighbourhood social environment (Wen et al., 2007, Kavanagh et al., 2006).

As reported above, a number of design variables were required to be included in the regression models as an alternative for the weightings provided. The variables capture the probability of being sampled by the various sampling stages. They include ethnicity (already specified as a potential confounder), the size of primary sampling unit (PSU), the number of sampling stratum, and the number of adults in a household.

#### **6.1.3.4 Covariate Specification**

All covariates were specified as categorical variables. A decision on the number of levels for each covariate was made after examination in cross tabs and in trial regressions. The aim was to use a parsimonious number of levels that provided meaningful contrast in the analyses, with the aim to follow the general rule of thumb that three to four levels would be required to satisfactorily adjust for any confounding (Rothman and Greenland, 1998).

*'Ethnicity'*: Because ethnicity was part of the sampling strategy it was necessarily included as a design variable, with four pre-determined categories: 'Māori'; 'Pacific People'; Asian; and 'non Māori, non Pacific, non Asian' ('other') (used as the reference group). The categories reflect the major groups of relevance to the New Zealand population and follow standard practice in New Zealand epidemiological analyses.

*'Education'*: highest education qualification was dichotomized into 'no qualifications' and 'school or post-school qualifications' (reference group).

'Age' was categorized into '15-24 years'; '25-44 years'; '45-64 years' (reference group) and '65+ years'. The '45-64' year age group was chosen as the reference group in order to make the baseline population more interpretable because it was felt that they would be the most exchangeable of the age groups (after much consideration). For example, it was considered less likely that a 65+ year old living in decile 10 would be similar to one living in moderate or low levels of fragmentation, compromising the exchangeability of the cells. On the other hand, 45-64 year olds may be more similar across the NeighFrag range (although not completely, of course). Therefore when discussing the effect of NeighFrag it was felt that a baseline population of 'other', with qualifications, 45-64 year olds was more interpretable than 'other', with qualifications, 65+ year olds.

'Labour force status' was categorized into three levels: 'employed' (reference group), 'unemployed', 'not in the labour force'.

'Sex' was dichotomized into 'male', 'female'

'NZDep96'. Neighbourhood deprivation was modelled as a categorical variable in quintiles, with 'least deprived' (quintile 1) as the reference group.

The specification details of the remaining design variables have been fully detailed in Appendix:1.

'PSU': the size of primary sampling unit, categorised from one to seven.

'Stratum': the number of sampling stratum, categorised from one to eight.

'Adults in Household': the number of adults in a household, categorised from one to five.

## 6.1.4 Statistical Methods

### 6.1.4.1 *Unadjusted Analyses*

The distribution of the study sample across NeighFrag was first explored, both with and without weights. Detailed information on the weighted univariate distribution of mental and general health scores is available elsewhere (Ministry of Health, 2004). While it was checked for confirmation purposes they are not reported here.

The bivariate relationships between mental and general health scores and exposures were examined to observe how the mean (with confidence intervals) SF36 score changed by level of exposure, before adjusting for confounding factors. The summary relationships are presented in the results by sex.

### 6.1.4.2 *Adjusted Analyses*

#### 6.1.4.2.1 *Main Effect*

Multilevel linear regressions were used to calculate the predicted changes in Mental Health score for NeighFrag, but now accounting for the clustered nature of the data and also progressively adjusting for confounders with the model building strategy discussed in Chapter Five. The dataset has variables at a number of levels of clustering. Properly speaking four levels can be observed and are listed below.

Level 1i : individual level variables.

Level 2j : household level - including the 'adults in the household' variable.

Level 3k : primary sampling unit (psu) - NZDep and NeighFrag (created at census area unit level but assigned to the psu in this dataset).

Level 4l : urban/rural

While in theory multilevel models can cope with this many levels, in reality the data available here would have been insufficient. Also, the research question was focused on the neighbourhood and individual levels. Therefore a two level structure was

imposed on the data. Individual and household level variables were specified as level 1i and psu's (geographic neighbourhood) as level 2j. As the analysis was restricted to the urban population the highest level did not matter here.

The model building process started with an empty model with no exposures or design variables to examine the baseline clustering of the outcome by neighbourhood, or psu's. Because of the need to use design variables instead of weights, it was not possible to calculate a reliable, interpretable baseline association of NeighFrag alone. Therefore the confounders were added into the model building process, starting with a model containing just the design variables (including ethnicity) and NeighFrag.

The model building process has been illustrated in Table 6:1. Variables were progressively added in at each stage. The main effect of NeighFrag on predicted Mental Health scores was modelled in four stages with the following equations -

Model 1: 'baseline model'

$$MH_{ij} = \beta_0 + (u_{0j} + e_{0ij})$$

Model 2: NeighFrag and design variables

$$MH_{ij} = \beta_0 + \beta NeighFrag_j + \beta design_{ij} + (u_{0j} + e_{0ij})$$

Model 3: plus individual confounders

$$MH_{ij} = \beta_0 + \beta NeighFrag_j + \beta design_{ij} + \beta age_{ij} + \beta Education_{ij} + (u_{0j} + e_{0ij})$$

Model 4: plus neighbourhood deprivation

$$MH_{ij} = \beta_0 + \beta NeighFrag_j + \beta design_{ij} + \beta age_{ij} + \beta Education_{ij} + \beta NZDep_j + (u_{0j} + e_{0ij})$$

The regression calculated the change in the predicted score (the slope) for each exposure category, relative to the reference group. 95% confidence intervals are reported.

Table 6:1 Model building for Mental Health Analyses

	<i>Cross-Level Main Effect Analyses</i>				<i>Within-Level Effect Modification Analyses</i>			
<i>variables</i>	<i>Model 1</i> <i>no exposures</i>	<i>Model 2</i> <i>with NeighFrag and design variables</i>	<i>Model 3</i> <i>with individual confounders</i>	<i>Model 4</i> <i>individual and neighbourhood confounders</i>	<i>Effect Measure Modification</i> <i>model 4 by effect modifier categories</i> <i>e.g. labour force status</i> <i>strata strata strata</i> <i>1 2 3</i>			<i>Model 5</i> <i>model 4 with NF*modifier</i>
<i>NeighFrag, design variables (including ethnicity)</i>		X	X	X	X		X	X
<i>Age Groups, Education</i>			X	X	X		X	X
<i>NZDep</i>				X	X		X	X
<i>NeighFrag*Modifier</i>								X
<i>Random Variance</i>	X	X	X	X	X	X	X	X

All analyses were conducted using SAS version 9. The regressions were run using PROC Mixed. The default in SAS is to use Restricted Maximum Likelihood methods to reduce bias in multilevel models. Getting the models to successfully converge was a problem. While a model could often be made to converge by changing the specification of the covariates there was no consistency across all models, and some models would not converge at all. Accordingly, following the advice of the biostatistician the SAS Parms statement was used to set the initial value at a fixed range for all models to obtain estimates that could be compared across all models (that is, using the same variable specifications). An iterative process was employed to determine the best fitting parameters once the final models were established, starting with the parameters obtained from successfully converged fully adjusted models.

F statistics provided information on the source outcome by calculating the ratio of the between- neighbourhood mean squares and within neighbourhood mean squares. The larger the difference between groups, the larger the F value will be. Confidence intervals and the p value of the F statistics were used to assess the statistically significant contribution of the variables and also the fit of the models.

#### *6.1.4.2.2 Effect Modification*

Fully adjusted analyses were run with the data stratified by the levels of each potential effect modifier. The resulting NeighFrag estimates were then compared across the strata for evidence of heterogeneity. If heterogeneity in estimates was present across the strata, confirmation of any modification was then sought from evidence of statistical interaction by including an interaction term in the regression equation with the following equation.



Statistical Interaction Model:

$$MH_{ij} = \beta_0 + \beta_{NeighFrag_j} + \beta_{design_{ij}} + \beta_{confounders_{ij}} + \beta_{EM_{ij}} + \beta_{NeighFrag * EM_{ij}} + (u_{0j} + e_{0ij})$$

-where the fully adjusted model (including design variables and confounders) also included the effect modifier (EM) and the product term NeighFrag\*EM.

As noted above, the study power to determine statistically significant interactions is often poor (Kirkwood et al., 2003). Therefore in order to reduce the number of parameters NeighFrag was specified using the 'continuous' version NeighFrag (continuous). While this only provided evidence of the linear association between NeighFrag and Mental Health this was sufficient for the purpose of examining evidence of statistical effect modification in conjunction with the stratified analyses.

#### 6.1.4.2.3 Secondary Questions

The magnitude and precision of the two neighbourhood exposures were examined at each stage of the hierarchical model building process. For example, the size of the NeighFrag estimate were compared before and after the addition of NZDep to assess whether the NeighFrag/mental health association was independent of NZDep, and vice versa.

The possibility of co-linearity and interaction between NZDep and NeighFrag was examined. Cross tabulations were used to examine correlation. In the adjusted analyses the width of confidence intervals were observed for substantial increases when NZDep was added to the model (Rothman and Greenland, 1998) that might point to violations of the statistical assumptions of the model.

Evidence of interaction between NeighFrag by NZDep was also observed. Using NeighFrag (continuous) to increase precision, an interaction term between NeighFrag (continuous) and NZDep was included in the regression model.

The categorical specification of NeighFrag was compared with the continuous version. Selected sets of models were run with NeighFrag (continuous) substituted for the categorical version of NeighFrag. The resulting estimates were compared for consistency in the observed relationships to ensure that any observed effect was not mainly attributable to the way in which NeighFrag was modelled.

All main effect analyses were repeated using NeighFrag (Congdon). Only the fully adjusted results are shown here.

## 6.2 Results

### 6.2.1 Health Survey Dataset

The weighted distribution of NZHS survey participants by NeighFrag mirrors that of the 2001 census population. The dataset was first restricted to those with complete data resulting in a small reduction, less than a 2% (Table 6:2). Analyses were only done for the urban population, just over three quarters of the total dataset.

Table 6:2 Dataset Restriction Process

	<i>Number of Participants &amp; Percentage of total Dataset by Restriction Factors</i>					
	<i>Female</i>		<i>Male</i>		<i>Total</i>	
	<i>n</i>	<i>%</i>	<i>n</i>	<i>%</i>	<i>n</i>	<i>%</i>
<i>Whole Data Set</i>	7,658	61.1	4,871	38.9	12,529	100
<i>Complete Data</i>	7,552	60.2	4,816	38.4	12,368	98.7
<i>Urban</i>	5,995	47.8	3,764	30.0	9,759	77.9

Further details of the dataset characteristics have been fully reported in *A Portrait of Health* (Ministry of Health, 2004). There were important differences in the response

rate by key demographic characteristics (as illustrated in table 6:2) by the larger proportion of women respondents. The NZHS has survey weights to allow analyses that could be adjusted for the differing response rates and the over sampling survey design. The unadjusted analyses were completed using the provided survey weights. However, as discussed above, design variables were used as covariates in the adjusted regression analyses, rather than the problematic survey weights.

### **6.2.2 Unadjusted Analyses**

The weighted distribution of mental health scores have been summarized by individual and neighbourhood characteristics, for women and men respectively, in Tables 6:3a and b. The analyses are bivariate only, and are not adjusted for covariates. Overall, women report slightly lower MH scores than men, illustrated in Figure 6:1 (results reported in the Portrait of Health suggest that while the differences were small they were statistically significant (Ministry of Health, 2004)). The predictors of better or poorer scores are similar within each sex group but with a few variations. Unemployment, no qualifications, younger age group and living in more fragmented and deprived neighbourhoods all predict the worst mental health scores for both men and women. For women, identifying as Māori was associated with the poorest scores, whereas for men those with the poorest scores identified as Pacific people. Conversely, identifying as non-Māori, non-Pacific, non-Asian, ('other'), or being employed, or having more education qualifications, being older, or living in the least fragmented neighbourhoods and least deprived neighbourhoods all predicted better mental health scores.

Table 6:3a The Unadjusted, Weighted Distribution of SF36 Mental Health Scale Scores by Exposure Categorical Variables:  
Women

<i>Exposure</i>	<i>n</i>	<i>SF36 Mental Health Scale</i>		<i>95% CI</i>	
		<i>mean</i>	<i>lower</i>	<i>upper</i>	
<b><i>Ethnicity</i></b>					
<i>Asian</i>	668	82.32	81.18	83.45	
<i>Maori</i>	2,648	79.78	79.16	80.41	
<i>Pacific</i>	561	81.44	80.02	82.86	
<i>other</i>	3,680	82.48	82.03	82.93	
<b><i>Labour force status</i></b>					
<i>not in labour force</i>	3,383	81.19	80.65	81.73	
<i>unemployed</i>	330	76.03	74.19	77.87	
<i>employed</i>	3,844	83.01	82.60	83.42	
<b><i>Education</i></b>					
<i>no qualifications</i>	2,243	81.52	80.85	82.19	
<i>school qualifications</i>	2,128	81.53	80.91	82.14	
<i>post school quals.</i>	3,186	82.85	82.39	83.31	
<b><i>Age groups</i></b>					
<i>15-24 years</i>	902	78.23	77.18	79.28	
<i>25-44 years</i>	3,227	81.82	81.35	82.29	
<i>45-64 years</i>	2,140	83.44	82.84	84.05	
<i>65+ years</i>	1,288	84.48	83.73	85.24	
<b><i>NeighFrag</i></b>					
<i>NF 1</i>	266	86.37	85.16	87.58	
<i>NF 2,3</i>	1,033	82.64	81.85	83.44	
<i>NF 4,5</i>	1,574	84.09	83.47	84.72	
<i>NF 6,7</i>	1,805	82.18	81.49	82.87	
<i>NF 8,9</i>	2,075	80.85	80.19	81.51	
<i>NF10</i>	804	79.80	78.70	80.89	
<b><i>NZDep</i></b>					
<i>Most deprived</i>	962	83.93	83.13	84.73	
<i>Quintile 2</i>	936	83.00	82.14	83.85	
<i>Quintile 3</i>	1,097	82.54	81.75	83.33	
<i>Quintile 4</i>	1,449	81.14	80.35	81.93	
<i>Most deprived</i>	3,113	80.28	79.70	80.85	

Table 6:3b The Unadjusted, Weighted Distribution of SF36 Mental Health Scale Scores by Exposure Categorical Variables: Men

<i>Exposure</i>	<i>n</i>	<i>SF36 Mental Health Scale</i>		<i>95% CI</i>	
		<i>mean</i>	<i>lower</i>	<i>upper</i>	
<b><i>Ethnicity</i></b>					
<i>Asian</i>	493	84.19	83.02	85.36	
<i>Maori</i>	1,406	83.57	82.83	84.31	
<i>Pacific</i>	328	82.58	80.95	84.20	
<i>other</i>	2,593	84.90	84.41	85.39	
<b><i>Labour force status</i></b>					
<i>not in labour force</i>	1,567	83.22	82.47	83.98	
<i>unemployed</i>	207	81.77	79.71	83.83	
<i>employed</i>	3,046	85.24	84.81	85.66	
<b><i>Education</i></b>					
<i>no qualifications</i>	1,378	83.76	82.96	84.56	
<i>school qualifications</i>	1,092	84.34	83.61	85.08	
<i>post school quals.</i>	2,350	85.11	84.61	85.61	
<b><i>Age groups</i></b>					
<i>15-24 years</i>	630	83.06	82.01	84.11	
<i>25-44 years</i>	1,757	83.91	83.29	84.53	
<i>45-64 years</i>	1,544	85.34	84.73	85.95	
<i>65+ years</i>	889	87.07	86.20	87.95	
<b><i>NeighFrag</i></b>					
<i>NF 1</i>	181	86.46	84.74	88.19	
<i>NF 2,3</i>	722	85.29	84.39	86.19	
<i>NF 4,5</i>	957	86.02	85.27	86.77	
<i>NF 6,7</i>	1132	84.40	83.62	85.18	
<i>NF 8,9</i>	1257	84.02	83.26	84.78	
<i>NF10</i>	571	82.79	81.68	83.91	
<b><i>NZDep</i></b>					
<i>Most deprived</i>	731	86.01	85.22	86.81	
<i>Quintile 2</i>	626	84.41	83.33	85.50	
<i>Quintile 3</i>	743	84.40	83.51	85.30	
<i>Quintile 4</i>	907	85.14	84.33	85.95	
<i>Most deprived</i>	1,813	83.14	82.45	83.84	

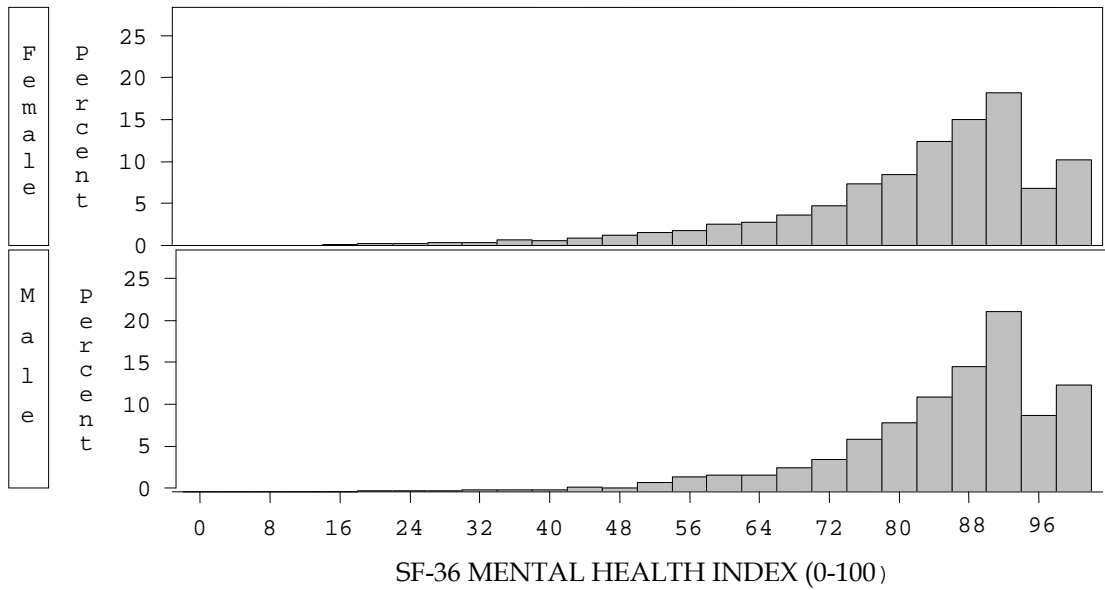


Figure 6:8 The NeighFrag Distribution of SF-36 Mental Health Scores, by Sex

The unadjusted relationship between General Health and the levels of the various exposures are summarized in Tables 6:4:a and b. Overall, better GH scores are reported by the employed, those with school or post school qualifications, and those living in the least deprived and fragmented neighbourhoods. Gender differences in the association of General Health and ethnicity were observed, where Pacific men reported the best general health, whereas Asian women reported the highest scores. The association between age and General Health also varied by gender: 25-44 year old women reported the best general health, whereas 15-24 year old men had higher scores than other age groups.

Table 6:4a The Weighted Distribution of SF36 General Health Scale Scores by Exposure Categorical Variables: Women

<i>Exposure</i>	<i>SF36 General Health Scale</i>		<i>95% CI</i>	
	<i>n</i>	<i>Mean</i>	<i>Lower</i>	<i>Upper</i>
<b><i>Ethnicity</i></b>				
<i>Asian</i>	668	77.86	76.50	79.23
<i>Maori</i>	2,647	71.97	71.12	72.81
<i>Pacific</i>	562	75.33	73.70	76.97
<i>Other</i>	3,681	75.89	75.24	76.53
<b><i>Employment</i></b>				
<i>not in labour force</i>	3,383	71.70	70.95	72.44
<i>unemployed</i>	330	72.66	70.49	74.82
<i>employed</i>	3,845	78.14	77.56	78.72
<b><i>Education</i></b>				
<i>no qualifications</i>	2,244	72.61	71.72	73.50
<i>school qualifications</i>	2,128	76.06	75.23	76.90
<i>Post school quals.</i>	3,186	76.67	75.99	77.36
<b><i>Age group</i></b>				
<i>15-24 years</i>	901	71.76	70.42	73.10
<i>25-44 years</i>	3,229	78.15	77.50	78.80
<i>45-64 years</i>	2,140	76.25	75.38	77.12
<i>65+ years</i>	1,288	71.68	70.53	72.83
<b><i>NeighFrag</i></b>				
<i>NF 1</i>	266	80.77	78.54	83.01
<i>NF 2,3</i>	1,033	77.40	76.23	78.56
<i>NF 4,5</i>	1,573	76.22	75.25	77.19
<i>NF 6,7</i>	1,806	75.80	74.88	76.72
<i>NF 8,9</i>	2,076	74.57	73.68	75.46
<i>NF10</i>	804	72.36	70.91	73.82
<b><i>NZDep</i></b>				
<i>Most deprived</i>	962	78.77	77.64	79.90
<i>Quintile 2</i>	936	78.03	76.81	79.25
<i>Quintile 3</i>	1,097	75.22	74.04	76.40
<i>Quintile 4</i>	1,449	72.85	71.76	73.95
<i>Most deprived</i>	3,114	73.48	72.73	74.23

Table6:4b The Weighted Distribution of SF36 General Health Scale Scores by Exposure Categorical Variables: Men

<i>Exposure</i>	<i>SF36 General Health Scale</i>		<i>95% CI</i>	
	<i>n</i>	<i>Mean</i>	<i>Lower</i>	<i>Upper</i>
<b><i>Ethnicity</i></b>				
<i>Asian</i>	493	78.19	76.45	79.93
<i>Maori</i>	1,406	73.04	72.00	74.07
<i>Pacific</i>	328	80.11	78.09	82.13
<i>Other</i>	2,593	75.55	74.81	76.28
<b><i>Labour Force Status</i></b>				
<i>not in labour force</i>	1,567	69.60	68.48	70.71
<i>unemployed</i>	207	69.56	66.45	72.67
<i>employed</i>	3,046	77.99	77.38	78.60
<b><i>Education</i></b>				
<i>no qualifications</i>	1,378	72.55	71.43	73.67
<i>school qualifications</i>	1,092	76.86	75.73	77.99
<i>Post school quals.</i>	2,350	76.28	75.53	77.02
<b><i>Age groups</i></b>				
<i>15-24 years</i>	630	78.45	77.07	79.84
<i>25-44 years</i>	1,757	77.56	76.72	78.41
<i>45-64 years</i>	1,544	74.71	73.74	75.68
<i>65+ years</i>	889	68.51	67.06	69.95
<b><i>NeighFrag</i></b>				
<i>NF 1</i>	181	78.81	76.27	81.34
<i>NF 2,3</i>	722	76.82	75.52	78.12
<i>NF 4,5</i>	957	75.73	74.51	76.94
<i>NF 6,7</i>	1,132	73.92	72.76	75.07
<i>NF 8,9</i>	1,257	75.90	74.80	77.00
<i>NF10</i>	571	75.13	73.56	76.69
<b><i>NZDep</i></b>				
<i>Most deprived</i>	731	80.00	78.83	81.16
<i>Quintile 2</i>	626	75.72	74.25	77.18
<i>Quintile 3</i>	743	74.91	73.48	76.33
<i>Quintile 4</i>	907	74.21	72.93	75.50
<i>Most deprived</i>	1,813	73.64	72.68	74.60



Evidence was sought for possible co-linearity between the two neighbourhood measures. Tables 6:5a-d examines the distribution of survey participants by NeighFrag and NZDep across the whole survey population. At this level the results supported the moderate correlation observed above but also demonstrated that there were participants from a range of NZDep levels living in the full range of NeighFrag. However, once the survey population was first restricted to urban (Table 6:5b) and then stratified by sex (Table 6:5c&d), the reduced numbers in NF1 resulted in sparse data in these cells, noticeably at moderate levels of deprivation.

From a purely statistical point of view it may be preferable to exclude NZDep from the models because of the challenges posed by multicollinearity and sparse data. However, leaving NZDep out of the model would be to omit valuable information about how NeighFrag works in the New Zealand population, the primary purpose of the analysis. So with the research questions in mind NZDep was included but the results were observed for the possible effect of collinearity, for example, increasing standard errors, widening confidence intervals and unstable estimates (Gujarati, 1999). The NF1 estimates in particular would also need to be interpreted carefully, given the sparse data.

## **6.2.3 Adjusted Analyses**

### ***6.2.3.1 Cross-level Main Effect***

#### ***6.2.3.1.1 NeighFrag and Mental Health***

The association between NeighFrag and mental health has been shown in Tables 6:6 and 6:7 for women and men respectively. The estimates show the predicted changes in Mental Health scores by each exposure level in the four models.

Table 6:5a-d Cross tabulations of NeighFrag and NZDep: population numbers across the restriction process

<i>A: Total Survey</i>	<i>NeighFrag decile groups</i>						
<i>NZDep quintiles</i>	<i>NF 1</i>	<i>NF 2,3</i>	<i>ref N 4,5</i>	<i>NF 6,7</i>	<i>NF 8,9</i>	<i>NF 10</i>	<i>total</i>
<i>Quintile 1</i>	190	477	393	246	297	89	1,692
<i>Quintile 2</i>	88	310	369	288	403	103	1,561
<i>Quintile 3</i>	25	282	337	489	522	183	1,838
<i>Quintile 4</i>	18	262	502	486	735	353	2,356
<i>Quintile 5</i>	126	424	927	1,425	1,372	647	4,921
<i>total</i>	447	1,755	2,528	2,934	3,329	1,375	1,2368

<i>B: Urban</i>	<i>NeighFrag decile groups</i>						
<i>NZDep quintiles</i>	<i>NF 1</i>	<i>NF 2,3</i>	<i>ref N 4,5</i>	<i>NF 6,7</i>	<i>NF 8,9</i>	<i>NF 10</i>	<i>total</i>
<i>Quintile 1</i>	82	352	342	246	279	89	1,390
<i>Quintile 2</i>	28	157	296	271	403	103	1,258
<i>Quintile 3</i>	-	168	225	467	522	183	1,570
<i>Quintile 4</i>	18	123	370	445	734	353	2,043
<i>Quintile 5</i>	26	98	507	881	1,354	632	3,498
<i>total</i>	159	898	1,740	2,310	3,292	1,360	9,759

<i>C: Female, Urban</i>	<i>NeighFrag decile groups</i>						
<i>NZDep quintiles</i>	<i>NF 1</i>	<i>NF 2,3</i>	<i>ref NF 4,5</i>	<i>NF 6,7</i>	<i>NF 8,9</i>	<i>NF 10</i>	<i>total</i>
<i>Quintile 1</i>	42	198	211	141	148	44	784
<i>Quintile 2</i>	20	98	187	148	246	65	764
<i>Quintile 3</i>	-	104	149	278	314	102	949
<i>Quintile 4</i>	15	63	223	271	463	222	1,257
<i>Quintile 5</i>	18	64	324	589	884	362	2,241
<i>total</i>	97	527	1,094	1,427	2,055	795	5,995

<i>D: Male, Urban</i>	<i>NeighFrag decile groups</i>						
<i>NZDep quintiles</i>	<i>NF 1</i>	<i>NF 2,3</i>	<i>ref NF 4,5</i>	<i>NF 6,7</i>	<i>NF 8,9</i>	<i>NF 10</i>	<i>total</i>
<i>Quintile 1</i>	40	154	131	105	131	45	606
<i>Quintile 2</i>	-	59	109	123	157	38	494
<i>Quintile 3</i>	-	64	76	189	208	81	621
<i>Quintile 4</i>	-	60	147	174	271	131	786
<i>Quintile 5</i>	-	34	183	292	470	270	1,257
<i>total</i>	62	371	646	883	1,237	565	3,764

Cells with counts less than 10 indicated with a - , in keeping with confidentiality requirements

The estimates for both the categorical and continuous specification of NeighFrag within the same table for ease of comparison (NeighFrag (continuous) is shown separately at the bottom of the table to avoid confusion). Note, they were not included in the model simultaneously and the continuous NeighFrag estimate only is presented. The accompanying covariate results were not shown as they were very similar to the ones shown for the categorical NeighFrag estimate. The results will be reported for women then men.

Starting with model 2 (just NeighFrag and design variables), the estimates for NeighFrag indicated that urban women living in neighbourhoods with progressively higher levels of fragmentation reported decreasingly poorer mental health compared to those living in moderately fragmented neighbourhoods. The NF estimate for each decile group represents the difference in the predicted change in mental health (MH) score from the reference group (that is, the intercept). The association shows some evidence of a dose response as the estimate decreases in a monotonic fashion for each decile grouping above the moderate reference group. So, the predicted difference in MH for NF6,7 compared to NF4,5 was **-2.04** (-3.39- -0.68); NF8,9 and NF4,5 was **-2.84** (-4.15- -1.53); and NF10 and NF4,5 was **-3.36** (-5.05 - -1.68). All of the estimates (highlighted in bold) in this range of fragmentation were statistically significant with the 95% confidence intervals excluding 0.00.

For deciles below the reference group, the pattern was less clear. The negative estimate for NF2,3 compared to NF4,5 was **-1.10** (-2.85 - 0.64), indicating that poorer mental health was reported by these women compared to the reference group. The women in the least fragmented neighbourhoods, NF1 reported better mental health compared to the reference group, with a positive estimate of **2.63** (-0.81 - 6.07). None of the estimates for the deciles below NF4,5 were statistically significant with the confidence intervals including 0.00. Nevertheless the pattern across NeighFrag categories is mostly linear.

Table 6:6 NeighFrag and SF-36 Mental Health Scale: Women (95% Confidence Intervals)

	<i>Predicted change in mental health score</i>			
	<i>Model 1</i>	<i>Model 2</i>	<i>Model 3</i>	<i>Model 4</i>
<b>Intercept</b>	81.74 (81.31 - 82.18)	90.26 (82.80 - 97.72)	93.97 (86.48 - 101.46)	95.68 (88.06 - 103.31)
<b>NeighFrag</b>				
NF 1		2.63 (-0.81 - 6.07)	2.59 (-0.82 - 6.01)	2.33 (-1.10 - 5.75)
NF 2,3		-1.10 (-2.85 - 0.64)	-1.23 (-2.96 - 0.50)	-1.49 (-3.24 - 0.25)
NF 4,5 (ref)		0.00	0.00	0.00
NF 6,7		<b>-2.04 (-3.39 - -0.68)</b>	<b>-1.89 (-3.23 - -0.54)</b>	<b>-1.78 (-3.13 - -0.43)</b>
NF 8,9		<b>-2.84 (-4.15 - -1.53)</b>	<b>-2.64 (-3.94 - -1.34)</b>	<b>-2.37 (-3.69 - -1.04)</b>
NF10		<b>-3.36 (-5.05 - -1.68)</b>	<b>-2.78 (-4.46 - -1.10)</b>	<b>-2.45 (-4.15 - -0.74)</b>
<b>Ethnicity</b>				
Asian		0.53 (-0.97 - 2.04)	1.26 (-0.25 - 2.78)	1.27 (-0.24 - 2.78)
Maori		<b>-1.89 (-2.98 - -0.80)</b>	-0.51 (-1.63 - 0.60)	-0.24 (-1.38 - 0.90)
Pacific		-0.44 (-2.08 - 1.19)	0.56 (-1.07 - 2.20)	0.92 (-0.75 - 2.59)
other		0.00	0.00	0.00
<b>Education</b>				
No qualifications			<b>-2.32 (-3.24 - -1.41)</b>	<b>-2.20 (-3.13 - -1.28)</b>
School or post school qualifications			0.00	0.00
<b>Age groups</b>				
15-24 yrs			<b>-4.56 (-5.94 - -3.18)</b>	<b>-4.52 (-5.90 - -3.14)</b>
25-44 yrs			<b>-2.02 (-3.00 - -1.05)</b>	<b>-2.01 (-2.98 - -1.03)</b>
45-64 yrs (ref)			0.00	0.00
65+ yrs			<b>2.65 (1.42 - 3.89)</b>	<b>2.67 (1.44 - 3.91)</b>
<b>NZDep2001 Quintiles</b>				
least deprived				0.00
Quintile 2				-1.16 (-2.83 - 0.51)
Quintile 3				-0.91 (-2.53 - 0.71)
Quintile 4				-1.40 (-2.98 - 0.19)
Most deprived				<b>-2.11 (-3.88 - -0.34)</b>
<b>Random var.(s.e of variance)</b>				
Neighbourhood-level	7.90 (1.64)	7.83 (1.81)	7.71 (1.79)	7.71 (1.81)
Individual-level	225.84 (4.34)	223.80 (4.31)	220.25 (4.24)	220.19 (4.24)
ICC	0.034	0.034	0.034	0.034
<b>NeighFrag(continuous)</b>		<b>-4.70 (-6.70 - -2.70)</b>	<b>-4.01 (-6.01 - -2.02)</b>	<b>-3.29 (-5.39 - -1.19)</b>

Model 1 design variables only (including ethnicity)

Model 2 NeighFrag and design variables (including ethnicity),

Model 3 NeighFrag and individual confounders (age, education, and ethnicity & design variables)

Model 4 NeighFrag and individual confounders (age, education, and ethnicity & design variables) and neighbourhood NZDep, by sex

Bold indicates significance at the 95% confidence level

Further evidence of a dose response for women across the full range of fragmentation can be seen in the estimate for the NeighFrag (continuous) variable (bottom of Table 6:6). Here, the estimated change in score from least to most fragmentation, -4.70 (-5.05 - -1.68), was smaller than the total difference observed across the full range of decile categories -5.99 (-3.36 minus 2.63). But it was also larger than the difference between decile NF 4,5 and decile NF 10, -3.36 (-5.05 - -1.68). The lack of monotonicity in the moderate deciles may explain these differences, but overall there was some support for a linear association, and therefore the use of the more parsimonious continuous variable. The value of the F statistics obtained for the NeighFrag(continuous) in model 2 was 5.99 (<0.01), suggesting that the variable contributed a statistically significant amount of variation in the outcome, over and above the variation contributed by the other variables included in the regression.

Model three and four (Table 6:6) show the effect of first adding individual and then neighbourhood confounders to the model. Confounding exposures accounted for approximately a third of the NeighFrag/MH association seen in model 2, the least adjusted regression. The association of NeighFrag(continuous) on Mental Health was reduced by approximately 15% (that is a change from -4.70 to -4.01) when individual confounders were added, indicating that the relationship in model 1 was confounded by the individual risk factors of age and labour force status. The effect was reduced again by approximately 18% (a change from -4.01 to -3.29) when neighbourhood deprivation was added to the equation, providing support for its inclusion as a confounder in the regression model (Rothman and Greenland, 1998).

While the magnitude of the association was reduced after controlling for confounding, the nature of the association remained. The size of the negative association increased relative to the reference levels with increasing levels of fragmentation and remained statistically significant. The estimate for decile NF 1 remained positive, relative to the reference levels, but was not statistically significant as the confidence intervals included 0.00. The estimate for NeighFrag (continuous)

remained significant. The fully adjusted association between NeighFrag and mental health in urban women is summarized in Figure 6:9. The values of the F statistics reduced as individual confounders were added into the model (4.91(<0.01)) and then NZDep (3.70(<0.01)). The reduction reinforced the evidence above of confounding but also highlighted that the contribution of NeighFrag to variation in the outcome remained even when other important factors were included.

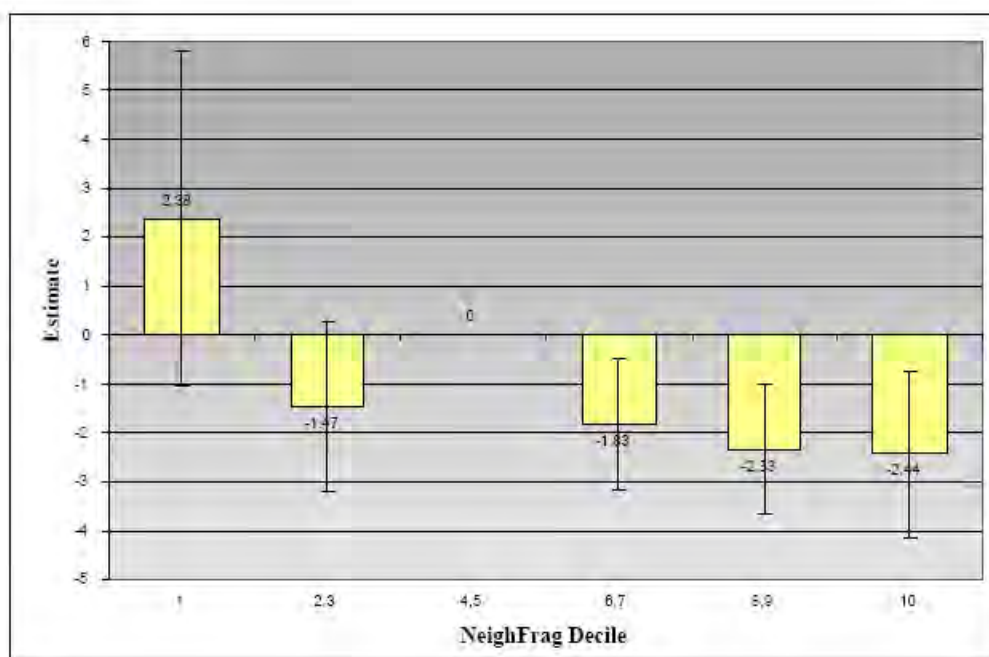


Figure 6:9 Predicted Change in Mental Health Score by NeighFrag: Women (Fully Adjusted, 95% Confidence Intervals)

There was no convincing evidence of an association of neighbourhood fragmentation with the self-reported mental health of urban men, after controlling for confounders (Table 6:7). A statistically significant negative association was observed for decile NF 10 in model 2, but not at other levels of NeighFrag. After adjusting for individual confounders the estimate was reduced by a third and was no longer statistically significant. Additional adjustment for NZDep decreased the estimate further. Further, small, negative estimate for NeighFrag (continuous) was observed but it was not statistically significant. In keeping with the fixed effect estimates the F statistics were all close to 1 and did not have statistically significant p values. However, it

should also be noted that men's health did vary by individual factors, and that neighbourhood deprivation was an important contextual predictor of their mental health status.

#### *6.2.3.1.2 Covariates and Mental Health*

As seen in table 6.6 and 6.7 in the adjusted analyses, age group had the largest impact on predicted mental health scores, relative to the reference category. Compared to the 45-64 year old reference population, younger women were predicted to report mental health that was over 4 points worse, whereas those who were 65+ years were likely to report almost 3 points better. The same pattern was observed for men, but on a smaller scale.

An association was also observed for education qualifications. In both men and women, having no qualifications was significantly associated with reporting worse mental health. The size of the association, however, was smaller than that observed for living in NeighFrag deciles 8-10 compared to the moderate reference group (unusually for a neighbourhood characteristic (Diez-Roux, 2000)). No significant association for the ethnicity was observed for women, once other individual and neighbourhood factors were included. Asian men, on the other hand were predicted to have slightly better mental health than the reference group.

The inclusion of NZDep significantly changed the NeighFrag estimates, indicating that it was an important factor (and possibly a confounder). In the fully adjusted model a statistically significant evidence of an independent association of NZDep and mental health was only present for those living in the most deprived compared with the least deprived neighbourhoods. Further, the magnitude of the association across the range of NZDep appeared smaller than across NeighFrag.

Table 6:7 NeighFrag and SF-36 Mental Health Scale: Men (95% Confidence Intervals)

	<i>Predicted Change in Mental Health Score</i>			
	<i>Model 1</i>	<i>Model 2</i>	<i>Model 3</i>	<i>Model 4</i>
<b>Intercept</b>	84.57 (84.11 - 85.04)	91.68 (84.22 - 99.15)	93.26 (85.74 - 100.79)	94.94 (87.29 - 102.60)
<b>NeighFrag</b>				
NF 1		-0.16 (-3.82 - 3.49)	-0.06 (-3.70 - 3.57)	-0.43 (-4.10 - 3.23)
NF 2,3		-0.40 (-2.23 - 1.43)	-0.41 (-2.23 - 1.41)	-0.62 (-2.45 - 1.22)
NF 4,5 (ref)		0.00	0.00	0.00
NF 6,7		-0.67 (-2.13 - 0.79)	-0.46 (-1.91 - 1.00)	-0.33 (-1.79 - 1.14)
NF 8,9		-1.15 (-2.57 - 0.28)	-0.90 (-2.32 - 0.51)	-0.67 (-2.10 - 0.77)
NF10		<b>-2.02 (-3.79 - -0.26)</b>	-1.37 (-3.14 - 0.39)	-1.10 (-2.89 - 0.69)
<b>Ethnicity</b>				
Asian		1.07 (-0.47 - 2.60)	<b>1.74 ( 0.19 - 3.28)</b>	<b>1.78 ( 0.23 - 3.33)</b>
Maori		<b>-1.27 (-2.51 - -0.02)</b>	-0.29 (-1.56 - 0.99)	-0.14 (-1.44 - 1.16)
Pacific		-1.35 (-3.11 - 0.42)	-0.64 (-2.40 - 1.13)	-0.34 (-2.14 - 1.46)
other		0.00	0.00	0.00
<b>Education</b>				
No qualifications			<b>-1.17 (-2.22 - -0.13)</b>	<b>-1.11 (-2.17 - -0.06)</b>
School or post school qualifications			0.00	0.00
<b>Age Groups</b>				
15-24 yrs			<b>-2.25 (-3.75 - -0.76)</b>	<b>-2.23 (-3.72 - -0.73)</b>
25-44 yrs			<b>-1.39 (-2.46 - -0.33)</b>	<b>-1.38 (-2.45 - -0.32)</b>
45-64 yrs (ref)			0.00	0.00
65+ yrs			<b>2.77 ( 1.51 - 4.04)</b>	<b>2.82 ( 1.55 - 4.08)</b>
<b>NZDep2001 Quintiles</b>				
least deprived				0.00
Quintile 2				-1.21 (-2.92 - 0.49)
Quintile 3				-1.24 (-2.89 - 0.42)
Quintile 4				-0.47 (-2.09 - 1.15)
Most deprived				<b>-2.04 (-3.81 - -0.26)</b>
<b>Random var.(s.e of variance)</b>				
Neighbourhood-level	6.02 (1.93)	5.98 (2.12)	5.90 (2.07)	5.90 (2.07)
Individual-level	172.03 (4.33)	170.75 (4.34)	168.64 (4.28)	168.51 (4.28)
ICC	0.034	0.034	0.034	0.034
<b>NeighFrag(continuous)</b>		-2.00 (-4.08 - 0.09)	-1.32 (-3.41 - 0.76)	-0.67 (-2.86 - 1.51)

Model 1 design variables only (including ethnicity)

Model 2 NeighFrag and design variables (including ethnicity),

Model 3 NeighFrag and individual confounders (age, education, and ethnicity & design variables)

Model 4 NeighFrag and individual confounders (age, education, and ethnicity & design variables) and neighbourhood NZDep, by sex

Bold indicates significance at the 95% confidence level



The addition of deprivation into the model only slightly attenuated the effect of NeighFrag, indicating independence between the two neighbourhood exposures in their association with the outcome. This was confirmed in separate analyses reversing the order in which neighbourhood exposures were added (not shown). NZDep was initially positively associated with mental health, with some indication of a dose response. Once NeighFrag was added the association of NZDep with mental health largely disappeared for all but the most deprived neighbourhoods. The width of the confidence intervals around the NeighFrag estimates remained stable when NZDep was added to the model, suggesting no major statistical problems due to correlated ecological exposures (Rothman and Greenland, 1998). The significant p values of the F statistic for women confirmed that the non-normal distribution of the outcome measure did not unduly compromise the fit of the model.

### **6.2.3.2 Other SF-36 Health Scales**

#### *6.2.3.2.1 General Health*

While it would appear that NeighFrag is associated with health when controlling for individual confounders, there is limited evidence for an association once neighbourhood deprivation is included in the model. Table 6:8 reports the estimated changes in predicted General Health scores for men and women of model 3, adjusting for individual confounders, and then model 4, adjusted for both individual characteristics and neighbourhood deprivation. For brevity, only models 3 and 4 are shown here.

Table 6:8 NeighFrag and SF-36 General Health Scale

	<i>Predicted Change in General Health Score</i>			
	<i>Model 3</i>		<i>Model 4</i>	
	<i>Male</i>	<i>Female</i>	<i>Male</i>	<i>Female</i>
<b>Intercept</b>	74.50 (63.18 - 85.82)	79.33 (68.98 - 89.68)	79.08 (67.61 - 90.56)	82.51 (71.99 - 93.02)
<b>NeighFrag NF 1</b>	5.34 (-0.01 - 10.70)	2.76 (-1.84 - 7.35)	3.40 (-2.00 - 8.79)	2.06 (-2.56 - 6.67)
NF 2,3	1.06 (-1.63 - 3.74)	0.83 (-1.50 - 3.17)	0.14 (-2.56 - 2.85)	0.19 (-2.17 - 2.54)
NF 4,5 (ref)	0.00	0.00	0.00	0.00
NF 6,7	-0.70 (-2.85 - 1.45)	-1.62 (-3.43 - 0.20)	-0.22 (-2.38 - 1.94)	-1.27 (-3.09 - 0.55)
NF 8,9	-0.09 (-2.18 - 2.01)	<b>-2.25 (-4.01 - -0.50)</b>	0.60 (-1.51 - 2.72)	-1.60 (-3.38 - 0.19)
NF10	-1.49 (-4.10 - 1.12)	<b>-3.86 (-6.13 - -1.59)</b>	-0.55 (-3.19 - 2.09)	<b>-2.93 (-5.24 - -0.63)</b>
<b>Ethnicity</b>				
Asian	<b>3.40 ( 1.14 - 5.67)</b>	<b>2.32 ( 0.30 - 4.35)</b>	<b>3.57 ( 1.31 - 5.84)</b>	<b>2.39 ( 0.36 - 4.41)</b>
Maori	<b>-3.01 (-4.88 - -1.15)</b>	<b>-2.30 (-3.79 - -0.81)</b>	<b>-2.28 (-4.18 - -0.39)</b>	<b>-1.61 (-3.13 - -0.08)</b>
Pacific	<b>2.79 ( 0.19 - 5.38)</b>	-0.09 (-2.27 - 2.10)	<b>3.55 ( 0.92 - 6.18)</b>	0.70 (-1.53 - 2.93)
other	0.00	0.00	0.00	0.00
<b>Education</b>				
No qualifications	<b>-2.99 (-4.52 - -1.46)</b>	<b>-3.44 (-4.67 - -2.22)</b>	<b>-2.66 (-4.20 - -1.12)</b>	<b>-3.13 (-4.36 - -1.90)</b>
School or post school qualifications	0.00	0.00	0.00	0.00
<b>Age Groups</b>				
15-24 yrs	<b>5.01 ( 2.83 - 7.20)</b>	-0.47 (-2.31 - 1.38)	<b>5.26 ( 3.08 - 7.45)</b>	-0.34 (-2.18 - 1.50)
25-44 yrs	<b>4.13 ( 2.57 - 5.69)</b>	<b>2.02 ( 0.72 - 3.31)</b>	<b>4.27 ( 2.72 - 5.83)</b>	<b>2.05 ( 0.76 - 3.35)</b>
45-64 yrs (ref)	0.00	0.00	0.00	0.00
65+ yrs	<b>-3.21 (-5.06 - -1.36)</b>	<b>-2.18 (-3.83 - -0.53)</b>	<b>-2.99 (-4.84 - -1.14)</b>	<b>-2.09 (-3.74 - -0.44)</b>
<b>NZDep2001 Quintiles</b>				
least deprived			0.00	0.00
Quintile 2			<b>-3.87 (-6.38 - -1.35)</b>	-1.14 (-3.38 - 1.11)
Quintile 3			<b>-5.53 (-7.97 - -3.09)</b>	<b>-2.57 (-4.75 - -0.38)</b>
Quintile 4			<b>-5.31 (-7.69 - -2.92)</b>	<b>-4.22 (-6.35 - -2.09)</b>
Most deprived			<b>-6.03 (-8.65 - -3.41)</b>	<b>-4.33 (-6.71 - -1.94)</b>
<b>Random var.(s.e of variance)</b>				
<b>Neighbourhood-Level</b>	14.41 (4.67)	15.61 (3.09)	14.32 (4.77)	15.56 (3.13)
<b>Individual-Level</b>	360.29 (9.21)	390.16 (7.54)	358.03 (9.16)	389.11 (7.52)
ICC	0.038	0.038	0.038	0.038
<b>*NeighFrag (continuous)</b>			-1.12 (-4.31 - 2.08)	<b>-4.03 (-6.82 - -1.23)</b>

Model 3 adjusting for design variables and individual variables

Model 4 adjusting for design variables and individual variables and NZDep

\*NeighFrag (continuous) estimated change in predicted GH between least and most NeighFrag; estimates obtained from separate regressions, added here for comparison.

Once again, there was no evidence of an association with NeighFrag on the self-reported general health of men. For women there was evidence of a negative monotonic linear relationship, with better reported General Health in NF1 compared with NF4,5 (2.76 (-1.84-7.35)), and worse health in NF10 compared with NF4,5 (-3.86 (-6.13--1.59)). Unlike the Mental Health estimates the monotonicity held over moderate levels of fragmentation. However the categorical NeighFrag estimates for General Health were only statistically significant to the 95% confidence level in deciles 8-10. Once NZDep was added into the model the same monotonic nature of the associations remained, but the magnitude was attenuated. Only the NF10 estimates were statistically significant with the confidence intervals not including 0.00 (-2.93 (-5.24--0.63)). The statistically significant NeighFrag (continuous) estimate was negative, supporting the pattern from the categorical modelling of NeighFrag. The size of the association for the continuous version was also consistent with the categorical estimates.

NZDep was also associated with General Health in the full model for both men and women. Increasing deprivation was associated with increasingly poorer self-reported general health, an effect which was amplified, in this case, for men. When the magnitudes in the association of the two neighbourhood exposures were compared they appear to be roughly similar. However, the lack of precision in the NeighFrag categorical estimates suggests caution in interpreting the results.

#### *6.2.3.2.2 Other Health Scales*

The fully adjusted NeighFrag and NZDep estimates for the remaining seven SF36 scores have been shown in Tables 6:9 and 10 for women and men respectively. As the purpose of presenting these scales is primarily comparative, the estimates (with p values) are reported for the NeighFrag and NZDep variables only, but are from the fully adjusted model. Generally, the pattern of evidence has supported the hypothesized specificity of the association between fragmentation and mental health. It suggests that neighbourhood deprivation plays a greater part in predicting other

aspects of self-reported health status than fragmentation, particularly for men. For women, statistically significant associations for the effect of higher levels of NeighFrag were observed for the Vitality scales. The pattern of the estimates suggested a step function rather than the monotonic relationship seen for the Mental and General Health estimates.

Table 6:9 NeighFrag and Other SF-36 Health Scales: Women

	Predicted change in Health score													
	Physical Functioning		Role Physical		Bodily Pain		Vitality		Social Functioning		Role Emotional		Health Transition	
	Estimate	Pr >  t	Estimate	Pr >  t	Estimate	Pr >  t	Estimate	Pr >  t	Estimate	Pr >  t	Estimate	Pr >  t	Estimate	Pr >  t
<i>Neighbourhood exposures</i>														
<i>Intercept</i>	93.15	0.000	90.34	0.000	87.77	0.000	79.09	0.000	101.25	0.000	104.93	0.000	50.76	0.000
<i>NeighFrag</i>														
<i>NF 1</i>	-0.46	0.842	8.20	0.056	-2.45	0.444	2.28	0.356	1.39	0.574	-1.36	0.695	1.49	0.572
<i>NF 2,3</i>	-0.30	0.799	0.11	0.961	-0.25	0.879	-0.55	0.661	-0.25	0.840	2.36	0.180	0.99	0.461
<i>ref NF 4,5</i>	0.00	.	0.00	.	0.00	.	0.00	.	0.00	.	0.00	.	0.00	.
<i>NF 6,7</i>	-1.18	0.199	-1.14	0.499	-1.84	0.147	-1.93	<b>0.049</b>	-1.86	0.058	-1.51	0.270	0.98	0.346
<i>NF 8,9</i>	-1.10	0.219	0.33	0.843	-1.01	0.416	-1.94	<b>0.043</b>	-0.38	0.695	-0.52	0.700	1.01	0.323
<i>NF10</i>	-0.59	0.613	-0.64	0.766	-0.89	0.579	-2.07	0.094	-1.51	0.223	-0.77	0.657	-0.39	0.767
<i>NZDep</i>														
<i>least deprived</i>	0.00	.	0.00	.	0.00	.	0.00	.	0.00	.	0.00	.	0.00	.
<i>Quintile 2</i>	-1.84	0.103	0.45	0.830	-0.45	0.774	-0.78	0.515	-0.51	0.670	-2.86	<b>0.089</b>	-6.16	0.913
<i>Quintile 3</i>	-2.36	<b>0.031</b>	-3.05	0.133	-0.93	0.538	-2.61	<b>0.026</b>	-0.50	0.671	-2.34	0.152	-5.55	0.948
<i>Quintile 4</i>	-4.29	<b>0.000</b>	-4.46	<b>0.024</b>	-3.18	<b>0.032</b>	-3.18	<b>0.005</b>	-2.19	<b>0.056</b>	-3.38	<b>0.035</b>	-6.52	0.600
<i>Most deprived</i>	-6.60	<b>0.000</b>	-6.08	<b>0.006</b>	-4.69	<b>0.005</b>	-2.22	0.083	-2.63	<b>0.040</b>	-4.40	<b>0.014</b>	-7.91	0.399
<i>*NeighFrag (continuous)</i>	-1.16	0.410	-1.27	0.625	0.02	0.994	-3.01	<b>0.045</b>	-0.81	0.587	-2.84	0.175	-0.59	0.711

Adjusting for design variables and individual variables and NZDep

\*NeighFrag(continuous) estimated change in predicted GH between least and most NeighFrag; estimates obtained from separate regressions, added here for comparison

Table 6:10 NeighFrag and Other SF-36 Health Scales: Men

	Predicted change in Health score													
	Physical Functioning		Role Physical		Bodily Pain		Vitality		Social Functioning		Role Emotional		Health Transition	
Neighbourhood exposures	Estimate	Pr >  t	Estimate	Pr >  t	Estimate	Pr >  t	Estimate	Pr >  t	Estimate	Pr >  t	Estimate	Pr >  t	Estimate	Pr >  t
<i>Intercept</i>	93.72	0.000	90.53	0.000	82.87	0.000	81.76	0.000	95.17	0.000	96.35	0.000	57.47	0.000
<i>NeighFrag</i>														
<i>NF 1</i>	0.89	0.716	1.73	0.713	-1.77	0.629	5.44	0.048	0.93	0.725	1.02	0.783	-3.01	0.311
<i>NF 2,3</i>	0.92	0.452	1.94	0.412	2.34	0.201	1.25	0.364	2.06	0.121	-0.34	0.857	-1.93	0.196
<i>ref NF 4,5</i>	0.00	.	0.00	.	0.00	.	0.00	.	0.00	.	0.00	.	0.00	.
<i>NF 6,7</i>	0.38	0.696	1.24	0.510	1.73	0.236	0.41	0.708	2.34	0.027	0.36	0.811	-3.51	0.003
<i>NF 8,9</i>	-0.14	0.882	2.74	0.137	2.20	0.125	0.79	0.463	1.96	0.059	-0.35	0.808	-2.44	0.036
<i>NF10</i>	1.29	0.280	5.37	0.020	3.69	0.039	0.84	0.533	3.35	0.010	0.48	0.793	-2.57	0.078
<i>NZDep</i>														
<i>least deprived</i>	0.00	.	0.00	.	0.00	.	0.00	.	0.00	.	0.00	.	0.00	.
<i>Quintile 2</i>	-2.26	0.047	-1.82	0.407	-3.68	0.030	-2.00	0.119	-3.14	0.011	-3.49	0.044	1.80	0.195
<i>Quintile 3</i>	-3.42	0.002	-6.49	0.002	-3.49	0.035	-3.75	0.003	-3.19	0.008	-3.38	0.044	0.33	0.806
<i>Quintile 4</i>	-4.91	0.000	-5.24	0.012	-6.04	0.000	-4.02	0.001	-2.46	0.035	-3.28	0.046	1.69	0.198
<i>Most deprived</i>	-7.34	0.000	-9.13	0.000	-5.74	0.001	-2.88	0.032	-5.28	0.000	-4.62	0.010	-1.02	0.479
<i>*NeighFrag (continuous)</i>	-0.22	0.880	4.68	0.093	3.27	0.130	-0.65	0.691	2.90	0.065	-0.09	0.967	-2.13	0.228

Adjusting for design variables and individual variables and NZDep

\*NeighFrag (continuous) estimated change in predicted GH between least and most NeighFrag; estimates obtained from separate regressions, added here for comparison.

Social Functioning and Health Transition scores also demonstrated some sensitivity to NeighFrag for men. Again, while the estimates are statistically significant there was less evidence of a clear pattern of either a dose response or a threshold effect in the associations. The lack of pattern does not necessarily preclude an effect being present but it can also hinder the interpretation of the associations and increase the possibility of the effect being a chance finding.

Once again, the estimates for NeighFrag (continuous) generally support the evidence above and are reported separately at the bottom of the table.

### *Intraclass Correlations (ICC)*

The ICC's were calculated for each main effect model for the Mental and General Health scales to examine the extent to which the variation in self-reported health could be attributed to neighbourhood variation, aside from variation between individuals. Overall, there was little evidence from the ICCs of significant between-neighbourhood variations in how mental health scores are reported. This was the case for the empty model with no exposures, and all the way through to the final models (Tables 6:6 and 7). In fact the ICC stayed remarkably stable across the models. It may have been a result of the nested data being forced into a two level model or from other challenges to the multilevel modelling used here. Or it may simply be a reflection that while important confounders have been included, the ecological variations in how people report mental health can be attributed to the composition of the neighbourhood that haven't been captured in the confounders used here. In other words, the larger, individual level variation remained largely unexplained. Whilst it would have been reassuring to have seen the ICC diminishing with the inclusion of NeighFrag, it is possible to have significant ecological main effects without a large ICC (Blakely and Subramanian, 2006, Merlo, 2003).

### **6.2.3.3 Effect Measure Modification.**

#### *6.2.3.3.1 Cross-level Effect Measure Modification*

Evidence of cross-level effect measure modification was sought from a combination of two methods. Firstly, stratified analyses were run using the NeighFrag (continuous) variable for added parsimony. The association of NeighFrag (continuous) with Mental Health was examined for homogeneity across the strata of sex, labour force status, age groups, and NZDep. If there was any evidence of heterogeneity, statistical confirmation of a difference in the association between strata was then sought using interaction term estimates for NeighFrag (continuous) and levels of the modifying factor.

In order to compare the two methods and thereby gain more evidence for any modification, estimates from pooled interaction regression results were then used to back calculate the estimated magnitude of the NeighFrag/MH association across the strata of the modifying factor, but this time taking into account any potential interaction between the individual level variables and NeighFrag. The main effect estimate for the reference group of each effect modifying variable was equivalent to the main effect NeighFrag (continuous) estimate adjusted, now adjusted for any interaction, and is given in column 2 of Table 6:11. The interaction term estimate for the non-reference levels (seen in column 3) represents the change in the association of NeighFrag between each level of the modifying variable and the relevant reference group. Column 4 shows how these were then added to the reference group estimate to calculate the magnitude of the NeighFrag/MH association for each level of the modifying factor, but now adjusted for any interaction between NeighFrag and that factor. The estimates in columns 1 and 4 should then be roughly comparable if effect modification is evident in the data. The full comparison of the two methods is presented for women. Because none of the stratified results for men suggested any effect modification, only the stratified results have been presented below.



Table 6:11 Effect modification; comparing the results from stratification and statistical interaction:  
Women

<i>Potential effect modifying factors (EM)</i>	<i>column 1 stratified method results</i>	<i>column 2 Pooled results - statistical interaction method</i>	<i>column 3</i>	<i>column 4 back calculation to compare pooled and stratified results in column 1</i>
		<i>reference group - main effect for NF(cont) estimate adjusted for interaction</i>		<i>NF*EM estimate + EM ref</i>
	<i>NF (cont) estimate</i>		<i>NF*EM estimate</i>	
<b>Sex</b>				
<i>female</i>	-3.29 (-5.39 - -1.19)		-2.67 (-5.25 - -0.09)	-0.70 + -2.67 = -3.37
<i>male (EM ref)</i>	-0.67 (-2.86 - 1.51)	-0.70 (-2.81 - 1.41)	-	-0.70
<b>Labour force</b>				
<i>not in labour force</i>	-1.87 (-5.08 - 1.34)		1.22 (-2.32 - 4.76)	-3.19 + 1.22+ = -1.97
<i>unemployed</i>	-22.43 (-35.04 - -9.83)		-16.44 (-25.56 - -7.32)	-3.19 + -16.44 = -19.63
<i>employed (EM ref)</i>	-2.30 (-4.75 - 0.15)	-3.19 (-5.73 - -0.65)	-	-3.19
<b>Age groups</b>				
<i>15-24 years</i>	-4.53 (-10.78 - 1.72)		-1.47 (-7.38 - 4.44)	-4.56 + -1.47 = -6.03
<i>25-44 years</i>	-3.81 (-6.90 - -0.72)		1.88 (-2.28 - 6.04)	-4.56 + 1.88 = -2.67
<i>45-64 years (EM ref)</i>	-4.04 (-7.69 - -0.39)		-	-4.56
<i>65+ years</i>	-0.04 (-4.03 - 3.95)	-4.56 (-7.85 - -1.27)	3.33 (-1.66 - 8.32)	-4.56 + 3.33 = -1.23
<b>NZDep</b>				
<i>least deprived (EM ref)</i>	-3.81 (-7.46 - -0.17)		-	-2.09
<i>Quintile 2</i>	-1.75 (-6.00 - 2.50)		-1.38 (-7.40 - 4.65)	-2.09 + -1.38 = -3.47
<i>Quintile 3</i>	-0.87 (-4.93 - 3.19)		-0.19 (-6.17 - 5.80)	-2.09 + -0.19 = -2.28
<i>Quintile 4</i>	-3.11 (-7.35 - 1.12)		-0.99 (-6.71 - 4.74)	-2.09 + -0.99 = -3.08
<i>Most deprived</i>	-6.27 (-10.91 - -1.62)	-2.09 (-6.30 - 2.12)	-3.36 (-9.15 - 2.43)	-2.09 + -3.36 = -5.45

There is evidence that individual characteristics modified the relationship between NeighFrag and Mental Health. As already reported above in the stratified analyses, sex appeared to modify the association of fragmentation with mental health (column 1, Table 6:11). NeighFrag did not appear to be a factor in predicting men's mental health status (-0.67(-2.86 to 1.51)) whereas a moderate, statistically significant association was observed for women (-3.29(-5.39 to -1.19)). When a sex\*NeighFrag interaction term was specified in pooled analyses (column 3) the estimate for women of -2.57 had a 95% confidence interval excluding 0 (i.e. -5.25 to -0.09) meaning that women had a 2.57 greater drop or gradient than men in MH score from least to most fragmented neighbourhoods. When the -2.57 is added to the adjusted NF main effect estimate ((-0.70) (column 2)), the back calculated estimate of -3.37 in column 4 is very similar in magnitude to the stratified estimate for women of -3.29.

There was strong evidence of effect measure modification of NeighFrag by labour force status for women, illustrated in Figure 6:10. For those not in the labour force, living in the most compared with least fragmented neighbourhoods did not appear to predict their self-reported Mental Health in either the stratified (column 1) or pooled (column 3) analyses. While the estimates suggested a small decrease (less than 2 point drop) in mental health from least to most NeighFrag, the 95% confidence intervals for both stratified and pooled analyses included 0.00, indicating that they were statistically non-significant. The back calculated estimates in column 4 also suggested only a small (if any) approximately 2 point decrease in MH scores for women in this strata living in the most compared to least fragmented neighbourhoods. A larger 2-3 point difference was seen for employed women, but statistical significance was only seen in the pooled analysis (95 % confidence intervals of -5.73 to -0.68), although in the stratified results the upper 95% limit almost excluded 0 (-4.75 to -0.15).

In both stratified and pooled analyses, the difference in Mental Health scores by NeighFrag for unemployed women (a much smaller stratum), was large and

statistically significant. The stratified results in column 1 suggested a 22 point decrease in MH scores for unemployed women living in the most compared to least fragmented neighbourhoods. The pooled analyses in column 3 indicated that adjusting for an interaction attenuated the magnitude of the difference somewhat, but it remained very large (-16.44) and the 95% confidence intervals excluded 0 (-25.56 to -7.32). Again the back calculation did not contradict the magnitude of the NeighFrag/MH association for this stratum seen in the stratified analyses. However, the rather large size and imprecision of the estimate for unemployed women warranted further investigation.

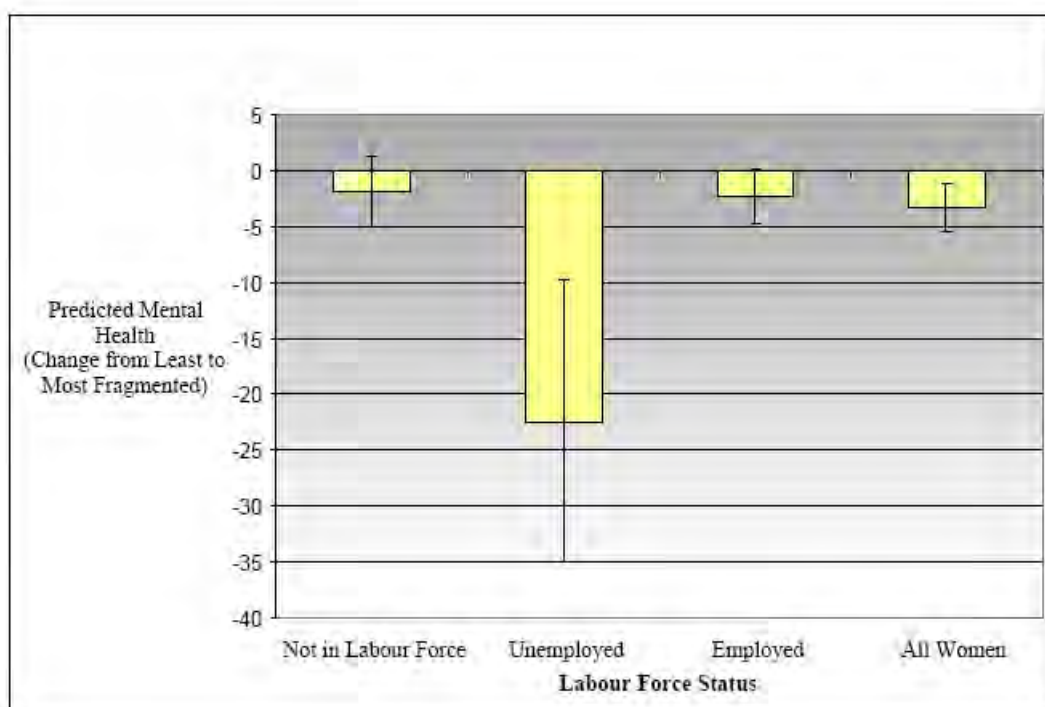


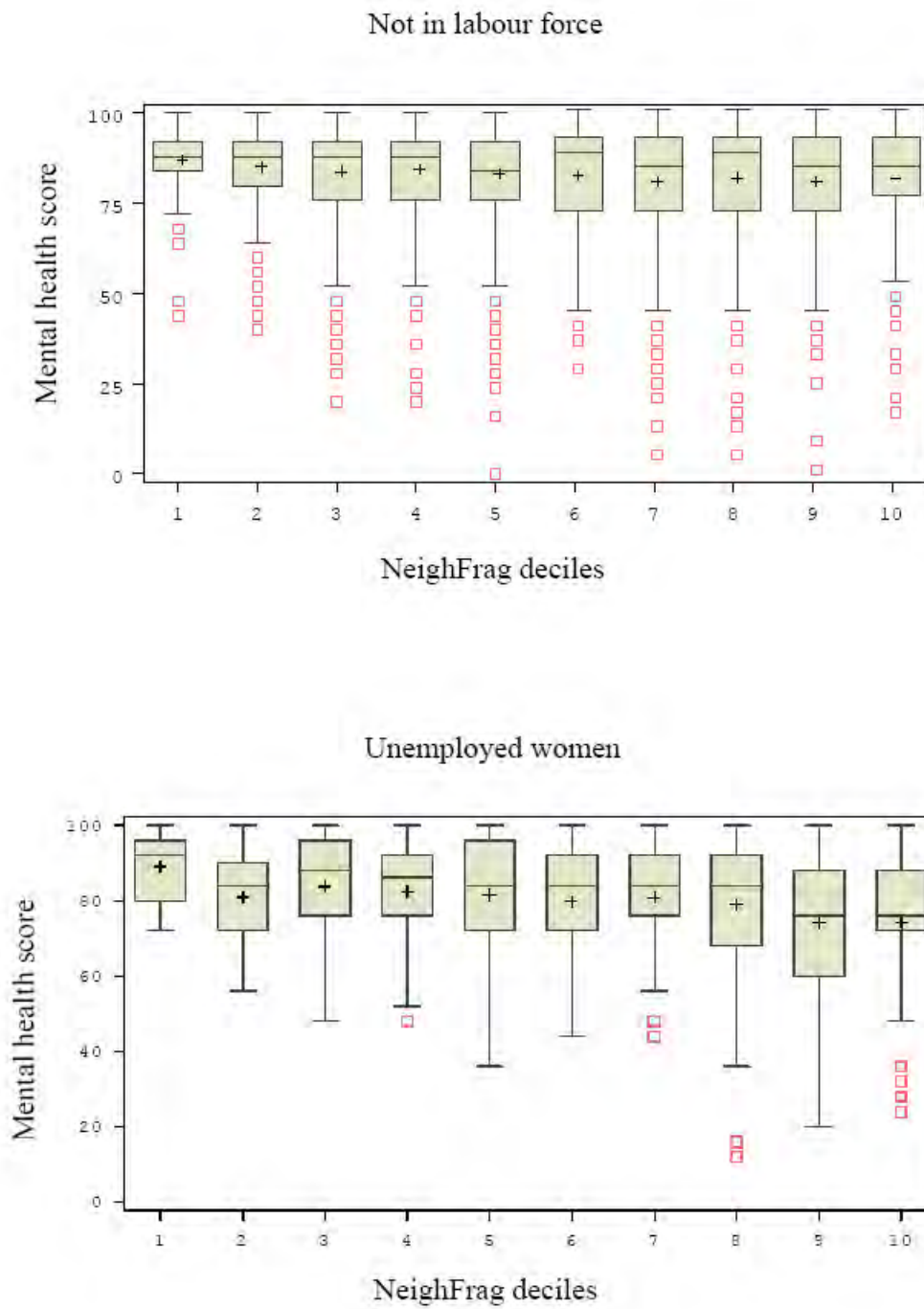
Figure 6:10 NeighFrag (continuous) and SF-36 Mental Health Scale, Stratified by Labour Force Status (Fully Adjusted, 95 % Confidence Intervals)

The unadjusted distribution of the MH scores across NeighFrag categories was examined by the three labour force strata and is illustrated in Figures 6:11 a-c. It suggested that the large size of estimate obtained for the unemployed strata (Figure

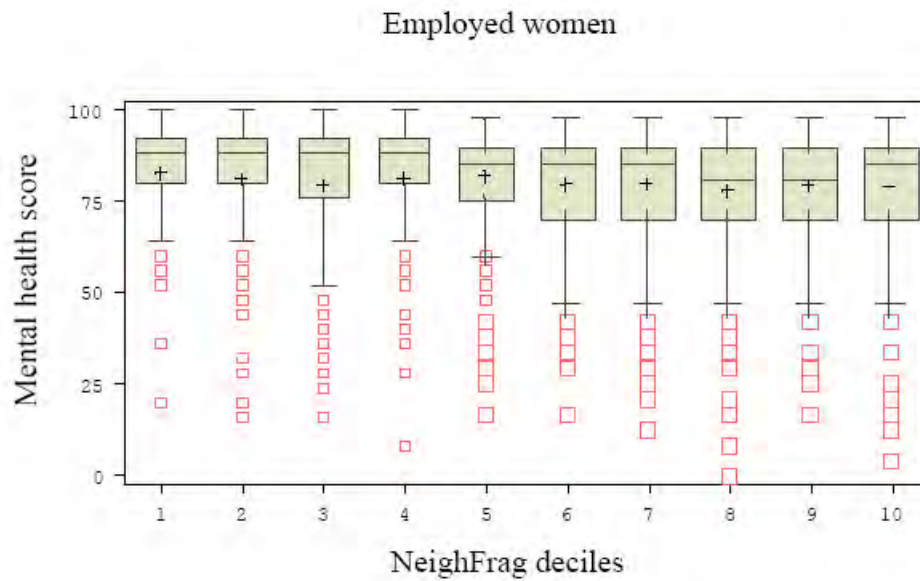
6:11b) may be in part due to leverage from the wider spread of scores at higher levels of NeighFrag, and the narrower range at low levels of NeighFrag, when compared to the other stratum. It was also important to bear in mind that the results in Figures 6:11 would still be confounded, and do not therefore capture the adjusted relationships seen in the stratified and pooled results. While the effect may indeed be due to the outliers, the distribution of MH scores was different across the three strata. So, while the magnitude of the interaction term estimate may have been distorted, assessing the combined evidence from the crude, stratified, and pooled analyses provided some (cautious) support for effect modification by labour force status.

There was inconsistent evidence of a modifying effect of age group on NeighFrag for women. The stratified results in column 1 suggested that there was an approximately 4 point difference in the MH scores for 15-64 year old women in the most compared to least fragmented neighbourhoods, but no difference for 65+ year olds which suggested that there was some heterogeneity across strata. However, the estimates included 0 for both 15-24 year olds (-10.78 to 1.72) and 65+ year olds (-4.03 to 3.95). It is therefore possible that the 'true' estimates for women did not vary significantly across strata. The estimates (and the 95% confidence intervals) for the pooled analyses in column 2 did not support the pattern seen in the stratified analyses. The reference group estimate suggested a slope of 4.56 points in mental health scores for 45-64 year old women across the NeighFrag range. However the 25-44 years\*NeighFrag estimate of 1.88 (which was non-significant) suggested the NeighFrag gradient was approximately half that of the older reference group. The results from the back calculation in column 4 highlighted the inconsistencies between the two methods and point to other potential statistical interactions, for example between covariates, and the exposure and outcome. The width of the confidence intervals also highlight the possibility that the estimates may be due to chance or statistical artefact.

Figure 6:11 Unadjusted NeighFrag Distribution of Mental Health Scores, by Labour Force Status – Women



Boxplots showing 5<sup>th</sup> and 95<sup>th</sup> percentile as horizontal lined, 25<sup>th</sup> and 75<sup>th</sup> percentiles as boxes, and median as horizontal ;lines within boxes. The + is the mean MH score in each level of NeighFrag. Small squares are outliers.



Boxplots showing 5<sup>th</sup> and 95<sup>th</sup> percentile as horizontal lined, 25<sup>th</sup> and 75<sup>th</sup> percentiles as boxes, and median as horizontal ;lines within boxes. The + is the mean MH score in each level of NeighFrag. Small squares are outliers.

#### 6.2.3.3.2 Within-Level Effect Measure Modification

There was some evidence of modification of the NeighFrag/Mental Health association by NZDep. In column 1 the estimates for NeighFrag/MH were the largest (and statistically significant) in the least and most deprived neighbourhoods (-3.81 and -6.27 respectively). The evidence was less clear at more moderate levels. While the magnitude of the NF estimates was smaller in NZDep quintiles 2, 3 and 4, the confidence intervals for the three estimates were wide and all included 0.00. The pattern of the heterogeneity suggested a 'U' shaped relationship, where the association of NeighFrag was strongest at the extremes of NZDep, and weaker at more moderate levels. In column 2 The NZDep\*NeighFrag interaction terms estimates followed a similar non-linear pattern to that observed in the stratified analyses but were attenuated. However, none of the estimates were statistically significant with 0.00 included in all of the 95% confidence intervals. No clear picture emerged from the back calculation in column 4. It may well be that the effect of

fragmentation on mental health was modified by deprivation but no conclusion could be made from the data available here.

In keeping with the non-stratified analyses there was little clear evidence of effect measure modification for men (Table 6:12). Therefore only the stratified results have been presented here.

The absence of an association between NeighFrag and the self-reported mental health of men and was remarkably persistent. Notably, given the large estimate observed for unemployed women, there was no suggestion that labour force status increased the chance of poorer mental health in men.

## **6.2.4 Secondary Questions**

### ***6.2.4.1 NZDep/NeighFrag Relationship***

As reported above, the statistical evidence suggests that NZDep acts as either a confounder or an intermediary in the regression model. There was an approximately 18% reduction in the NeighFrag estimate when NZDep was added into the model. There is only limited evidence of any interaction between the two variables. The correlation between the neighbourhood measures did not appear to violate the statistical assumptions of the regressions.

### ***6.2.4.2 Congdon(NZ) Analyses.***

It did not appear that Congdon(NZ) was a significant predictor of self-reported Mental or General Health status of men or women in the sample population (Table 6:13) (for brevity only the Congdon(NZ) and NZDep estimates are provided here). There were no significant estimates observed at any level of fragmentation, relative to the moderate reference category.

Table 6:12 Modification of NeighFrag with a range of covariates in the association of Mental Health: Stratified Results for Men

<i>Potential effect modifying factors (EM)</i>	<i>Stratified Method NF (cont) estimate</i>
<b><i>Labour Force Status</i></b>	
<i>not in labour force</i>	-0.15 ( -4.32 - 4.02 )
<i>unemployed</i>	-0.27 ( -15.23 - 14.68 )
<i>employed</i>	0.15 ( -2.25 - 2.54 )
<b><i>Age Groups</i></b>	
<i>15-24 years</i>	-2.84 ( -9.36 - 3.68 )
<i>25-44 years</i>	-1.43 ( -4.96 - 2.11 )
<i>45-64 years</i>	0.36 ( -3.26 - 3.98 )
<i>65+ years</i>	1.73 ( -2.59 - 6.04 )
<b><i>NZDep</i></b>	
<i>least deprived</i>	1.67 ( -1.99 - 5.34 )
<i>Quintile 2</i>	-2.72 ( -8.11 - 2.67 )
<i>Quintile 3</i>	-0.64 ( -5.48 - 4.21 )
<i>Quintile 4</i>	-5.28 ( -10.07 - -0.50 )
<i>Most deprived</i>	1.59 ( -4.17 - 7.35 )
<b><i>Sex</i></b>	
<i>female</i>	-3.29 ( -5.39 - -1.19 )
<i>male</i>	-0.67 ( -6.01 - 1.51 )



Table 6:13 Neighbourhood Fragmentation (Congdon) and SF36 Mental General Health multilevel analyses (fully adjusted, 95% confidence intervals)

<i>Neighbourhood exposures</i>	<i>Predicted Change in Score</i>			
	<i>Mental Health</i>		<i>General Health</i>	
	<i>male</i>	<i>female</i>	<i>male</i>	<i>female</i>
<i>Intercept</i>	94.34 (86.46 - 102.23)	94.90 (86.96 - 102.85)	77.99 (66.47 - 89.51)	82.77 (72.20 - 93.34)
<b>Congdon(NZ)</b>				
<i>NF 1</i>	0.25 (-2.46 - 2.96)	0.54 (-2.10 - 3.18)	1.48 (-2.48 - 5.44)	-0.03 (-3.54 - 3.49)
<i>NF 2,3</i>	0.30 (-1.58 - 2.18)	-0.49 (-2.27 - 1.28)	0.55 (-2.20 - 3.29)	-0.66 (-3.02 - 1.70)
<i>NF 4,5 (ref)</i>	0.00	0.00	0.00	0.00..
<i>NF 6,7</i>	1.32 (-0.21 - 2.85)	0.10 (-1.34 - 1.55)	2.16 (-0.08 - 4.39)	0.27 (-1.65 - 2.19)
<i>NF 8,9</i>	1.18 (-0.32 - 2.67)	-0.48 (-1.88 - 0.93)	0.96 (-1.22 - 3.14)	-0.53 (-2.40 - 1.34)
<i>NF10</i>	0.09 (-1.77 - 1.95)	-0.90 (-2.68 - 0.88)	0.88 (-1.83 - 3.60)	-2.12 (-4.49 - 0.25)
<b>NZDep2001 Quintiles</b>				
<i>least deprived</i>	0.00	0.00	0.00	0.00..
<i>Quintile 2</i>	-1.28 (-3.04 - 0.48)	-1.28 (-3.00 - 0.43)	-3.96 (-6.53 - -1.40)	-1.38 (-3.67 - 0.90)
<i>Quintile 3</i>	-1.50 (-3.25 - 0.24)	-1.26 (-2.97 - 0.45)	-5.78 (-8.33 - -3.23)	-3.06 (-5.34 - -0.79)
<i>Quintile 4</i>	-0.84 (-2.58 - 0.91)	-1.71 (-3.42 - -0.01)	-5.74 (-8.29 - -3.20)	-4.76 (-7.03 - -2.49)
<i>Most deprived</i>	-2.52 (-4.40 - -0.64)	-2.63 (-4.50 - -0.77)	-6.29 (-9.03 - -3.54)	-5.07 (-7.55 - -2.59)
<i>Random variance (s.e of variance)</i>				
<i>Neighbourhood-level</i>	6.71 (2.19)	8.80 (1.92)	14.32 (4.80)	15.58 (3.12)
<i>Individual-level</i>	167.69 (4.27)	220.01 (4.25)	357.92 (9.16)	389.41 (7.53)
<i>ICC</i>	0.038	0.038	0.038	0.038

## 6.3 Discussion

### 6.3.1 Results Summary

NeighFrag was adversely associated with self-reported mental health in urban women, but not men, providing empirical support for a cross-level main effect (the first research question). Increasing levels of fragmentation predicted increasingly poorer mental health, with some evidence of a dose response. The association was attenuated after simultaneously controlling for important individual and neighbourhood risk factors, but remained larger than some individual factors, and was statistically significant. The direction of the association supported the hypothesis that low levels of integration and regulation in the neighbourhood social group would be associated with poorer health.

There was evidence that the association between NeighFrag and health was specific to the mental health domain. There was some evidence of a relationship between NeighFrag and General Health after controlling for individual factors. The addition of NZDep into the model subsequently reduced the association and a statistically significant effect remained only for NF10. There was less consistent evidence of an association of NeighFrag with other SF36 measures, providing some support for a specificity of effect of NeighFrag on health.

The second research question asked if individual factors modified the association. There was some cautious empirical support for cross-level effect modification of NeighFrag by some individual factors. The magnitude of the association of NeighFrag and mental health was not homogeneous across the strata of sex and labour force status for women only, which was supported by statistical tests of interaction. When women were stratified by labour force (an alternative social group membership proxy), the estimates suggested a small adverse association of NeighFrag with mental health for both the employed and 'not in labour force' strata, and a very large adverse association for unemployed women. The results from the

statistical interaction test confirmed the strength and direction of the modification, and subsequent checks on crude data did not contradict the findings.

Evidence of effect modification of NeighFrag by age among women was less consistent. There was some suggestion that variation in 65+ year old women's mental health was not predicted by NeighFrag, whereas a gradient from most to least fragmented neighbourhoods was present for the other age groups. The pooled analyses suggested a less consistent pattern of heterogeneity from that observed in the stratified analyses. Interpreting the estimates was also problematic because of wide confidence intervals. It is therefore possible that the heterogeneity was due to chance rather than effect modification.

There was no evidence of effect modification in the association between NeighFrag and mental health for men. This included the alternative social group measure proxy of labour force status, suggesting that a fragmented neighbourhood social group was not detrimental to men's mental health.

The third and fourth research questions addressed secondary issues. The association of NeighFrag and mental health appeared to be independent of neighborhood deprivation, an important finding. There did not appear to be statistical difficulties including both measures in a regression, although precision was compromised (further details below). However the interaction between NeighFrag and NZDep for women was problematic to interpret. The pattern suggested that there was a stronger association of NeighFrag with mental health in the most and least deprived neighbourhoods. However the results from the stratified and pooled analyses provided no consistent statistical evidence of effect modification.

In respect to the final research question, the Congdon(NZ) demonstrated a null association with Mental and General Health. It could be argued that the discrepancy between the measures could be evidence of chance in the reported findings for NeighFrag. Alternatively, the differences between the measures may explain the

variance in the results. These explanations will be discussed more fully in Chapter Eight.

### **6.3.2 Sources of Error**

Assessing the robustness of the associations reported was an important aspect when interpreting the evidence. The internal validity of the measurement has been discussed here: random error, or precision and systematic error. The wider issue of generalisability, that is, did the epidemiological evidence support the theory, has been discussed in Chapter Eight.

#### **6.3.2.1 Precision**

All of the estimates obtained have been reported with 95% confidence intervals. That is to say, there was a 95% chance that the 'true' value laid within the intervals. It was of course still possible that the 'true' value could have been outside of the intervals. Nevertheless, the confidence intervals have provided an indication that the estimates obtained were unlikely to have been purely due to chance.

An alternative would have been to set more rigorous criteria of, for example, 99%. Under a more stringent criteria, the chance of the "true" association falling outside of the confidence intervals would have been reduced, thereby decreasing the chance of a Type 2 error (that of failing to recognise a 'true' association). However, it also would have increased the chance of a Type 1 error if 'true' values outside of the tighter confidence intervals were dismissed as being non-significant.

Precision of the estimates can be affected by study size and comparison groups. While the original datasets were large the restriction and stratification processes resulted in relatively small comparison groups. This was particularly a problem for NF1 as the numbers living in this decile were in this group were considerably smaller than more fragmented neighbourhoods, notably for men. A consequence of this is that the estimates in the lower deciles are more difficult to interpret because of the

lack of precision. Because of this it has been useful to interpret the results visually with graphs as well as in tables in order to observe patterns of association. Nevertheless the sample sizes well exceeded those needed to detect a 2 point difference between a population norm and a group mean (Ware et al., 2000). Furthermore, comparing the pattern and magnitude of the associations between the categorical and continuous modelling of NeighFrag suggested that the NF1 estimates were not unreasonable.

Another threat to the precision of the estimates came from the possible collinearity between NeighFrag and NZDep. As reported above, small counts were observed in moderate levels of deprivation across lower levels of NeighFrag. This had implications for sparse data and imprecise standard errors. While NeighFrag and NZDep were correlated, the correlation did not appear to be so strong as to threaten the regression modelling assumptions, an important finding of these analyses. The use of NeighFrag (continuous) provided a useful sensitivity analysis: the estimates obtained for NZDep (and other variables) in both models were similar, indicating that the regression models were not overly sensitive to the sparse data in some cells and collinearity between the neighbourhood exposures.

#### **6.3.2.2 Systematic Error**

Systematic error arising from study design errors in the measurement of an association can occur in three key ways and can decrease the internal validity of an association (Rothman and Greenland, 1998). Associations can be biased because of how participants are selected into a study (selection bias); how information is used to classify participants by exposure, outcomes and covariates (information bias); and unmeasured or mismeasured confounding. The potential of each of these sources of distorting effects will be addressed in the above order.

How participants were selected into the NZHS study could have introduced selection bias if the relationship between the exposure and outcome was different between participants and non-participants. The NZHS was designed to be

representative of the New Zealand population, reducing the chance of difference between the two populations. In particular, weightings were designed to account for non-representative sampling and different response rates, decreasing the risk of bias. While there were differences in the distribution of the NZHS population across NeighFrag compared with the national distribution it appears that the weightings (and therefore the design variables) should have countered this difference.

Further opportunity for selection bias could have occurred when the dataset was restricted to those with complete data on the analysis variables, living in urban settings. It could be that the NeighFrag - mental health relationship was different in the rural population than urban dwellers. Thus the results can only be interpreted for New Zealand urban dwellers, rather than the whole NZ population. It should be noted of course that any different relationship between NeighFrag and health by rurality would be a substantive research question, rather than a source of bias.

Of more concern were the omitted participants with incomplete data. It may be possible that participants with incomplete data had poorer mental health. If they were more likely to live in highly fragmented areas and were not included in the analyses the effect of NeighFrag would have been underestimated. A second factor in determining the likelihood of selection bias was the relationship between incomplete data and NeighFrag: higher levels of incomplete data were observed in the most fragmented neighbourhoods. Again, in order for bias to have occurred, those with missing data would need to have demonstrated a null or positive association between NeighFrag and their mental health.

In order to determine the impact of selection bias two considerations needed to be noted. Firstly, while it was probable that those with the poorest health would be less likely to participate fully (Lundberg et al., 2005) there is little to suggest that the mental health status of the omitted participants would have an opposite relationship with NeighFrag to that of the 'complete data' population (Haaepa et al., 2007). Secondly, there was less than a 2% overall reduction in the dataset when incomplete

observations were omitted, suggesting that the effect of omission would be minimal. Therefore, given the relatively small number of incomplete observations it would be unlikely that the estimates obtained would have been significantly affected and that any bias would be towards the null.

Information bias can result from participants being wrongly classified by study variables. Misclassification of the exposure, outcome or covariates can occur randomly across the variables (that is non-differentially), with the likely effect of reducing the effect towards the null. Harder to interpret is differential misclassification, where the error is systematic. An example of differential outcome/exposure bias would be where the likelihood of being misclassified as having poorer mental health was different by the level of NeighFrag exposure.

Participants' exposure could be misclassified if their recorded area of residence was not their actual residential neighbourhood. It may be that participants who were relatively mobile recorded their address as a more permanent family home that was different from their current exposure. Alternatively, a participant may have been included in the survey if they were present at the time of interview and incorrectly classified as a usual resident there. If participants were equally likely to be wrongly classified (non-differential misclassification) the mixing would probably result in a bias towards the null. In order for differential bias of the association to have occurred the misclassification of neighbourhood of residence by NeighFrag would need to have been systematically related to mental health status.

The NZHS employed an established sampling process that restricted participants to those usually resident in the selected dwelling. There was no reason to suggest that substantial errors in the recording of address were made, particularly systematically by health status or levels of NeighFrag.

Information bias can also occur with the outcome variable, where participants self-reported health could be systematically differently recorded by the exposure. The

SF36 is a measurement tool that has demonstrated validity and reliability for the New Zealand population so it is unlikely that the participant error would vary by socioeconomic and demographic factors. Given this there was no reason to suggest that there would have been a difference in the validity of the tool across levels of NeighFrag that could lead to differential misclassification.

One important source of differential misclassification was avoided in this study. Often neighbourhood studies are reliant on measuring the exposure and the outcome in the same population (for example where individual perception of the neighbourhood is aggregated to make a neighbourhood measure). If participant's perception of the neighbourhood was different *by their health status* then neighbourhoods could be misclassified and the associations likely to be biased. Because this study was able to use an objective neighbourhood measure created from a separate data collection this source of bias was able to be avoided (note however, that the population itself was not different as NZHS was a subsample of the census population).

While there was a time lag between the 2001 census and NZHS data collections it was small (one to two years). It was therefore unlikely that the neighbourhoods changed substantially between the two data collections, and there was no reason to suggest that any changes would be related to the likelihood of outcome classifications.

Mismeasurement of the confounders can result in information bias (Fewell et al., 2007). The NZHS used validated measures of the covariates included in the analyses and it can therefore be reasonably assumed that error has been minimised – but not removed altogether. Measurement of confounders could also have introduced selection bias if there were systematic differences of incomplete covariate data, leading to non-participation in the analyses. As noted above, the overall percentage of missing data was very small and so any difference in mismeasurement by NeighFrag would have a minimal biasing effect on the estimates obtained.



### *6.3.2.3 Confounding*

As well as mismeasurement of the confounders that were included, error can occur if other confounding factors are not included. Substantial unmeasured confounding can occur when important, primary factors are not included in the analyses. This can be because they are not able to be adequately measured with the available dataset, a common occurrence when secondary datasets are used. It could also be the case that the process of establishing what factors met the confounding properties was flawed. However, the systematic and transparent process employed to select the confounders was used because NeighFrag and neighbourhood-level social fragmentation is a relatively novel exposure. Care needed to be taken to avoid relying on confounders typically included in, for example, analyses examining deprivation and mental health, which could have quite a different, non-confounding relationship with fragmentation and health.

These analyses were adjusted for notable socio-demographic factors at the individual and neighbourhood level that were established as confounders to the hypothesised relationship. It is possible however, that other factors were omitted, leading to unmeasured confounding. A more detailed discussion on unmeasured confounding by non-socioeconomic factors that was not able to be measured in the HS dataset is undertaken in Chapter Eight.

It may be that other primary factors, such as population density, explain all or most of the differences in reported mental health scores. If so, including them in the model would have therefore substantially reduced the NeighFrag estimates. The important factor though, is in deciding how much of the association might be spurious, knowing that it was not possible in this type of study design to fully control for all confounding factors. The intention here was to control for the known major factors where possible, and observe changes in the results over the staged models, The progressive addition of variables into the model allowed such observations to be made.

A risk of including covariate exposures as confounders is that they may in fact either be intermediary variables, or highly correlated with them, or act as both a confounder and intermediary (Kirkwood et al., 2003). The effect of adding them into the model is that part of the “true” association is then explained away. As discussed earlier, the nature of the relationship between NZDep and neighbourhood fragmentation was uncertain, in particular the direction of the association between NeighFrag and deprivation. In theory NeighFrag could in some way contribute to the degree of deprivation or material composition of the neighbourhood. It may be that historical patterns of mobility or the level of family type housing in an area may have an effect on the way in which SES factors are then clustered in a neighbourhood. In this instance NZDep could be regarded as an intermediary variable and its inclusion would result in over adjustment of the NF estimate.

On the other hand, the causal pathway could be reversed: the deprivation level in an area may lead to patterns of mobility and housing patterns. In this instance, deprivation would be acting as a confounder and should therefore be included to gain a “truer” estimate. In reality, there may be no clear causal pathway between the two neighbourhood characteristics.

Unfortunately there is no established, reliable way of distinguishing between a mediating or confounding effect from the cross-sectional regressions employed here. For this reason the decision was made to observe the NeighFrag – health estimates both with and without NZDep. Setting aside the role of NZDep, this step also provided information on the contribution of NZDep to the NeighFrag-health relationship and therefore allows for separate examination of the two neighbourhood factors, in keeping with the research goals of the previous social fragmentation literature. The fully adjusted results presented here may therefore represent an overly cautious estimation of the NeighFrag/mental health association.

Consideration was also given to other possible variables that were not included because they did not meet the three properties of a confounder. For example,

household tenure was not included as it was argued that NeighFrag could affect the probability of the type of tenure, introducing the possibility that tenure could be a mediating variable and should therefore not be controlled for. But it could also be argued that a high proportion of renters in a neighbourhood would influence the level of fragmentation, in which case tenure is a confounder and should properly be included in order to understand the actual relationship between NeighFrag and health. The direction of the association between NeighFrag and household tenure is unable to be established in this data set. A sensitivity analysis could have been undertaken to examine the effect of NeighFrag over and above the contribution of tenure. But because there was no means of determining whether any change in the final estimate was due to confounding or mediation, a conservative decision was taken not to do so.

All of these considerations need to be weighed in the interpretation of the associations. As discussed in Chapter Five, a judicious approach to the selection of confounders was taken. The primary aim for including confounders was to increase the internal validity of the associations. By selecting only those factors that met the properties of a confounder, and which could be well measured within the limits of the dataset, the risks associated with over adjustment and mismeasurement were reduced, and the interpretation more transparent.

## **6.4 Conclusion**

Self-reported mental health in urban areas was associated with the Index of Neighbourhood Social Fragmentation. An adverse pattern was observed, with increasing fragmentation predicting poorer mental health scores among women, and possibly more so among unemployed women. The association remained after adjusting for likely individual level socioeconomic confounders. The statistically significant NeighFrag/mental health association also remained after the inclusion of NZDep. It is unclear whether the final model is an overly conservative estimate as it is possible that NZDep may be a mediating factor. It is unlikely that the findings were due to random error. There was also little to suggest that the association was

largely due to systematic error in the mismeasurement of the outcome, exposure or confounding. However it is possible that other factors have confounded the association, but were not able to be measured from the Health Survey dataset.

The substantive interpretation of the associations observed here is undertaken in Chapter Eight. This part of the discussion focuses on the contribution of the analyses to the research questions, asking if the data provides empirical support for the proposed theory. Alternative explanations are considered, particularly the role of unmeasured confounding.



# Chapter 7

## Measuring the NeighFrag - Smoking Relationship

The 1996 New Zealand national census asked all adult participants about their smoking habits (Statistics New Zealand, 2007a). Appending neighbourhood characteristics to the national census data provided a valuable opportunity to examine the relationship between neighbourhood fragmentation and individual health practices. There was no clear evidence in the literature of a relationship between neighbourhood-level social fragmentation and smoking. Indeed there was a reasonable risk of being seen as data-dredging when taking advantage of a large dataset such as this, especially the limited a priori understanding of the pathways. There was, however, support in the literature and in the theoretical framework developed in Chapter Four for regarding the social context as an important factor for understanding individual health practices.

Therefore the focus was on exploring the various pathways in which neighbourhood fragmentation might operate on smoking practices. The analyses presented in this chapter have investigated two potential mechanisms that relate types of neighbourhoods to individual smoking practices: firstly asking if varying levels of integration and regulation meant that fragmented neighbourhoods were more impoverished and therefore generally more stressful places for residents. Here NeighFrag might be a proxy for a 'social deficit' of the neighbourhood in a similar fashion to measures of neighbourhood deprivation. Secondly, the analyses investigate whether the type of neighbourhood (as measured by NeighFrag) affected the 'transmission' of smoking practices between residents within the neighbourhood.

The smoking outcome was used to test both mechanisms. While the analyses use the same dataset and outcome they have been presented in separate sections to aid clarity. The transmission analyses required that the dataset be split into the 'study' population (15-24 years) and the remainder acting as the 'exposure' population (25+

years) (further details below). It was decided to restrict the analyses for the first mechanism to the same study population for the sake of simplicity and continuity. The first section reports the analyses that were designed to test a variation of the 'integration and regulation' mechanism, asking if NeighFrag might act as a neighbourhood 'social deficit' in a similar way that exposure to neighbourhood deprivation can act as a contextual stressor. The results offered contradictory support. They revealed variations by age group in the pattern of association between NeighFrag and smoking. An adverse association was observed for 15-19 year olds, but an opposite, protective association was observed for 20-24 year olds. The magnitude of the association varied by sex and age group and the precision of the individual estimates was at times poor.

The second section of the chapter examines the 'transmission' mechanism. The question here is whether NeighFrag modified the individual smoking risk of youth exposed to neighbourhood level smoking practices. In order to assess the modifying effect of NeighFrag, another neighbourhood level exposure needed to be created (but specifically one that would theoretically be expected to be modified by NeighFrag). The area unit smoking prevalence of 25+ year olds was calculated and neighbourhoods then ranked into deciles to create the Neighbourhood Adult Smoking measure (NAS). The modification of smoking transmission practices could then be observed using within-level effect modification analyses of NAS by NeighFrag. According to the hypothesised mechanism, it was expected that the transmission of smoking practices would be lower in the more fragmented neighbourhoods. The results contradicted the expected relationships. Stratified and pooled analyses revealed a stronger association between NAS and youth individual smoking risk in the most fragmented neighbourhoods. The strength, precision and consistency of the statistical results suggested that the effect modification was unlikely to be due to chance.

The chapter concludes with a brief discussion focusing on the role of chance systematic error with respect to the associations. The extent to which the associations

provide evidence for and against the proposed theoretical mechanisms (the external validity) is discussed in more detail in Chapter Eight. While it is most likely that other factors may be either confounding or mediating the associations, the primary value of the exploratory analyses in this section can be seen when the empirical data is interpreted in light of the proposed theoretical mechanism.

## **Section 1: Integration/Regulation - NeighFrag as a Social Deficit**

The first research question used a cross-level main effect analysis to examine the association of NeighFrag on the individual risk of smoking. The cross-level analyses of the NeighFrag/smoking relationship are the most 'risk factor' or atheroretical of the analyses undertaken in this thesis. Nevertheless it presented me with an opportunity to test of the degree to which NeighFrag may represent a deficit in some way, where living in areas with high levels of fragmentation was generally stressful for residents, and that the neighbourhood can be regarded as socially impoverished. If this were the case it would be expected to observe a similar relationship to that seen for socioeconomic deprivation, where the relationship between smoking and neighbourhood deprivation has often been explained as a response to the stresses of living in an impoverished environment (Barnett et al., 2004, Moon and Barnett, 2003, Stead et al., 2001). If NeighFrag did represent a social deficit in this sense then an increase in fragmentation would be associated with an increased risk of smoking.

The research questions were -

- 1) *Was there a cross-level main effect of NeighFrag on smoking, controlling for individual and neighbourhood covariates?*
- 2) *What was the strength and precision of the association of NeighFrag and smoking?*
- 3) *Did the direction of the association support the 'social deficit' hypothesis: that is, was increasing fragmentation a proxy for a socially impoverished neighbourhood and therefore associated with an increased risk of smoking?*



## 7.1 Methods

### 7.1.1 Selection of Target Population and Dataset Creation

While there are a growing number of smoking studies on adolescents and a large body of work on adult smoking, there is little research on the ages in between. It is known that smoking patterns are often established early in life (Nelson et al., 2008, Graham et al., 2006). Unfortunately national census data on smoking was not available for the younger population and would have been of dubious reliability as the questions asked of children were collated by an adult in the household. Nevertheless, the transitional nature of the selected age group was interesting for a number of reasons.

The 15-24 year old age group is considered to be critical in establishing future smoking behaviours (Graham et al., 2006). It is also more likely that their current neighbourhood is where their current smoking behaviours were established. The age groups also represent key developmental stages as the individual moves from the influence of the family environment to the wider world. By comparison, smokers from an older age group are more likely to have been exposed to a wider range of residential environments over their lifecourse and therefore less can be interpreted about the effect of their current residential environment on their current smoking practices.

The 1996 national census dataset has a total of 534,390 15-24 year olds. The eligible population was first examined to see who lives where by NeighFrag deciles and to determine the extent to which information was available on outcomes and exposures across the deciles. This information provided a basis for decision making about the restriction process, what potential confounders were available, and reducing potential selection bias.

The analysis was restricted to the urban population. Firstly, as noted in Chapter Three, there was concern that CAU's may not capture 'neighbourhoods' to the same

degree as more urban settings. Secondly, previous trials (not shown) on the whole population suggested that the effect of fragmentation on smoking maybe modified by urbanicity. However because there was insufficient power in the whole population dataset to examine the effect separately for the rural population, let alone in the smaller youth dataset, it was decided to restrict the analysis to the urban population. A consequence of this was the inability to generalise beyond the non-urban population.

The restriction process has been illustrated in Figure 7:1. The total study population was firstly restricted to urban 15-24 year olds living in their usual residence who were potentially 'exposed' to their neighbourhood. Usually resident youth in hospitals and prisons were not included as they were not considered to be ordinarily exposed to the neighbourhood. All others usually resident in non-private dwellings were retained, including those living in, for example, student hostels. As well as seeking to be as inclusive as possible, a failure to include the population in non-private dwellings could have potentially lead to biased estimates if the relationship between smoking and fragmentation were different for those living in non-private dwellings from the rest of the youth population.

The next stage in the restriction process was to include only those with complete data on the outcome and covariates for the regression analyses. The analyses used complete-data methods, where observations with missing data were excluded from the regressions. One option would have been to better utilize observations with missing data on confounding exposures with imputed values or weights (Rothman and Greenland, 1998). These options were limited due to Statistics New Zealand data access protocols. Fortunately, the overall percentage of missing values was relatively small (approximately 7%). Because of the logistical difficulties and compromises of the imputation method it was decided to restrict the analyses to observations with complete data and interpret accordingly.

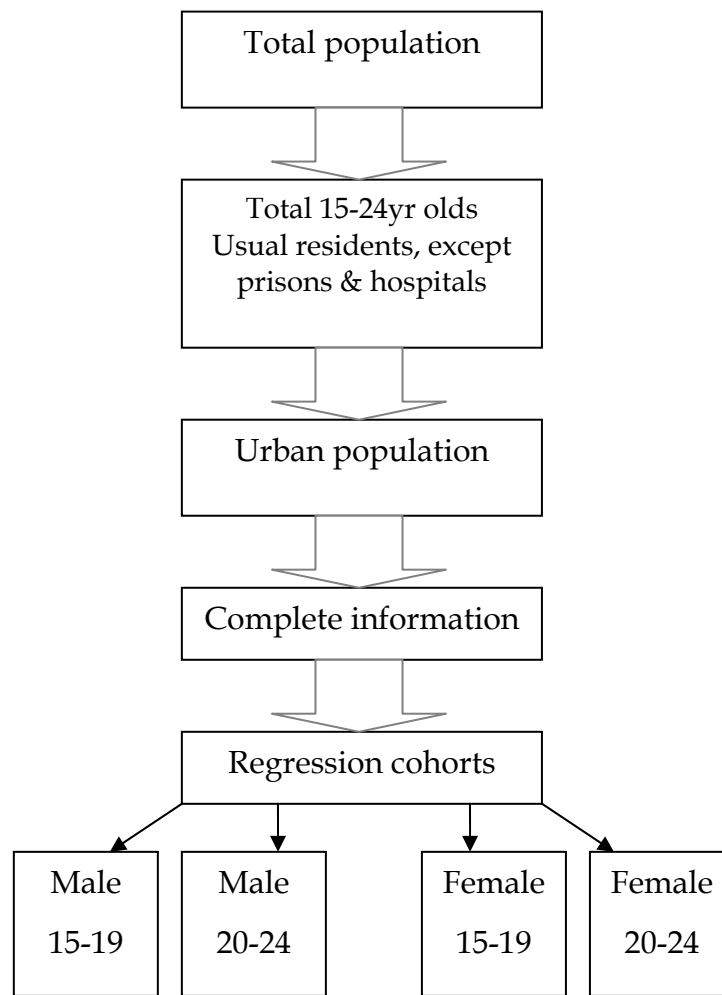


Figure 7:1 Smoking Dataset Restriction Process

The inclusion of non-private dwellings had implications for measurement of confounding factors. For example, it meant that controlling for household income, an important risk factor in the general population for smoking, would be problematic. This is because income on the census dataset was calculated at household levels and was therefore calculated only for private households. Using household level variables might have introduced selection bias, as those living in more fragmented areas would have been less likely to be included in the study because of their increased likelihood of living in non-private dwellings. Further, those with full data

who do live in such areas may have quite different drivers for their smoking risk than those without data, biasing any association observed between neighbourhood fragmentation and individual smoking. Consideration of the potential for bias was therefore an important factor when selecting confounders.

### **7.1.2 Outcome**

The 1996 census included the following yes / no questions on smoking

“Do you currently smoke cigarettes regularly (that is, one or more per day)

- *count only tobacco cigarettes.*
- *don't count pipes, cigars or cigarillos”*

“Have you ever been a regular smoker of one or more cigarettes per day?”

The responses were dichotomized into regular smokers and all others were classed as non-smokers.

### **7.1.3 Exposures**

NeighFrag was specified as a categorical variable with the reference group set as deciles NF4, 5 (described further in Chapter Five).

#### ***7.1.3.1 Selection of the Covariate Exposures***

Many factors are known to predict smoking. In this analysis covariates were included as confounders only if they could be demonstrated as potential confounders of the NeighFrag/smoking association. Covariates were selected using the confounding triangle process described in the methodology section. The strongest individual risk factors signalled in the literature and available in the dataset were first selected to test property 1. The relationships between the potential confounders and NeighFrag have been established in Chapter Three and are described below. The confounding triangle process will be used to discuss the rationale for each factor and explore whether the third property has been met using a priori understanding and logic.

No doubt reflecting the life course stage of the age group there was limited information in the literature of socioeconomic risk factors for smoking. The life course stage of the cohort presented challenges in using the standard measures of adult risk factors from the census data. For example, having no qualifications at 15 years would have different meaning from having no qualifications at 24 years. The confounding variables were important risk factors for smoking, but had varying meaning by age and for the risk of smoking. Therefore the population was stratified into five year age groups and by sex.

Ethnicity has been demonstrated to predict smoking in this age group in New Zealand studies (Kypri and Baxter, 2004) and in the general population (Borman et al., 1999), fulfilling the first property. An association has also been observed between NeighFrag and ethnicity (Table 3:5), fulfilling the second property. It is possible that youth with multiple ethnic identities in less fragmented neighbourhoods with (for example) more Pacific residents may be more likely to prioritise their Pacific identity as a means of identifying with fellow residents or because of local norms. In balance though it was felt that other factors would be more important and that any causal effect of fragmentation on self identification of ethnicity would be relatively small. It is less likely that neighbourhood fragmentation would have an effect on the process of identifying one's ethnicity in the census, fulfilling the third property. As all the criteria for all properties have been met ethnicity was included as a potential confounder (Figure 7:2).

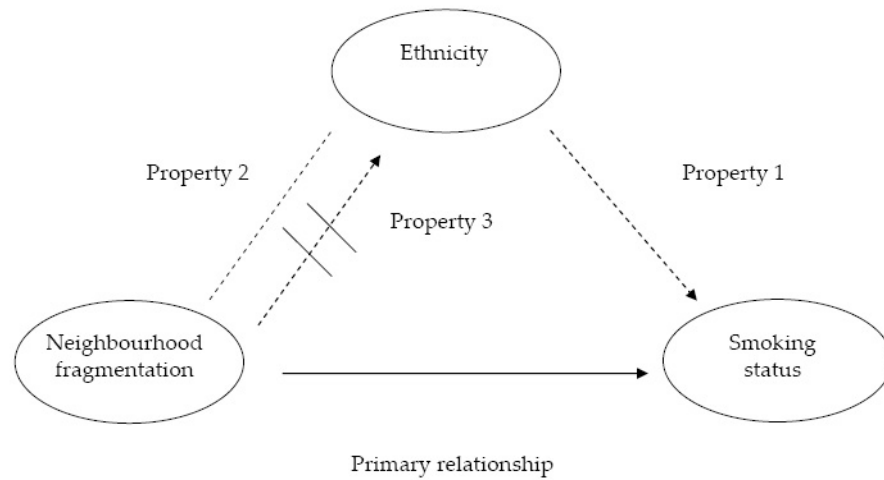


Figure 7:2 Ethnicity Confounding Triangle

Education met the properties of a confounder in this population (Figure 7:3). It is a recognized predictor of smoking in the New Zealand 1996 census population (Borman et al., 1999, Hill et al., 2005). The level of education qualifications was shown to be a predictor of smoking levels in the target youth population in Table 7:4 (below). Education status was also demonstrated to vary by NeighFrag (Table 3:5). Again, it is possible that local norms around schooling and higher education could potentially be stronger in less fragmented neighbourhoods. However it is considered less likely that levels of fragmentation would substantially influence the level of qualifications reported by youth. Therefore the level of education qualifications was included as a potential confounder.

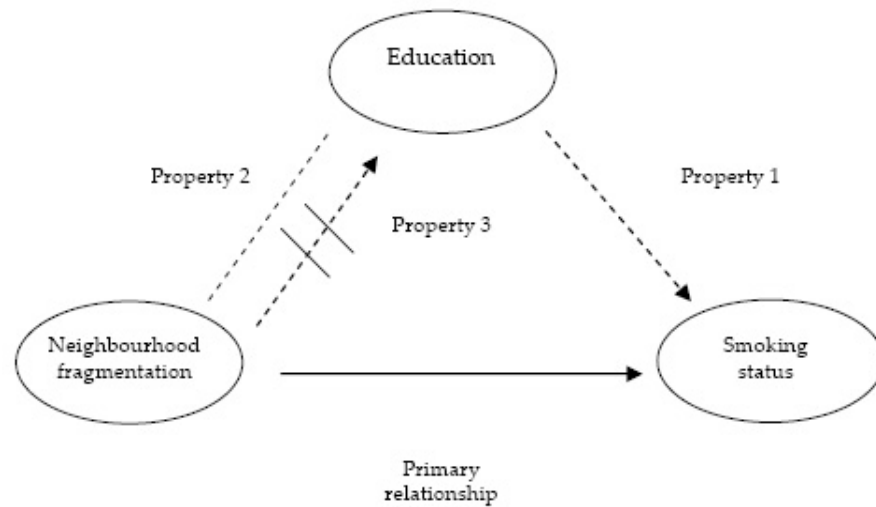


Figure 7:3 Education Confounding Triangle

Participation in the labour force was a predictor of smoking risk in the general New Zealand 1996 census population (the first property) (Borman et al., 1999). It was also shown to predict smoking in the target youth population (Table 7:4 (below)). The second property is also met in the youth population: while labour force status isn't associated with NeighFrag for the whole population it was for this smaller age range, no doubt reflecting the higher proportion of students in the age group (results not shown). There is the possibility that having more students in the neighbourhood might have increased the likelihood of becoming a student, but it is likely that there are much stronger drivers of labour force status than neighbourhood fragmentation. Therefore labour force status was included as a potential confounder (Figure 7:4)

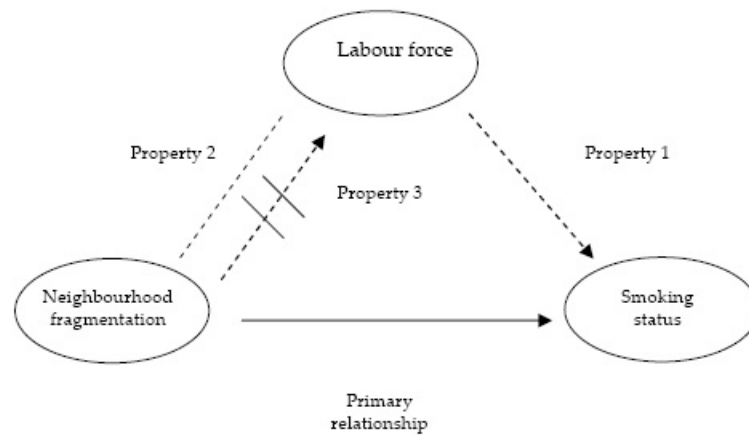


Figure 7:4 Labour Force Status Confounding Triangle

While household tenure is commonly used as a measure of socioeconomic status, and therefore a possible predictor of smoking in the literature (property 1); it is likely to have different meanings for this age group than for the general population. The likelihood of, for example, renting a house, is increased in the higher levels of fragmentation. However, the direction of the relationship between neighbourhood fragmentation and household tenure is unclear; tenure could be a result of fragmentation and therefore be on the causal pathway. Because of the uncertainty about the relationship between the variables and its meaning for the target population the second and third properties may be violated (Figure 7:5). Therefore household tenure was not included as a possible confounder in this analysis.



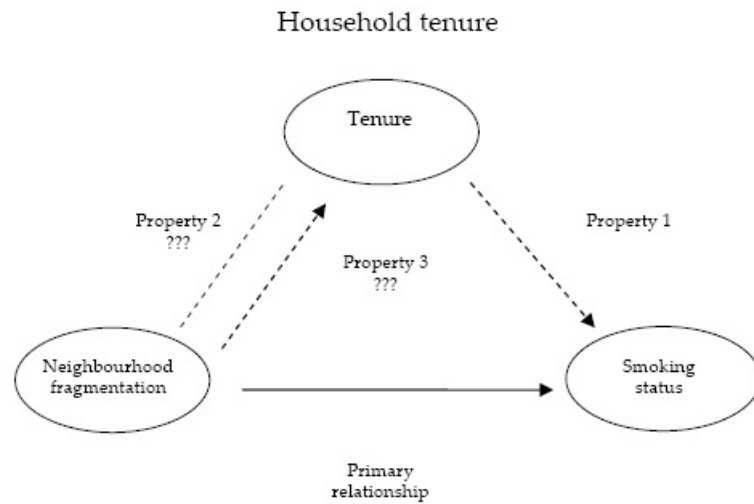


Figure 7:5 Household Tenure Confounding Triangle

Income, a commonly used marker of socioeconomic status, is also a commonly used predictor of smoking in the New Zealand adult population (Borman et al., 1999, Hill et al., 2005). However, there was limited evidence of an association with NeighFrag, challenging the second property. Furthermore, the reliability and validity of income data for this age group would have been uncertain. Finally, income information was only available at the household level and therefore would have not been available for those living in non-private dwellings. Because income did not appear to meet all properties (Figure 7:6) it was not included as a confounder.

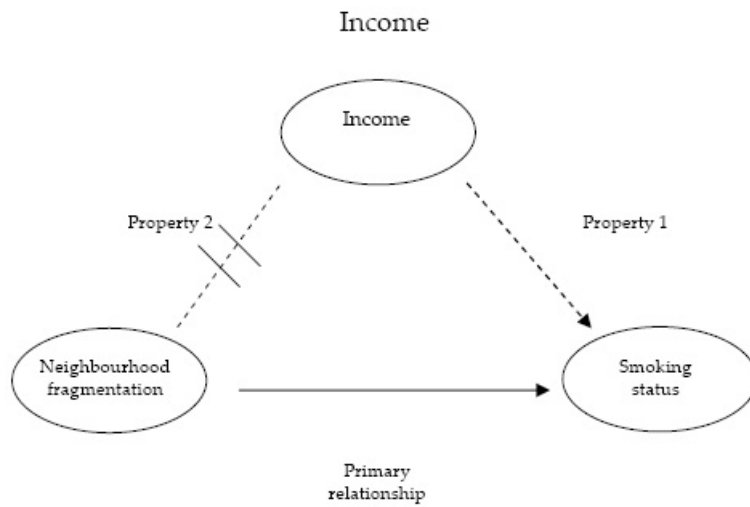


Figure 7:6 Income Confounding Triangle

As discussed above, area deprivation is a risk factor for individual smoking in the literature (Barnett, 2000), fulfilling property 1. NeighFrag and NZDep are moderately correlated (property 2). It is not known to what extent area fragmentation may influence the level of deprivation in a neighbourhood, which would mean that deprivation was in fact a mediator (Figure 7:7). However, given its frequent use in the literature as a confounder its contribution to confounding of the NeighFrag/smoking relationship will need to be explored. Therefore it will be included as a confounder in these analyses, but interpreted carefully.

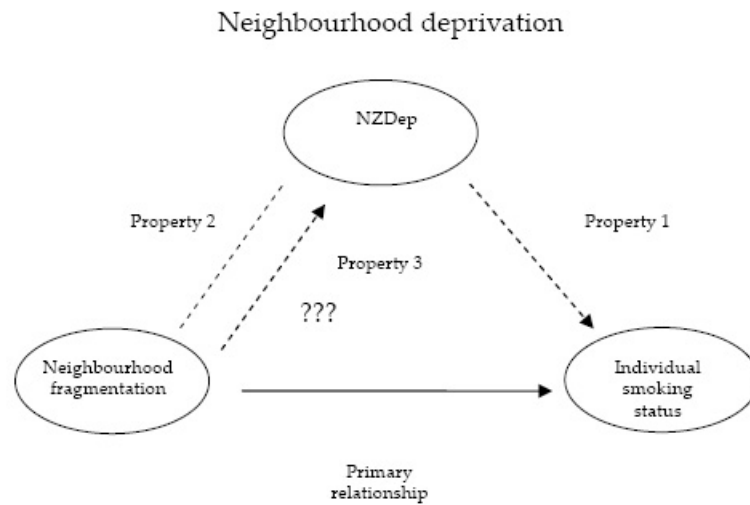


Figure 7:7 NZDep Confounding Triangle

### 7.1.4 Covariate Specification

Measures of the covariates that fulfilled the three properties were then operationalized from the dataset. All covariates were specified as categorical variables. The levels of each covariate were examined with cross tabulations with the neighbourhood exposures to enable groupings that provided meaningful contrast, whilst reducing the total number of categories for each regression, aiming for the general rule of thumb of three to four levels (Rothman and Greenland, 1998).

#### *Individual Level*

Self identified *ethnicity* was categorized into three groups; 'Māori'; 'Pacific People'; and 'non Māori, non Pacific' (reference group). The categories reflect the major groups of relevance to the New Zealand population and follow standard practice in New Zealand epidemiological analyses.

Highest *education* qualification was categorized into 'no qualifications'; 'school qualifications'; and 'post-school qualifications' (reference group).

*Labour force status* was categorized as 'unemployed' (and seeking work), 'employed' (reference group) and all other observations who were classified as 'non-labour force'.

#### *Neighbourhood Level*

*NZDep96*. Neighbourhood deprivation was modelled as a categorical variable in quintiles, with 'least deprived' (quintile 1) as the reference group.

## **7.2 Statistical Modelling**

### **7.2.1 Descriptive and Crude Analyses**

The data was initially examined to explore how the youth population was distributed by risk factors and NeighFrag, and to inform how the dataset should be restricted. The crude (unadjusted) relationships between the outcome and all exposures were examined, firstly for the whole youth population and then by age and sex groups to assess how the risk of smoking for the exposures might change across these groups. Risk ratios were calculated for the whole youth population to firstly compare the risk of smoking between categories of exposure. Crude odds ratios were also calculated for the whole youth population and then by age and sex group so that comparisons could be made between the unadjusted and fully adjusted odds ratios.

### **7.2.2 Adjusted Analyses**

Leading on from the crude analyses, multilevel logistic regressions were then used to calculate the adjusted odds ratios for NeighFrag and individual smoking, now accounting for the clustered nature of the data and available confounders. The model building process has been illustrated in Table 7:1. The dataset had a simple two-level data structure. Individuals were specified as level 1 (represented by the subscript  $i$  in the equations), and census area units (geographic neighbourhood) as level 2 (represented as  $j$  in the equations below). While NZDep is calculated at the meshblock level (Statistics New Zealand's smallest geographical scale) through a

process of aggregation it was provided in the dataset at the census area unit level, the same level as NeighFrag. All analyses were done by five year age and sex groups.

Table 7:1 Model Building for Research Question 1: Main Effect of NeighFrag

<i>Youth by 5year age &amp; sex groups</i>	<i>Model 1</i>	<i>Model 2</i>	<i>Model 3</i>
	<i>baseline model</i>	<i>with individual confounders</i>	<i>with individual and NZDep</i>
<i>with NeighFrag</i>	X	X	X
<i>with individual confounders</i>		X	X
<i>with NZDep</i>			X

The main effect of NeighFrag on risk of youth smoking was modelled in three stages with the following equations -

Model 1: 'empty model'

$$\log it(smoking_{ij}) = \beta_0 + \beta NeighFrag_j + (u_{0j} + e_{0ij})$$

Model 2: plus individual confounders

$$\log it(\gamma)_{ij} = \beta_0 + \beta NeighFrag_j + \beta Ethnicity_{ij} + \beta Labourforce_{ij} + \beta Education_{ij} + (u_{0j} + e_{0ij})$$

Model 3: plus neighbourhood deprivation

$$\log it(\gamma)_{ij} = \beta_0 + \beta NeighFrag_j + \beta Ethnicity_{ij} + \beta Labourforce_{ij} + \beta Education_{ij} + \beta NZDep_{ij} + (u_{0j} + e_{0ij})$$

The beta coefficients for NeighFrag generated the odds ratio of NeighFrag for smoking with 95% confidence intervals. Changes in magnitude and precision of the NeighFrag odds ratios were observed at each stage as evidence of the magnitude of confounding (Rothman and Greenland, 1998).

The analyses were completed in SAS version 8.2 in Statistics New Zealand secure data laboratory. All output counts of data have been random rounded to 3 in

accordance with Statistics New Zealand confidentiality requirements. The adjusted analyses were run with actual unit level data using the SAS Glimmix macro.

## **7.3 Results**

### **7.3.1 Descriptive Analyses: Who Lives Where?**

Generally, the youth population was distributed by neighbourhood fragmentation in a similar fashion to the adult population (as reported in Chapter Three). They tended to live in more fragmented neighbourhoods, with 17% of all youth living in decile NF10 (Table 7:2). As seen in the whole population, in decile NF 1 youth were distributed almost evenly across urban and non-urban settings, whereas in decile NF10 almost all live in urban settings. While there was little difference by sex across the deciles, age was related to where the target population lived. 20-24 year olds were more likely to be found in more fragmented neighbourhoods and 15-19 year olds in the least fragmented areas. The percentage of complete information on exposure and outcome reduced as fragmentation increased, except for a slight drop at decile 6. However, overall the range was still relatively small, between approximately 7% and 10%.

The proportion of youth living in non-private dwellings increased with fragmentation. In NF8-10 almost 12% of the youth population would have been excluded if the analysis had been restricted to usual residents, private dwellings, compared to an average of only 1% in the less fragmented areas. While household level information would have increased the ability to control for confounding, the corresponding potential increase in selection bias may have threatened the validity of any interpretation.

Table 7:2 Distribution of all Youth Across NeighFrag Deciles, by Restriction Factors.

Restriction factors	Percentage in Each Category for NF Decile Groups						Total % for Variable Level
	<i>NeighFrag Deciles</i>						
<i>Variable</i>	<b>1</b>	<b>2,3</b>	<b>4,5</b>	<b>6,7</b>	<b>8,9</b>	<b>10</b>	
<b><i>Urbanicity</i></b>							
Minor urban & rural	51.5	39.0	35.3	16.3	3.8	4.5	19.0
Urban	48.5	61.0	64.7	83.7	96.2	95.5	81.0
<b><i>Sex</i></b>							
female	45.7	47.6	48.7	49.7	51.0	52.1	49.8
Male	54.3	52.5	51.3	50.3	49.0	47.9	50.2
<b><i>Age</i></b>							
15-19 years	58.6	54.9	53.2	50.9	46.9	39.5	49.2
20-24 years	41.4	45.1	46.8	49.1	53.1	60.5	50.8
<b><i>Outcome &amp; Exposure Data</i></b>							
complete data	93.0	91.9	91.4	91.2	90.3	90.0	91.0
missing data	7.0	8.1	8.6	8.8	9.7	10.0	9.0
<b><i>Dwelling Type</i></b>							
Usual resident (UR), private dwelling	91.9	92.8	93.0	93.1	93.2	84.1	91.5
UR, non-private dwelling, in neighbourhood	1.0	0.8	1.3	1.4	2.0	11.8	3.2
UR, not exposed to neighbourhood	7.1	6.4	5.7	5.5	4.7	4.2	5.3
<b><i>Total % for NF decile</i></b>	<b>4.5</b>	<b>13.9</b>	<b>17.7</b>	<b>20.1</b>	<b>26.7</b>	<b>17.0</b>	<b>100</b>

All counts have been random rounded.

Figure 7:8 illustrates the reduction in the available observations following the restriction process outlined in the methods section. Of the whole youth census population, 94.7% lived in dwellings hypothesised to expose them to the neighbourhood environment. 77.1% of all youth lived in urban settings and of these, approximately 10% had neither outcome nor exposure information, leaving approximately 376,700 observations (all counts have been random rounded to meet confidentiality requirements). As discussed above, the dataset was stratified into five year age and sex groups, across which the youth population was relatively evenly spread. The restriction process excluded almost a third of youth, with urbanicity being the primary factor.

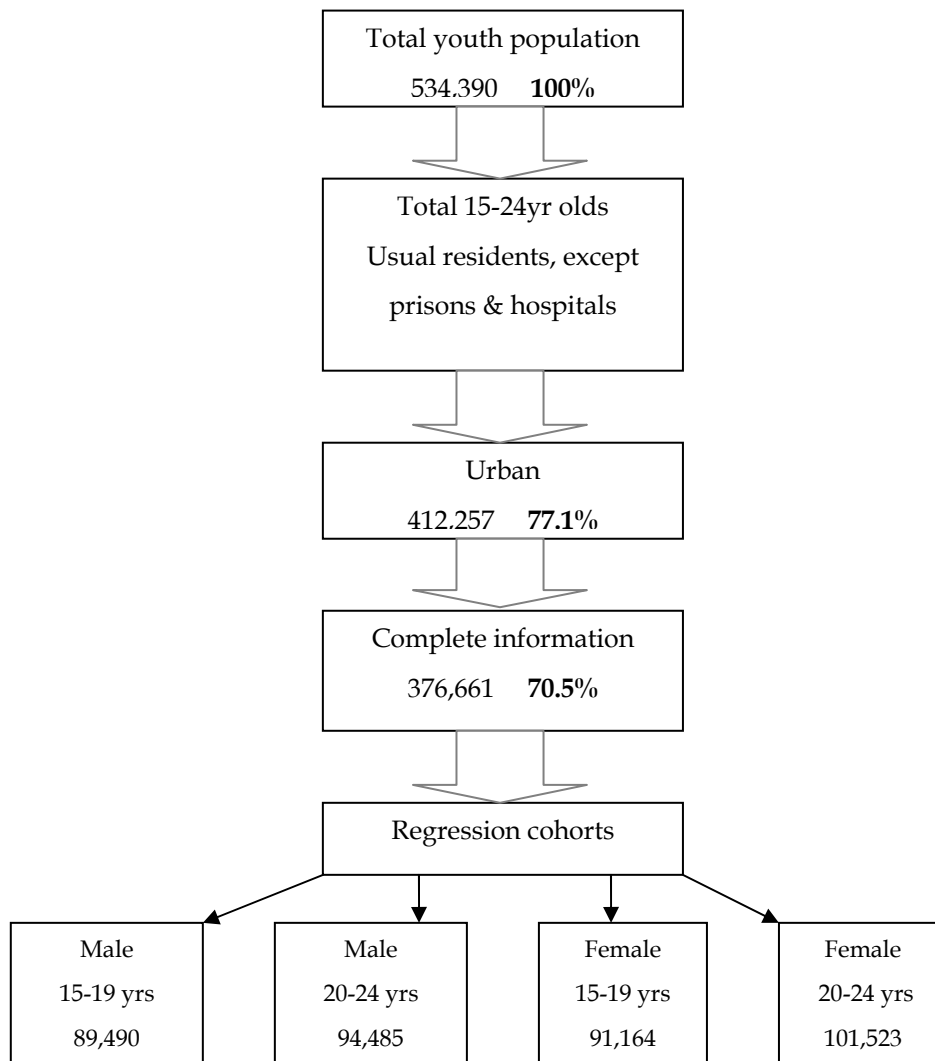


Figure 7:8 Numbers and Percentages for Each Restriction Stage – Total Youth as Denominator. All counts have been random rounded.



The distribution across NeighFrag by age and sex did not vary substantially from that shown when the data was restricted to the exposed urban youth population and has not been therefore shown separately.

Cross tabulations of NeighFrag and NZDep were examined to further assess the possibility of correlation between the two neighbourhood exposures. Table 7:3 demonstrated a higher level of co linearity for urban 15-24 year olds than had been observed for the whole population. Also of concern were the empty cells in the highest levels of deprivation for NF1 and the generally small numbers living in decile NF1. While sparse data and high correlation between variables create many statistical problems, on a pragmatic level they reflect the reality of how the New Zealand population is structured by neighbourhood. As leaving NZDep out of the model would have omitted important information on the independent NeighFrag/smoking association it was important to retain it in the analyses. But careful observation of the possible effect of co linearity was equally important, for example, increasing standard errors and widening confidence intervals and unstable estimates (Gujarati, 1999).

Table 7:3 Cross Tabulation of NeighFrag and NZDep for 15-24 Year Olds

<i>NZDep deciles</i>	<i>NeighFrag Decile Groups</i>						<i>Total</i>
	<i>NF 1</i>	<i>NF 2,3</i>	<i>NF 4,5 (Ref)</i>	<i>NF 6,7</i>	<i>NF 8,9</i>	<i>NF 10</i>	
<i>Dec 1(least deprived)</i>	4,530	9,135	8,346	8,676	4,698	1,845	37,230
<i>Dec 2</i>	1,419	8,118	5,397	7,986	8,934	3,084	34,938
<i>Dec 3</i>	1,623	6,465	4,989	5,241	9,597	3,483	31,398
<i>Dec 4</i>	501	4,059	7,293	4,536	12,225	4,398	33,012
<i>Dec 5</i>	636	2,976	5,541	10,281	7,755	4,776	31,965
<i>Dec 6</i>	576	3,501	5,529	11,691	9,720	5,364	36,381
<i>Dec 7</i>	402	2,778	6,873	9,957	10,608	13,038	43,656
<i>Dec 8</i>	528	1,119	3,531	8,172	15,783	11,907	41,040
<i>Dec 9</i>	-	780	2,223	8,229	18,747	19,473	49,452
<i>Dec10</i>	-	1,065	3,066	5,766	19,935	7,746	37,578
<i>Total</i>	10,215	39,996	52,788	80,535	118,002	75,114	376,650

All counts have been random rounded.

### 7.3.2 Crude Analyses: Who Smokes?

Overall, 25.6% of youth with complete data smoked regularly (Table 7:4). However the percentage varied considerably according to individual and neighbourhood characteristics. Smokers were more likely to be women, 20-24 year olds, Māori, have no education qualifications and be unemployed. Those least likely to be smokers were nonMāori, nonPacific (abbreviated to nMnP in the tables), have school or post school qualifications and not be in the labour force. Of the neighbourhood level exposures, those living in the most deprived and living in NF8,9 neighbourhoods were most likely to smoke.

Table 7:4 The characteristics of Youth who Smoke in the Regression Population

	<i>n with smoking data</i>	<i>n smokers</i>	<i>% who smoke</i>	<i>Risk Ratio</i>	<i>Unadjusted Odds Ratio</i>
<b><i>Ethnicity</i></b>					
<i>Māori</i>	64,407	26,370	40.90%	1.84	2.43
<i>Pacific</i>	27,552	6,774	24.60%	1.11	1.14
<i>nonMāori,nonPacific</i>	279,885	62,046	22.20%	1	1
<b><i>Education</i></b>					
<i>no qualifications</i>	86,640	32,742	37.80%	1.73	2.16
<i>Qualifications</i>	290,019	63,597	21.90%	1.	1.
<b><i>Labour Force Status</i></b>					
<i>unemployed</i>	40,917	14,874	36.40%	1.43	1.68
<i>non-labour</i>	111,831	24,543	21.90%	0.86	0.82
<i>employed</i>	223,911	56,925	25.40%	1	1
<b><i>NeighFrag</i></b>					
<i>decile 1</i>	10,215	2,136	20.90%	0.84	0.79
<i>deciles 2,3</i>	39,999	9,477	23.70%	0.95	0.93
<i>deciles 4,5</i>	52,791	13,203	25.00%	1	1
<i>deciles 6,7</i>	80,535	21,504	26.70%	1.07	1.09
<i>deciles 8,9</i>	117,999	32,295	27.40%	1.10	1.13
<i>decile 10</i>	75,123	17,727	23.60%	0.94	0.93
<i>decile 10/decile 1</i>				1.13	1.17
<b><i>Neighbourhood Deprivation</i></b>					
<i>quintile 1 (least deprived)</i>	72,174	12,312	17.10%	1	1
<i>quintile 2</i>	64,410	13,629	21.20%	1.24	1.30
<i>quintile 3</i>	68,355	17,667	25.80%	1.51	1.69
<i>quintile 4</i>	84,696	24,249	28.60%	1.67	1.95
<i>quintile 5 (most deprived)</i>	87,030	28,485	32.70%	1.91	2.37
<b><i>total</i></b>	376,659	96,339	25.60%	1.50	1.67

All counts have been random rounded.

In order to observe the strength of the crude association between variables and smoking, odds ratios (OR) were calculated. The odds ratios were calculated from the odds of smoking (numbers of smokers divided by non-smokers) in the exposed category, divided by the odds in the unexposed category. They were later compared with the fully adjusted ratios calculated from the logistics regressions to assess the effect of adjusting for confounders and clustering. It was interesting to note the divergence between the OR's and RR's in the higher risk categories (for example, the 'Māori compared to non-Māori, non-Pacific' RR was 1.84 compared to the OR 2.43). Given the commonness of the outcome in the 'unexposed' category this was not surprising as the two measures are known to diverge when the outcome is common in the unexposed group (Kirkwood et al., 2003).

Overall, the size of the NeighFrag OR's was relatively small when compared to other contextual and individual factors, but not inconsequential (Table 7:4). Living in either the highest or lowest NeighFrag deciles was associated with a lower risk of odds of smoking compared with the moderate reference group (deciles NF4,5). The strongest association was when comparing decile NF1 with the 'unexposed' reference group, an OR of 0.79. A protective association was also found when comparing the odds of smoking in NF10 and NF4,5 but it was much smaller (OR 0.93). However, higher odds ratios were found at moderately higher levels indicating a non-linear trend. When the odds were compared across the whole NeighFrag range the OR was 1.17, indicating that for youth generally, living in levels of the highest fragmentation compared with the least fragmented neighbourhoods was associated with a slightly increased risk of smoking.

When the youth population was stratified by age and sex group, however, two different patterns emerged. The magnitude and direction of the association between NeighFrag and smoking varied by the four sex and age groups (Tables Appendix 2:1-4). A protective association was observed for the younger ages when comparing the least fragmented decile with the moderate reference group, most notably for 15-19 year old women (OR 0.61) (Tables Appendix 2:1). In contrast, living in the most

fragmented neighbourhoods (compared with NF4,5) group was associated with a decrease in smoking risk for the 20-24 year olds, most notably again for women (OR 0.71) (Tables Appendix 2:3).

While not being in the labour force was protective against smoking for all youth when compared to all other categories, it was associated with increased odds of smoking for women aged 20-24 (OR 1.40). This may be because the category did not distinguish between students and, for example, young mothers (early parenting being a known risk factor for smoking (Graham et al., 2006). Similarly, while having no qualifications was associated with an increased risk of smoking for 15-19 year olds (men: OR 1.95), it was much larger in the older age group (men: OR 3.44), indicating the increased probability that they have no qualifications because they are no longer in the education system rather than still working towards a qualification. It was therefore possible that proxies used to measure confounding captured different constructs in each group.

The effect of confounding exposures on the risk of smoking was greater for younger women than for other age and sex groups. For example, the odds of smoking for younger Māori women compared to non-Māori, non-Pacific greatly increased (OR 3:10), whereas, for example, the comparable OR for 20-24 year old men was almost half (1.86). Younger women also demonstrated a greater sensitivity to contextual factors than any other group, with an OR of 2.93 for most deprived (compared to least deprived), compared with 1.93 for 20-24 year old men.

### **7.3.3 Adjusted Analyses**

The adjusted analyses provided further evidence of the associations seen in the crude analyses, this time adjusting for likely known confounders. There was some initial empirical support for a cross-level main effect of neighbourhood level of fragmentation on the risk of smoking. In 15-24 year olds the individual odds of smoking was associated with extreme levels of NeighFrag, and this association

generally remained after adjusting for ethnicity, education, labour force status and NZDep (Table 7:5).

Table 7.5 The Association of NeighFrag and Youth smoking: Adjusted Odds Ratios (95% confidence intervals)

	The Association of NeighFrag and Youth smoking: Adjusted Odds Ratios (95% confidence intervals)			
	15-19 year old women	15-19 year old men	20-24 year old women	20-24 year old men
<i>Neighbourhood fragmentation</i>				
<i>NF Dec 1 (least fragmented)</i>	0.85 (0.74-0.99)	0.92 (0.80-1.06)	1.21 (1.06-1.39)	1.04 (0.92-1.19)
<i>NF Dec 2,3</i>	0.96 (0.87-1.05)	1.08 (0.99-1.18)	1.15 (1.05-1.25)	1.02 (0.95-1.11)
<i>NF Dec 4,5 (Ref)</i>	1	1	1	1
<i>NF Dec 6,7</i>	0.97 (0.89-1.05)	1.04 (0.96-1.12)	0.97 (0.90-1.04)	0.96 (0.90-1.03)
<i>NF Dec 8,9</i>	0.97 (0.90-1.05)	1.07 (0.99-1.15)	0.90 (0.84-0.97)	0.95 (0.89-1.01)
<i>NF Dec 10 (most fragmented)</i>	1.06 (0.96-1.16)	1.22 (1.12-1.34)	0.74 (0.68-0.80)	0.84 (0.78-0.91)
<i>Random variance (s.e. of variance)</i>				
<i>Individual level</i>	0.970 (0.0046)	0.980 (0.0047)	0.980 (0.0044)	0.980 (0.0046)
<i>Neighbourhood level</i>	0.074 (0.0067)	0.059 (0.0064)	0.066 (0.0055)	0.045 (0.0045)

Adjusted for ethnicity, education, labour force status and neighbourhood deprivation

For the 15-19 year olds the direction of the association suggested initial support for NeighFrag acting as a ‘social deficit’ exposure. In this age group increasing fragmentation was associated with an increased risk of smoking. In the older age group the opposite was observed with a protective association between NeighFrag and the risk of smoking. The pattern of the opposing associations has been illustrated in Figure 7:9. The figures have been presented by sex so that the contrasting associations by age group could be more easily observed. The text below has concentrated on comparing the age related differences. Presenting the results visually allowed attention to be placed on the patterns of the associations. Of course the interpretation of the pattern would still need to be cognisant of the precision of the estimates. The report below has focused on the fully adjusted model (model 3) presented in Figure 7:9a&b and Table 7:5, with more detailed results tables presented in Appendix 3:1-4.

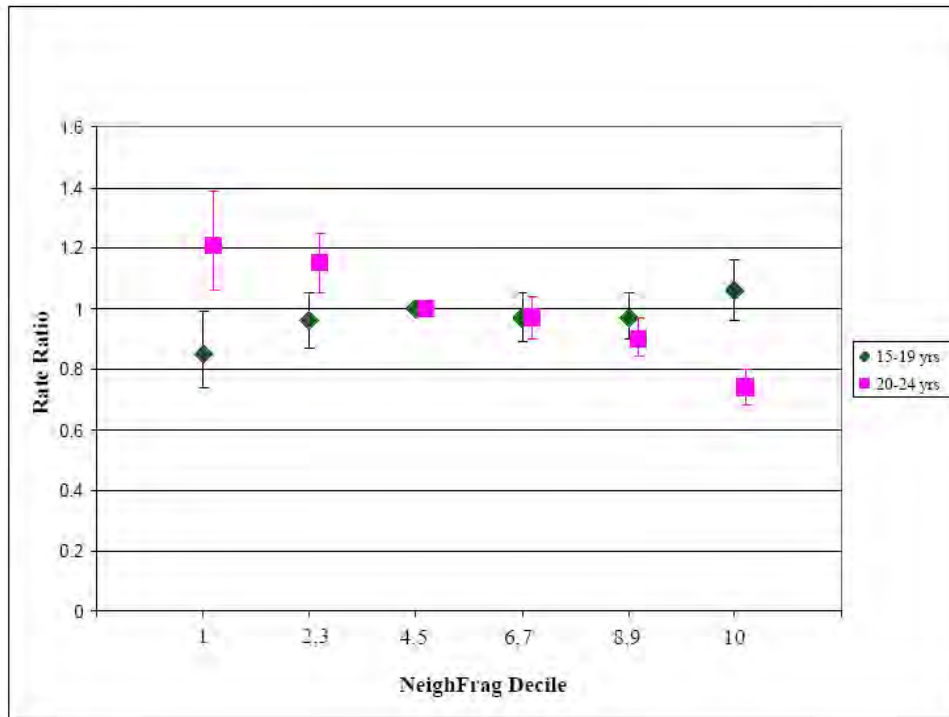


Figure 7:9a The Association of NeighFrag and Youth Smoking: Adjusted Odds Ratios – Women

Adjusted for individual education, ethnicity, labour force status, neighbourhood deprivation. 95% confidence intervals

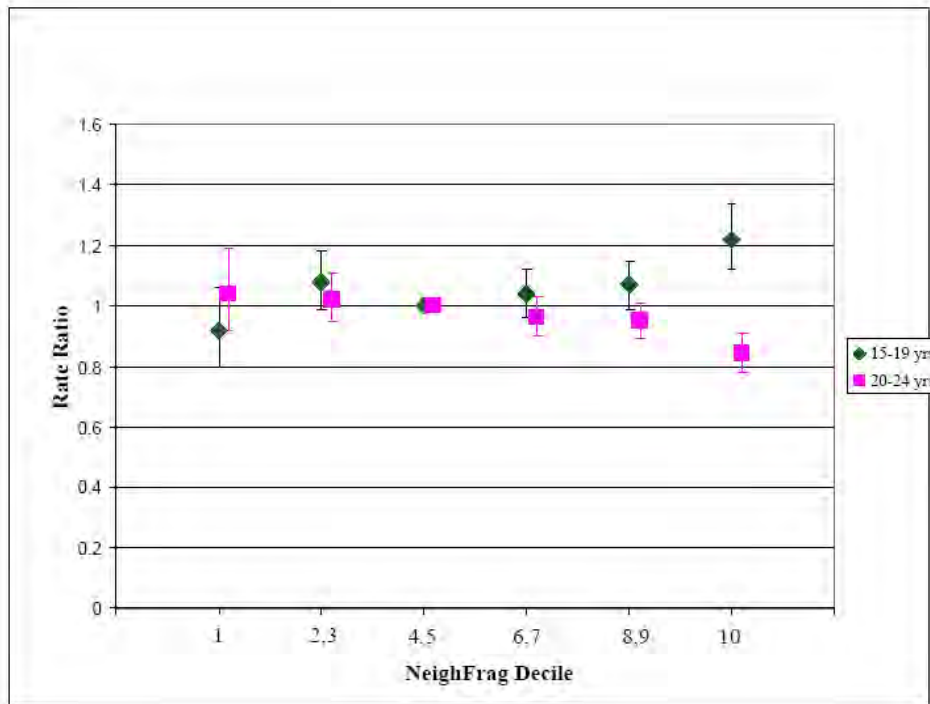


Figure 7:9b The Association of NeighFrag and Youth smoking: Adjusted Odds Ratios – Men

Adjusted for individual education, ethnicity, labour force status, neighbourhood deprivation. 95% confidence intervals

### ***7.3.3.1 15-19 Year Olds: Adverse Association***

Increasing neighbourhood fragmentation, relative to the reference group (NF4,5) was associated with an increased odds ratio of smoking for 15-19 year old women and men (Table 7:5 and Appendices 3:1-2). While deciles NF8-10 had an adverse risk of smoking (that increased in magnitude), living in deciles NF1-3 had an increasingly protective effect on the risk of smoking for this group. A dose response relationship is suggested by the change from a protective effect for decile NF1, relative to the reference group (0.85(0.74-0.99) for women; 0.92(0.80-1.06) for men), all the way through to the increasingly protective effect in decile NF10 (1.06(0.96-1.16) for women; 1.22(1.12-1.43) for men).

The magnitude and precision of the association varied by sex. For women the confidence intervals for the fully adjusted odds ratios all included 1, except for NF1. The statistically significant association observed in NF1 needs to be interpreted with the sparse data in NF1 in mind. However, when the pattern of the association is observed in the Figures 7:9a and b, a suggestion remained of an adverse dose response relationship between NeighFrag and smoking for 15-19 year olds women. Importantly, the NF1 OR did not contradict the protective pattern, despite the wide confidence intervals.

A similarly weak association, but with the same pattern, was observed for 15-19 year old men. In the fully adjusted model, the only statistically significant effect was for NF10 and the dose response was less clear between deciles NF3-7. However, when the pattern was compared across the two sex groups the nature of the adverse association of NeighFrag on the risk of smoking was similar, even though the statistically significant odds ratios were at opposite ends.



### ***7.3.3.2 20-24 Year Olds: Protective Association***

The pattern of the association of NeighFrag and smoking in the older age groups was opposite to that observed in the younger age group: the individual risk of smoking decreased for those living in higher rather than lower levels of fragmentation (Table 7:5 and Appendices 3:3-4). The magnitude of the association was larger for women, with evidence of a dose response. In the fully adjusted model statistically significant odds ratios were observed for NF1-3 and NF8-10. Again, the estimates for NF 1 should be interpreted with caution because of the sparse data, particularly as NZDep was added to the model.

In 20-24 year old men a protective effect was observed for living in NF10, which remained after controlling for individual and deprivation factors (0.84(0.78-0.91)) (Table 7:5). The magnitude of the effect in NF10 was similar to that observed for other age and sex groups, and the direction was in keeping with the protective effect observed for the older women (0.74(0.68-0.80)), suggesting that the association of NeighFrag on smoking was modified by age group. There was no evidence of a statistically significant association of fragmentation on the individual odds of smoking at other levels, with the OR's of small size and the confidence intervals all including 1.

### ***7.3.3.3 Controlling for Confounding.***

Observing the change in the unadjusted OR's from the crude estimates with the fully adjusted ones emphasised the effect of simultaneously controlling for all covariates and the multilevel nature of the data. Generally there was little change in the NeighFrag OR's obtained from the crude and baseline regression estimates (model 1 in Table Appendix 3:1-4), suggesting that controlling for clustering had a minimal effect. The exception was in the NF10 estimates for where the OR was larger in the regression model (for example, 15-19 year old women went from 1.08 (crude) to 1.24(1.09-1.44) (adjusted). There was no change in the NF10 OR for 20-24 year old men, remaining at 0.79 in both calculations (Table Appendix 3:4).

The addition of covariates had a varying impact on the NeighFrag estimates (model 2). Generally, the NeighFrag OR's were weaker when compared to the baseline OR's when individual factors were added in model 2. In 15-19 year olds however, the size of the NF10 OR was larger in the second model. The addition of NZDep in model 3 produced the biggest change in estimates. Of interest was the indication of negative confounding by NZDep for 20-24 year old women; the OR's in model 3 (0.74(0.68-0.80) were much stronger than model 2 (0.88(0.80-0.97)). The width of the confidence intervals for NeighFrag remained stable as area characteristics were added in to the models, indicating that co linearity with NZDep was not an issue (Kirkwood et al., 2003).

The association between smoking and the individual confounding exposures was generally weaker when comparing the crude and adjusted analyses. The change suggested that there were important interrelationships between confounders that were better captured in the simultaneous adjustment, compared with treating each risk factor as separate. This was particularly noticeable for 15-19 year old men. Only a small change in the estimates was observed between the baseline and fully adjusted model, which could indicate that confounding factors had been poorly captured for this group. On the other hand, a larger change was observed when comparing the unadjusted and fully adjusted regression OR's. It may therefore be that the factors were a distorting influence when they were in combination with each other, rather than when treated as individual confounders in the stratified analyses.

In summary, NeighFrag was found to have an association with the individual risk of smoking. That is to say, there was evidence of a cross-level main effect between the ecological level exposure and individual level outcome. The association remained, but was generally weaker, after adjustment for individual and neighbourhood level confounders. Interestingly, the magnitude and direction of the association was modified across the sex and age groups. An adverse association was observed for 15-19 year olds, suggesting support for the 'social deficit' hypothesis. However a contrasting protective pattern was observed in the NeighFrag estimates for 20-24

year olds. Possible sources of error for the associations are discussed at the end of the chapter. A more substantive interpretation of the results is undertaken in Chapter Eight, focusing on the external validity of the associations and potential alternative explanations.

## **Section 2: Transmission of Smoking Practices**

The second research question examines whether NeighFrag modified the effect of another neighbourhood exposure in the individual risk of smoking, using a within-level effect modification analysis. The epidemiological approach employed allowed empirical observation of the transmission mechanism discussed in chapter three: specifically, might the transmission of health-related social practices or norms within neighbourhoods vary by NeighFrag levels? This was examined in the following analyses by observing the relationship between the neighbourhood adult smoking environment (representing the norms and practices around individuals) and youth individual smoking behaviour, as potentially modified by NeighFrag.

It has been hypothesised that less fragmented neighbourhoods would be more likely to have strong ties and networks necessary for transmission of health norms within the neighbourhood. It was expected that those living in less fragmented neighbourhoods will not only be more exposed to the smoking practices of their neighbours, but also that, because of the higher levels of regulation and integration, they will be more strongly affected by the neighbourhood smoking environment than those living in highly fragmented neighbourhoods. For example, youth living in a low adult smoking neighbourhood would be less likely to smoke, and youth in a high smoking neighbourhood would be more likely to be likely to smoke.

On the other hand, it was expected that the within-neighbourhood transmission of neighbourhood adult smoking norms to youth would be weaker in highly fragmented neighbourhoods. In other words, the effect of adult residents' smoking practices on the smoking behaviours of individuals was expected to be weaker in the more fragmented neighbourhoods.

The research questions for these analyses were –

- 1) *To what extent did NeighFrag modify the relationship between neighbourhood adult smoking environment and individual youth smoking risk?*
- 2) *If so, was the association between the neighbourhood adult smoking environment and individual youth smoking risk weaker in highly fragmented neighbourhoods, in keeping with the transmission hypothesis?*

## **7.4 Methods**

### **7.4.1 Dataset**

In order to examine the modifying effect of neighbourhood smoking on the individual risk of smoking, the census dataset was split in two: the ‘target population’ and the ‘exposure’ population. This was to avoid the tautology created when attempting to measure the outcome and exposure using the same observed behaviour in one population. The target population was selected as 15-24 year olds (referred to as ‘youth’), and the exposure population was the remaining adult population (25+ year olds and referred to as ‘adults’). The target population was the same as that used in Section 1 analyses and so the details will only be specified here if they differ in some way.

### **7.4.2 Neighbourhood Adult Smoking (NAS)**

In order to measure the effect of the neighbourhood adult smoking environment, a measure needed to be created of the proportion of the remaining adult population who smoked, for each census area unit. The full census data set was first restricted to all usually resident 25 plus year olds with smoking data. Usual residents in hospitals and prisons were not included as youth were unlikely to be exposed to their smoking behaviour in the neighbourhood. From this restricted cohort proportions were created of the current smokers living in each area unit.

Number of current eligible adult smokers in area unit

Total eligible adults in area unit

The resulting area proportions were ranked and then grouped into deciles. Each area unit was assigned a decile ranking for Neighbourhood Adult Smoking (NAS), with decile 1 as the least proportion of current smokers (the least 'exposed') and decile 10 (the most 'exposed'). In order to examine the average difference in effect across the decile range, each decile was assigned a value between 0.05 and 0.95. The resulting ratios represented a log odds change in the association, or the slope, from neighbourhoods with the lowest proportion of adult smoking to the highest proportion of adult smoking.

The data was initially examined to explore the smoking/NAS relationship. Cross tabulations were used to examine how the individual risk of smoking changed for each level of the NAS exposure. Odds ratios were calculated to give the crude estimates without accounting for confounders or the clustering of the data.

There was considerable variation in the proportion of adult smokers at the census area unit level, ranging from 8% - 56 % (not shown). Urban youth with smoking data were unevenly distributed across the quintiles, with 16% in the least smoking compared to 36% in the most (Table 7:6). Living in a high smoking compared to a low smoking neighbourhood had an expectedly large effect on the risk of smoking with a risk ratio of 2.18. The increase in risk by NAS was monotonic, indicating a linear trend.

Table 7:6 Unadjusted Risk of Youth Smoking by Neighbourhood Adult Smoking – All Youth

	<i>n with smoking data</i>	<i>n smokers</i>	<i>%</i>	<i>Risk ratios</i>	<i>Crude odds ratios</i>
<b><i>Neighbourhood Adult Smoking (NAS)</i></b>					
<i>Quintile 1 (least smoking)</i>	96,891	16,095	16.6%	1	1
2	81,372	17,979	22.1%	1.33	1.42
3	65,379	17,790	27.2%	1.64	1.88
4	73,938	23,070	31.2%	1.88	2.28
<i>5 (most smoking)</i>	59,079	21,408	36.2%	2.18	2.85

All counts have been random rounded

### 7.4.3 Adjusted Analyses

A three step process was used to investigate effect modification by NeighFrag on the effect of Neighbourhood Adult Smoking on individual youth smoking, as illustrated in Table 7:7. Because the primary focus was on the modifying effect of NeighFrag only the fully adjusted results for NAS will be presented. The same confounding variables were used as for Section 1 analyses. As all the variables selected for those analyses would be risk factors for the adult population and would therefore be associated with NAS, it was considered satisfactory to assume that important factors for youth had been adequately accounted for. Furthermore, the primary focus was on NeighFrag, not on establishing NAS as a risk factor exposure.

Table 7:7 Model Building for Effect Modification of NAS by NF

	<i>Model 1</i>	<i>Model 2</i>			<i>Model 3</i>
		<i>A</i>	<i>B</i>	<i>C</i>	
<i>Youth by 5yr Age &amp; Sex Groups</i>	<i>youth population</i>	<i>youth in each NF decile group stratified</i>			
	<i>fully adjusted</i>	<i>NF 2&amp;3</i>	<i>NF 8&amp;9</i>	<i>NF 10</i>	<i>fully adjusted interaction</i>
<i>with NAS</i>	X	X	X	X	X
<i>with individual confounders</i>	X	X	X	X	X
<i>with NZDep</i>	X	X	X	X	X
<i>NAS*Nf</i>					X

Step 1: Main cross-level effect of NAS on individual youth smoking.

Multilevel logistic regressions were used to calculate the odds ratios as a measure of the effect of NAS on the odds of smoking in neighbourhoods with the highest level of adult smoking (the exposed group), compared to the least adult smoking neighbourhoods (the unexposed group). The model was fully adjusted for individual confounders and NZDep.

Model 1: fully adjusted NAS main effect

$$1. \logit(\gamma)_{ij} = \beta_0 + \beta NAS_j + \beta Ethnicity_{ij} + \beta Labourforce_{ij} + \beta Education_{ij} + \beta NZDep_j + (u_{0j} + e_{0ij})$$

Step 2: Stratification of the fully adjusted NAS model by NeighFrag.

Levels of NeighFrag exposure were selected that allowed the comparison of low (NF2,3) and high (NF8,9) fragmentation. Decile NF10 was also selected as it appears to represent an extreme. While it would have been interesting to have observed the other extreme of fragmentation, decile 1, the numbers in the strata were insufficient for meaningful interpretation of the estimates. The target population was first restricted to those living in the deciles of interest. The fully adjusted odds ratios for exposure to NAS were then calculated separately for each stratum. Evidence was sought of heterogeneity in the odds ratios across the stratified levels as evidence of effect modification.

Model 2 a, b, c: model 1 by NF2,3, NF8,9, NF10

$$\log it(\gamma)_{ij} = \beta_0 + \beta NAS_j + \beta Ethnicity_{ij} + \beta Labourforce_{ij} + \beta Education_{ij} + \beta NZDep_j + (u_{0j} + e_{0ij})$$

Step 3: statistical interaction between NAS and NeighFrag.

An interaction term was added to the regression equation, modelling an interaction between NAS and NeighFrag. The reference group for the NAS\*NeighFrag term was set as deciles NF2,3 so that the resulting OR's for deciles NF8,9 and NF10 could be compared to the stratified results.

Model 3: interaction terms

$$\log it(\gamma)_{ij} = \beta_0 + \beta NAS_j + \beta confounders_{ij} + \beta NZDep_j + \beta NeighFrag_j + \beta NAS * \beta NeighFrag_j + (u_{0j} + e_{0ij})$$

The analyses were completed in SAS version 8.2 in Statistics New Zealand secure data laboratory. All output counts of data have been random rounded to 3 in accordance with Statistics New Zealand confidentiality requirements. The adjusted analyses were run with actual unit level data using the SAS Glimmix macro.



## 7.4.4 Results

### 7.4.4.1 Step 1) Adjusted Cross-level Main Effect of NAS and Youth Smoking

An unsurprisingly strong association was observed between the fully adjusted risk of smoking and Neighbourhood Adult Smoking (NAS) for the four age and sex groups and has been illustrated in Figure 7:10. A substantial increase in smoking risk was observed for those living in neighbourhoods with the highest levels of adult smoking compared with those living in areas with the lowest levels of smoking, after controlling for individual risk factors and neighbourhood deprivation. The effect was modified by age and sex, being strongest for the 20-24 year olds (particularly women) and weakest for the younger age group (particularly men).

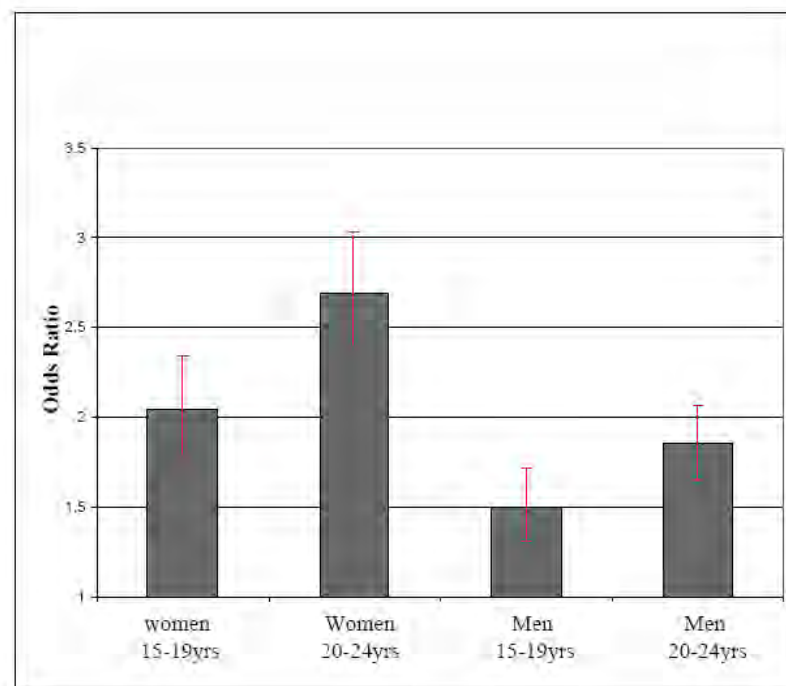


Figure 7:10 The Association of Neighbourhood Adult Smoking with Youth Smoking: Adjusted Odds Ratios by Strata of NeighFrag

Adjusted for individual education, ethnicity, labour force status, neighbourhood deprivation. 95% confidence intervals.

While no formal tests of interaction were run (as the NAS/smoking relationship was a tool for observing effect modification by NF, rather than being of substantive interest in itself), the non-overlapping confidence intervals between the two age groups of each sex suggests that the modification by age at least is unlikely to simply

be due to chance. Further details of the regression results are presented in Tables Appendix 4:1-4.

**7.4.4.2 Step 2) Stratification by NeighFrag**

The odds ratios for NAS and smoking was then calculated separately for three fragmentation strata and examined for heterogeneity. All age and sex groups demonstrated differences in the effect size for NAS across the three fragmentation strata, as seen in Figure 7:11. The heterogeneity supported a within-level effect modification by NeighFrag.

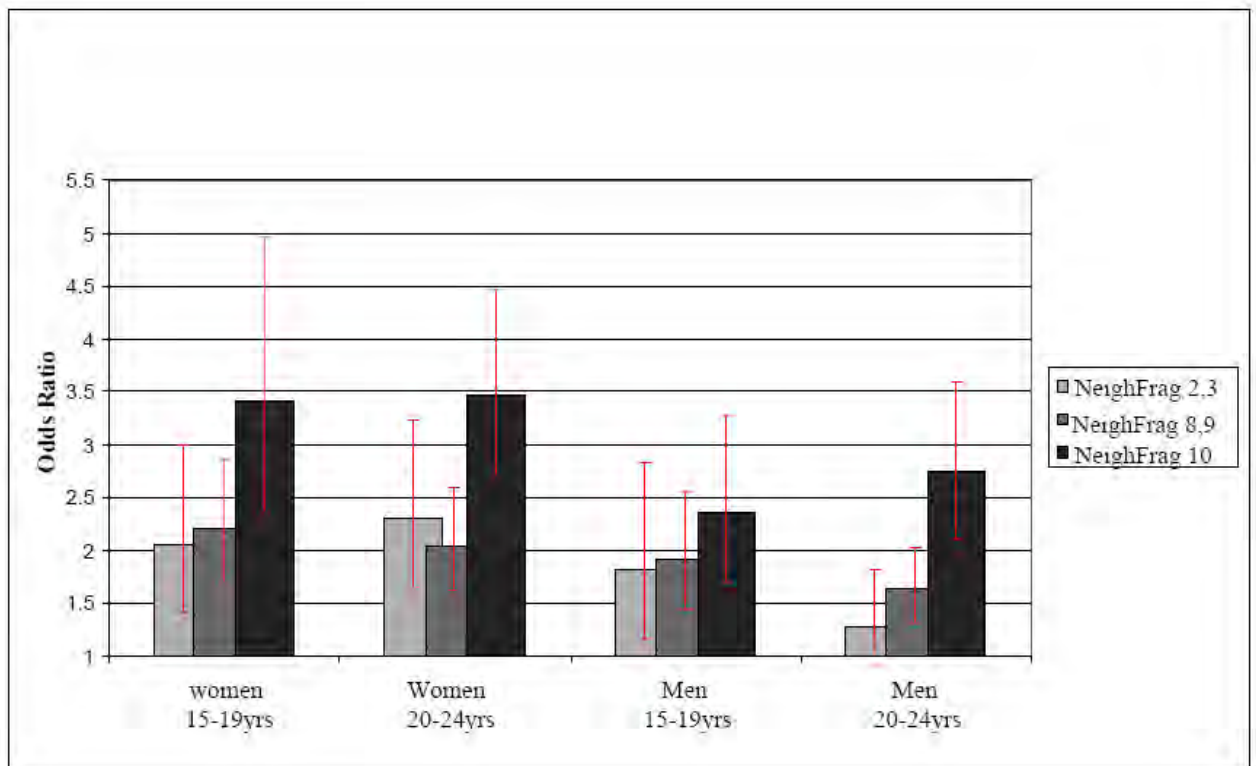


Figure 7:11 Adjusted Odds Ratios for NAS by NeighFrag Strata

Adjusted for individual education, ethnicity, labour force status, neighbourhood deprivation. 95% confidence intervals

However, the direction of modification was contrary to that hypothesised. The 'transmission' hypothesis had suggested that the association of NAS would be weaker in the higher levels of NeighFrag. Instead, the effect of NAS on the risk of smoking was consistently the strongest for those living in NeighFrag decile 10, compared to other levels of fragmentation and the risk observed in the non stratified cohort. Further, the confidence intervals of the NAS estimates for NF 2,3 and NF10 did not overlap for the 20-24 year olds, which provided further evidence for the modification being non-random. While there was an increase in the association of NAS in the deciles 8,9 for all but 20-24 year old women, the difference was much smaller than seen for decile NF10.

This suggested that the effect modification occurred primarily between the most highly fragmented neighbourhoods and all the other strata. If this were the case, a quick check could be done by comparing the NAS estimates and confidence intervals for model 1 (the whole youth estimate) with those in model 2c (NF 10). This was because the main driver of the estimates observed in the whole population would be of youth living in NF 1-9. Non-overlapping confidence intervals between the two models were in fact observed for 15-19 year old women and 20-24 year old men with only a 0.02 overlap for 15-19 year old men.

As would be expected with the reduction in the numbers of observations, confidence intervals for the neighbourhood characteristics were substantially increased in the stratified analyses when compared to the non-stratified cohort. The width of confidence intervals also varied by the level of fragmentation, reflecting the different sizes of each stratum. However, the decreased precision did not affect the statistical significance for individual characteristics or NAS.

#### ***7.4.4.3 Statistical Interaction Between NAS and NeighFrag***

An interaction term was specified in the regression to statistically test for heterogeneity as specified in the Model 3 equation above. Statistically significant interaction terms for all age and sex groups were observed for deciles NF10

compared to the reference group (deciles NF2,3), indicating that the difference was unlikely to simply have been due to chance (Table 7:8a-d). No statistically significant terms were observed at other levels, in line with the small differences observed between the decile groups NF8,9 and NF2,3. Hence the statistical interaction results support the findings from the stratified analyses and provide supportive evidence of a statistical interaction between NAS and the highest level of NeighFrag.

The results from the stratified and statistical methods were compared to further assess the empirical evidence for effect modification. The interaction term estimates were used to back calculate the odds ratios for NAS for the three strata of NeighFrag, this time taking into account the effect of statistical interaction between the neighbourhood exposures. The aim was to see if the interaction term estimates were comparable in magnitude to the difference in the odds ratios across the strata. The reference group was set as NAS \*NeighFrag deciles 2,3, the lowest of the NF strata. The NF2,3 OR used in the back calculation was taken from the NAS estimate in model 3 as it was equivalent to the reference group (NAS \*NeighFrag 2,3 OR). The interaction adjusted estimates for NF 8,9 and NF10 were obtained by multiplying this estimate by the interaction term estimates ( e.g. NAS \*NeighFrag decile10).

The results from the back calculation have been summarised in Table 7:9, showing OR's from the stratified and pooled analyses, with the added back calculation. The NAS OR's from NF2,3 strata and NF10 have been shown in column 1 and 2 respectively. The NAS\*NeighFrag 2,3 OR (that is, the NAS OR from model 3) in column 3 was comparable with the column 1 OR's, but now equivalently adjusted for covariates as in other strata. The interaction term OR in column 4 was the amount by which the reference group OR needs to be multiplied (being a logistic regression) to gain an equivalent estimate to the NF decile 10 strata in column 2. Therefore the OR's obtained by the statistical method in column 5 could be compared to those in column 2, obtained by the stratification method. For example, for 15-19 year old women, the back calculated OR of 3.18 ( $2.21 \times 1.44$ ) is similar in magnitude to the 3.42 OR obtained by the stratified results (in column 2).

Table 7:8a-d The Statistical Interaction of NAS and NeighFrag: Odds Ratios of Smoking

**A:15-19 yrs Women**

<i>Exposure Variables</i>	<i>Model 3</i>
<i>Neighbourhood Adult Smoking (NAS)</i> <i>Neighbourhood Adult Smoking (NAS)</i>	2.21 (1.71-2.85)
<i>NAS*NeighFrag</i>	
<i>NAS*Nf Dec 1 (least fragmented)</i>	0.96 (0.59-1.56)
<i>NAS*Nf Dec 2,3 (Ref)</i>	1
<i>NAS*Nf Dec 4,5</i>	1.01 (0.74-1.37)
<i>NAS*Nf Dec 6,7</i>	1.08 (0.81-1.43)
<i>NAS*Nf Dec 8,9</i>	0.99 (0.75-1.30)
<i>NAS*Nf Dec 10 ( most fragmented)</i>	1.44 (1.04-1.99)
<i>Random variance (s.e. of variance)</i>	
<i>Individual level</i>	0.980 (0.0046)
<i>Neighbourhood level</i>	0.052 (0.0057)

**B: 15-19 yrs Men**

<i>Exposure Variables</i>	<i>Model 3</i>
<i>Neighbourhood Adult Smoking (NAS)</i> <i>Neighbourhood Adult Smoking (NAS)</i>	1.87 (1.46-2.40)
<i>NAS*Neighbourhood Fragmentation</i>	
<i>NAS*Nf Dec 1 (least fragmented)</i>	1.12 (0.70-1.79)
<i>NAS*Nf Dec 2,3 (Ref)</i>	1
<i>NAS*Nf Dec 4,5</i>	0.86 (0.63-1.16)
<i>NAS*Nf Dec 6,7</i>	0.77 (0.59-1.02)
<i>NAS*Nf Dec 8,9</i>	0.83 (0.63-1.09)
<i>NAS*Nf Dec 10 ( most fragmented)</i>	1.40 (1.01-1.93)
<i>Random variance (s.e. of variance)</i>	
<i>Individual level</i>	0.980 (0.0047)
<i>Neighbourhood level</i>	0.046 (0.0058)

### C:20-24 yrs Women

<i>Exposure Variables</i>	<i>Model 3</i>
<i>Neighbourhood Adult Smoking (NAS)</i> <i>Neighbourhood Adult Smoking (NAS)</i>	2.05 (1.63-2.60)
<i>NAS*Neighbourhood Fragmentation</i>	
<i>NAS*NF Dec 1 (least fragmented)</i>	0.94 (0.60-1.49)
<i>NAS*NF Dec 2,3</i>	1
<i>NAS*NF Dec 4,5 (Ref)</i>	1.03 (0.77-1.36)
<i>NAS*NF Dec 6,7</i>	1.08 (0.83-1.40)
<i>NAS*NF Dec 8,9</i>	1.18 (0.92-1.52)
<i>NAS*NF Dec 10 ( most fragmented)</i>	1.43 (1.07-1.90)
<i>Random variance (s.e. of variance)</i>	
<i>Individual level</i>	0.990 (0.0044)
<i>Neighbourhood level</i>	0.044 (0.0045)

### D:20-24 yrs Men

<i>Exposure Variables</i>	<i>Model 3</i>
<i>Neighbourhood Adult Smoking (NAS)</i> <i>Neighbourhood Adult Smoking (NAS)</i>	1.40 (1.12-1.76)
<i>NAS*NeighFrag</i>	
<i>NAS*NF Dec 1 (least fragmented)</i>	1.17 (0.75-1.80)
<i>NAS*NF Dec 2,3</i>	1
<i>NAS*NF Dec 4,5 (Ref)</i>	1.26 (0.96-1.66)
<i>NAS*NF Dec 6,7</i>	1.17 (0.91-1.51)
<i>NAS*NF Dec 8,9</i>	1.22 (0.95-1.55)
<i>NAS*NF Dec 10 ( most fragmented)</i>	1.64 (1.25-2.17)
<i>Random variance (s.e. of variance)</i>	
<i>Individual level</i>	0.990 (0.0046)
<i>Neighbourhood level</i>	0.033 (0.0040)

Table 7:9 Effect Modification of NAS by NeighFrag; Comparing Odds Ratios From Stratified and Pooled Analyses

<i>Sub Groups</i>	<i>Stratified Method</i>		<i>Statistical Interaction Method</i>		<i>Back Calculation</i>
	<i>Col 1</i>	<i>Col 2</i>	<i>Col 3</i>	<i>Col 4</i>	<i>Col 5</i>
	<i>NAS OR NF decile 2,3 Strata</i>	<i>NAS OR NF decile 10 Strata</i>	<i>NAS*NeighFrag 2,3 OR (reference group)</i>	<i>Interaction term multiplier NAS*NF10</i>	<i>Reference group OR (col. 3) x interaction OR (col. 4)</i>
<i>Female</i>					
<b>15-19</b>	2.06	3.42	2.21	1.44	3.18
<b>20-24</b>	2.31	3.48	2.05	1.43	2.93
<i>Male</i>					
<b>15-19</b>	1.82	2.36	1.87	1.40	2.62
<b>20-24</b>	1.28	2.75	1.40	1.64	2.30

The statistical interaction confirmed that there were stronger associations of NAS with individual smoking in NeighFrag 10 compared to NeighFrag 2,3. There were some differences in the magnitude of the OR across the two methods. This was not unexpected as the estimates calculated from the statistical method used the whole age and sex group population rather than the smaller population within each stratum. For example, in all but the 15-19 year old men, the OR's obtained by the statistical method are smaller than those obtained by the stratified method. The differences between the two methods may have been due to the effect of the confounders simultaneously included in the equation which were also modified by NeighFrag, and therefore have had an effect on the OR's observed for NAS. Furthermore the differences between the two methods in the OR's reported were within the confidence intervals observed in the stratified analyses. Most importantly, the two methods produced OR's that were of similar magnitude and direction.

## 7.4.5 Discussion

### 7.4.5.1 Results Summary

Firstly, a substantial cross-level main effect of NeighFrag on the individual risk of smoking in the youth population was observed in the data. An association between the neighbourhood level exposure and individual level outcome remained after simultaneously controlling for individual and neighbourhood level confounders. The magnitude and direction of the association was modified by sex and age groups. The association for 15-19 year olds tended to show an adverse effect, in keeping with the 'social deficit' hypothesis. However a contrasting protective pattern was observed in the NeighFrag estimates for 20-24 year olds.

In the second set of analyses examining the transmission mechanism, there was empirical support for within level effect modification of NAS and NeighFrag. The association between NAS and the individual risk of smoking in youth was strongly modified in NF10, compared with all of the lower levels of fragmentation. This was seen in the heterogeneity of the associations when the NAS/smoking association was observed by strata and confirmed in the statistical tests of interaction. However, the direction of the modification was different to that hypothesised. In the most fragmented decile of neighbourhoods, the effect (measured by OR's) of the neighbourhood adult smoking environment was considerably than that observed in less fragmented neighbourhoods. Thus, the empirical evidence was contradictory to the hypothesised transmission mechanism.

### 7.4.5.2 Sources of Error

In order to assess the robustness of the associations reported above two important measurement factors need to be considered, random error and systematic error.

#### 7.4.5.2.1 Precision (random error)

As discussed above, the emphasis in the analyses has been on interpreting the pattern of the estimates rather than the single odds ratios in isolation from each



other. However in order to assess the robustness of the pattern of association it was still necessary to consider the role of chance for each OR. It should of course be kept in mind that there was a one in twenty chance that the 'true' estimate was outside of the intervals.

The width of confidence intervals increases as sample size decreases (Kirkwood et al., 2003). Therefore a particular concern here was the uneven sample sizes across NeighFrag decile groups, especially after the target population was restricted to urban dwellers. The width of the confidence intervals for NF1 reflected the smaller sample size. For example, in the case of 15-19 year old women, the model 3 OR confidence intervals for NF8,9 (the largest decile strata) were 0.90 – 1.05, considerably smaller than the 0.74 – 0.99 for NF1.

The robustness of the estimates for the smallest sized group, NF1, was placed under more pressure from the co linearity between NZDep and NeighFrag. Cross tabulations of NeighFrag and NZDep revealed sparse data problems in the most deprived, least fragmented cells. Importantly however, it should be noted that the width of the confidence intervals for NF1 did not increase substantially once NZDep was added into the regression model. In the same sub-group the OR confidence intervals for NF1 in model 2 were 0.58 – 0.80, compared to only a small change in model 3, 0.74 – 0.99.

Another check of the robustness of the fully adjusted NF1 OR's was the comparison of the crude and adjusted OR's (Rothman and Greenland, 1998). In 15-19 year old women, the baseline OR's from model 1 were comparable in size to the crude OR's, (with the exception noted above of NF10). The model 3 OR's were all attenuated when compared to the crude OR's, as would be expected once the confounders were simultaneously added. But the change in OR's between models for NF1 between the models was in keeping with the change observed across the models at other levels. Therefore it did not appear that the difference between the crude and adjusted OR's

could be attributed to the model assumptions being violated by co linearity or sparse data.

As discussed above, the inclusion of NZDep in the regression models was critical to the analyses, statistical assumptions notwithstanding. Therefore the interpretation of the NF estimates in particular have reflected the conservative approach recommended (Vittinghoff and McCulloch, 2007). It is also important to remember that the regressions are seeking to obtain estimates that illuminate theoretical pathways, rather than precisely estimating a single risk factor. With this in mind, the focus in the text below and in the discussion chapter has been based on the assumption that the results were unlikely to have been largely due to random error.

#### 7.4.5.2.2 Systematic Error

##### *Bias*

Bias, the distortion of the association due to errors in study design, can decrease the internal validity of an association (Rothman and Greenland, 1998). Associations can be biased because of how participants are selected into a study (selection bias); how information is used to classify participants by exposure, outcomes and covariates (information bias); and unmeasured or mismeasured confounding. The potential of each of these sources of distorting effects will be addressed in the above order.

Selection bias can occur if the relationship between the exposure and outcome is different between participants and non-participants. This could have occurred in the smoking analyses if sectors of the population did not participate equally in the census. Because the New Zealand census aims to include information from the whole population rather than a sample, the survey design therefore seeks to reduce selection bias as much as possible. Post enumeration studies of the 1996 census indicated that while there were some differences, non-participation was minimal and therefore unlikely to be a major contributor to biased estimates (Ewing, 1997).

Bias could also have been introduced with the restriction process used to create the study population. The youth study population only included participants with complete smoking and covariate data. Selection bias could therefore have been introduced if those that were not included because of missing outcome or covariate data had a different NeighFrag/smoking relationship than youth with complete data. However, the percentage of incomplete data ranges from 5-8% across NeighFrag deciles, suggesting that the biasing effect of omission would be minimal.

Information bias can result from participants being wrongly classified by exposure, outcome or covariate variables. Misclassification can occur randomly across variables, that is, non-differentially, with the probable effect of reducing the effect towards the null. Harder to interpret is differential misclassification where the error is systematic, for example, occurring more frequently in one extreme of fragmentation.

Misclassification could have occurred in a number of ways in the smoking analyses reported here. Participants' exposure could be misclassified if their recorded area of residence was not their actual residential neighbourhood. It may be that participants who were relatively mobile, or who lived in non-private dwellings recorded their address in another neighbourhood. Alternatively, a participant may have been included in the study if they were present at the time of census and incorrectly classified as a usual resident there. This would have meant that their current neighbourhood exposure would be misclassified. If participants were equally likely to have their exposure misclassified by the smoking outcome the probable effect would have been to bias the results towards the null. If, on the other hand, misclassification was more likely to happen for smokers (for example) then differential bias would have occurred.

Information bias can also occur with the outcome variable, where participants' smoking status is differently recorded systematically by NeighFrag. In order for bias to occur, the reporting of smoking status would firstly need to be recorded

differently by NeighFrag. There is little reason to suggest that either sources of differential misclassification would have distorted the associations in the census data. That is, there was no obvious reason why participants in NF10 were more likely to record themselves as non smokers than those in NF1, or why smokers were more likely to record themselves as living in a more or less fragmented neighbourhood than their current exposure. While the NeighFrag were created from the same dataset as the outcome variable, there was no reason to suggest that youths' likelihood of classifying their smoking status would have affected their responses to the NeighFrag component variables (and potentially misclassifying the exposure).

Mismeasurement of the confounders was another potential source of information bias. The confounders used in the analyses were recognised measures of known socioeconomic and demographic risk factors and it can therefore be reasonably assumed that this error would have been minimised. However there was good reason to suggest that the covariates used may have meant different things for each sub group, most notably for the education and labour force variables. This was a more substantive research issue rather than misclassification and has been treated as such in the presentation of the results.

### *Confounding*

Error in the estimation of the association can occur if confounding factors are not accounted for. These analyses were adjusted for notable socio-demographic factors at the individual and neighbourhood level that were established as confounders to the hypothesised relationship. It is possible however, that other important factors were omitted, leading to unmeasured confounding (Fewell et al., 2007).

As discussed above, one of the challenges faced by selecting the 15-24 year olds as the target population was the difficulty in selecting and measuring meaningful socio-demographic confounders. It could well be that the confounders used were not adequate for each age and sex group. Of note was the difference in the magnitude of the OR's of the age and sex groups for variables such as education. While education

met the properties of a confounder, the measure used here (highest qualification) did not act as a confounder for it appeared to mean different things (for men in particular) and its inclusion in the model may therefore have led to biased estimates. At the same time, other more relevant factors to do with education would have been omitted, such as years of schooling.

## 7.5 Conclusion

The relationship between smoking and NeighFrag has been assessed in two ways, reflecting the two hypothesised mechanisms. The analyses in the first section of the chapter tested the 'integration and regulation' mechanism, asking if there was evidence of NeighFrag acting as a proxy measure of a neighbourhood 'social deficit' in some way. NeighFrag was found to have a cross-level main effect association with the individual risk of smoking. The association was weakened after adjustment for individual and neighbourhood level confounders, and was modified across the sex and age groups. While the adverse association observed for 15-19 year olds suggested support for the 'social deficit' hypothesis the contrasting pattern for 20-24 year olds suggested a more protective association, contradicting of the hypothesis.

The second set of analyses examined the transmission mechanism, asking if there was evidence of lower levels in the transmission of smoking practices within highly fragmented neighbourhoods. The results from the stratified and pooled analyses suggested that this was the case for the most fragmented neighbourhoods compared with other levels. However, the direction of the modification was contradictory to the hypothesised transmission mechanism. That is, in the most fragmented neighbourhoods, the association of the neighbourhood adult smoking environment with youth smoking was stronger, rather than weaker.

The emphasis in the interpretation of the analyses has been on the patterns of associations. This was felt to be the more appropriate and informative approach, given the exploratory nature of the main effect analyses in particular. The selection

and measurement of the confounders for the 15-24 year old age group proved problematic and it is probable that the associations in both analyses may be (partly) biased due to residual confounding. It is likely that other factors may be more important predictors, and also potential confounders to the NeighFrag/smoking relationship. A more substantive interpretation of the results and possible explanations is undertaken in chapter eight, focusing on the external validity of the associations, along with a discussion of the empirical support for the theory.



## **Chapter 8**

### **Bringing Data and Theory Together - What do we Now Know?**

Neighbourhood level social fragmentation has been found to be associated with mental health, and to a lesser extent, smoking in New Zealand. Associations between neighbourhood measures of social fragmentation and health outcomes remained after controlling for available, known individual and neighbourhood confounders. Questions remain about the external validity of the associations. What inferences can be made from the associations about the relationship between where we live and our health? The discussion below brings together the theory and data developed over the course of the thesis in three steps: a theoretically grounded measure, a theory of how that measure might be related to health, and analyses designed to empirically test the hypothesized mechanisms. The results from each step are critiqued, asking if they have been able to shed more light on the complex relationship between where we live and our health.

To recap the steps briefly, the measurement of the construct was grounded in a theoretical model of neighbourhood collective social functioning, presented in chapter three. A weighted index was created from census data. It was hypothesized that the Index of Neighbourhood Social Fragmentation (NeighFrag) was measuring the compositional structural antecedents to the social life of the neighbourhood. It was argued that geographical areas with a highly fragmented composition would have more difficulty establishing and maintaining strong collective social functioning.

Understanding why NeighFrag might be related to health was developed by examining Durkheim's thesis on social groups, and discussed in Chapter Four. NeighFrag was theorized as capturing types of neighbourhood level social groups with different levels of social integration and regulation. It was thought that this



could be important for health in two ways, summarized as the “social integration and regulation” mechanism (whereby the wellbeing of individuals benefits from a well balanced type of social group) and “transmission” mechanism (that health-related practices are more or less easily transmitted in different types of social groups).

The empirical means of analyzing the two mechanisms was developed in chapter five. Using a social epidemiological framework, multilevel regressions were designed to explicitly test specified cross-level pathways related to each mechanism. Chapters six and seven detail the analyses of mental health and smoking outcomes with NeighFrag. Both outcomes were found to be associated with NeighFrag in various ways. Matters of precision and internal validity have been addressed in the discussions following each analysis chapter. Both chapters concluded that unmeasured confounding remained a threat to the validity of the observed associations.

The question remains as to what degree the associations give support for the theory, that is, are they internally and externally valid. That is, are the proposed mechanisms generalizable to, for example, other health outcomes? The following questions will be asked of the theory and data: Are there alternative explanations for the observed associations, such as unmeasured confounding? Does the empirical evidence support the interpretation of the index as capturing different types of neighbourhood level social groups? Do the associations support the theory that the “social integration and regulation” and “transmission” mechanisms of neighbourhoods would have specific effects on health? Finally, are other mechanisms suggested by the data?

## **8.1 Index of Neighbourhood Social Fragmentation - an Illuminating Measure?**

The first challenge faced in the development of this thesis was the construction of a neighbourhood measure of social fragmentation. The measurement of neighbourhood properties has posed challenges for the research field (Kawachi and

Subramanian, 2007). The review of the social fragmentation/health literature established that published definitions of social fragmentation were limited, as was discussion on why fragmentation would be an important factor for health. Rather than relying on “off the shelf” definitions and measures of “social fragmentation”, this thesis has therefore sought to contribute to the field by developing a theoretically grounded empirical measure of the neighbourhood social environment (Macintyre et al., 2002).

The task of creating a measure of social fragmentation at the neighbourhood level began with the development of a conceptual model of neighbourhood social functioning. Based on research investigating the antecedents to collective social functioning of neighbourhoods, three interlinking domains described the structural antecedents to the social functioning of the neighbourhood: the means of sharing norms and values across the neighbourhood, social resources required for establishing and maintaining networks, and attachment to people and place. Factors which inhibited the functioning in the domains were considered to be fragmenting characteristics: for example, high mobility into and out of a neighbourhood was considered a fragmenting characteristic because of the impact on attachment processes within the neighbourhood. In order to better represent the ‘whole’ that was fragmented, the index was renamed the Index of Neighbourhood Social Fragmentation, and abbreviated to NeighFrag.

Operationalisation of the index was dependent on individual level census data, an acknowledged weakness of the index. Finding variables to measure the three domains highlighted the difficulties faced by researchers when using individual level administrative data to measure a neighbourhood level social property. However, the domains proved to be a valuable means of ensuring that each variable included had to earn its place. It also forced me to think how commonly used variables such as residential mobility might contribute to the wider social fragmentation construct.

The development of the index (by a team including myself) was both theoretically and statistically rigorous, enhancing its utility as a valid research tool (Frohlich et al., 2007). Rather than rely on the collection of selected variables, Factor Analysis and Principal Components Analysis were used to select variables from a larger set and assign weights for creating a single score. Nine variables were found to be statistically related, and appeared to be capturing a single latent construct. A version of the Congdon index was also created using the same methods as a useful comparison. Thus the variables in the index were retained for their theoretical contribution and on the basis of the statistical relationships between them.

Further understanding of the index and the construct of collective social functioning was developed by examining the validity of the index. The NeighFrag index demonstrated expected relationships with other neighbourhood exposures. It was moderately correlated with NZDep, suggesting that the index had been successful at capturing a different construct from deprivation, in keeping with Congdon's definition of social fragmentation (Congdon, 2004a). A moderate inverse correlation was found with a neighbourhood volunteering measure, SoCInd, suggesting that while the index may have been related to the social capital proxy, it was not simply the inverse. Other analyses demonstrated that the index predicted variation in the demographic composition of neighbourhoods, suggesting that NeighFrag was capturing non-random variation in neighbourhoods. For example, the age structure of highly fragmented neighbourhoods was weighted towards early adulthood, with fewer children and older residents than the composition of less fragmented neighbourhoods.

An important factor that was not able to be assessed was the causal relationship between NeighFrag and collective social functioning in the neighbourhood. While NeighFrag was framed as a structural antecedent to social life of neighbourhoods it would not be possible to establish causal direction from cross-sectional data. On the other hand, it is likely that a more recursive relationship may exist between, for

example, an area's reputation as a highly mobile neighbourhood and the composition of that neighborhood.

It was argued in the discussion sections of Chapters Six and Seven that NZDep could potentially be a mediating rather than confounding factor if NeighFrag were a contributor to the socioeconomic composition of a neighbourhood. Again, an historical approach would be better placed to examine the role of area deprivation on fragmentation processes.

The index was also unable to capture other structural antecedents to neighbourhood social life. Strictly speaking then, the index should be seen as measuring the compositional structural antecedents of collective social functioning. While population composition may be important, geographical factors, local personalities, events and so forth have all been recognized as being critical to understanding how communities function (Cattell, 2001, Baum and Palmer, 2002, Stephens, 2008). For example, Witten et al (2007, , 2001) have demonstrated the importance of the school as institutional antecedent for neighbourhood social life. It would be interesting to examine where composition fits in the cycle that leads to a neighbourhood's current status.

The index development successfully demonstrated the value of using theory to drive the measure. Individual level administrative data was able to be used to logically operationalize the concept (Frohlich et al., 2007). The rigorous process of developing the index and then examining its validity and reliability provided increased confidence that something important about neighbourhoods in New Zealand had been captured by the index. The next question was therefore: why and how would it matter for health?

## **8.2 Theory**

Chapter Four addressed this question in detail. A key purpose of the exercise described in the chapter was to address criticisms in the neighbourhoods and health

field by moving away from a 'risk factor' approach and specifying why and how the index might conceptually be related to health outcomes (Galea and Ahern, 2006). It was unclear to me exactly why the particular composition of the neighbourhood captured would predict health status. These two drivers fostered a strong theoretically based definition of the underlying construct, and why and how it might be important for health, meeting the sustained and concerted call from the Neighbourhoods and Health literature for clearer conceptual understanding to drive analyses (Frohlich et al., 2007, Frohlich et al., 2004, Galea and Ahern, 2006, Mujahid et al., 2007, Roosa et al., 2003, Carpiano and Daley, 2006a).

From examination of Durkheim's original work on social groups it was concluded that geographical neighbourhoods could plausibly be considered as an intermediary social group between the wider world and the individual (Berger and Neuhaus, 2002). Social groups, in Durkheimian terms, were established as sites where the needs of the collective and the individual were balanced (Durkheim, 1964). The neighbourhood-based social group would therefore act as a source of integration and regulation in Durkheimian terms or, calling upon Bourdieu's interpretation, a source of social cohesion or capital. As discussed earlier in Chapter Three, the NeighFrag index was not able to measure actual neighbourhood levels of cohesion or capital, or the social ties within a neighbourhood, but potentially provided the antecedent conditions and social ties for their creation.

As a result, NeighFrag was framed as a proxy for different types of neighbourhoods that represented different types of social groups, with potential consequences for health. Two mechanisms were suggested from Durkheim's theories of social groups. Firstly, different types of neighbourhoods could offer residents varying levels of social integration and regulation. Both extremes of fragmentation may be more stressful because of imbalanced levels of integration and regulation. Secondly, NeighFrag could represent varying ability of health-related practices to be transmitted within a neighbourhood. In a highly fragmented neighbourhood, other

residents' practices may have little influence on ones own behaviour, whereas in a less fragmented environment, neighbours practices could be highly influential.

The above reading of Durkheim's thesis had important implications for understanding how 'social fragmentation' at the neighbourhood level might be understood. Rather than treating it as a lack in some way, as with deprivation or disorganisation, the index represented types of neighbourhood social groups, where the implications for health were then dependent on what was being sought from the social group. In other words, the establishment of a theoretical model of neighbourhood level social fragmentation was an important result of the thesis and makes a significant contribution to the literature by showing the benefits of this approach.

The two mechanisms provided a useful lens through which to question the research on the dynamic between individuals and society. Could neighbourhood level social groups act as an important source of social integration and regulation for residents? Was the neighbourhood social group a site where health-related social practices were practised and shared, and therefore transmitted?

While the role of the geographic neighbourhood is much disputed, there is increasing evidence that it can be an important social setting. The role of the neighbourhood as a source of social resources and as a site where health-related practices are enacted was supported in the literature. Research has also suggested that individual characteristics, as well as other neighbourhood characteristics, are clearly an important part of understanding how neighbourhoods might matter for health. The literature suggested that a number of outcomes available to me could provide useful tests of the mechanisms. The mental health and smoking outcomes supported by the literature were then used as tests of the mechanisms.

### 8.3 Epidemiological Exploration and Interpretation

The mechanisms were successfully tested in the thesis using carefully designed empirical analyses. Employing a social epidemiological methodology, a set of analyses was developed to test hypothesized pathways of the two mechanisms. Multilevel regressions were used to test specific hypotheses for each pathway, examining both main effects and effect modification. The degree to which the analyses provide empirical support for the mechanisms is discussed in detail below. Systematically developing the analyses to test specified pathways has provided a valuable means of evaluating evidence for a causal effect. Using a variety of analyses examining a range of pathways with different outcomes and indices had benefits and drawbacks. A single outcome, pathway, or index might have allowed time to more closely examine matters around statistical precision and assumptions. It also could have been an opportunity to examine alternative statistical methods, for example: ecological analyses.

But an important advantage of examining a range of pathways, datasets, and outcomes was the ability to explore the contrasts between analyses. The contrasts in findings have provided valuable evidence in themselves. Making interpretations from a set of analyses has made me feel more confident that the associations testing the mechanisms were less likely to be due to chance. Therefore I would have more confidence that the empirical association of NeighFrag with health observed here might be generalizable to other health outcomes – if they were considered to be responsive to a specific mechanism.

This was particularly evident when the smoking analyses were being developed. As discussed above, there was no clear indication in the neighbourhood-level social fragmentation literature as to why smoking would be related to neighbourhood fragmentation. But there is good evidence in the wider health literature to suggest that smoking was a health-related social practice (Frohlich et al., 2002, Turner et al., 2006). Regarding the neighbourhood as a social group where social practices are

constructed and practiced allowed smoking to be seen as an individual social practice that would be sensitive to the practices of other residents.

The analyses from Chapters Six and Seven were intended to provide empirical tests of the hypothetical mechanisms. In order to establish whether they have provided support for an effect of neighbourhood fragmentation on individual health, and whether causal inferences can be made, the associations need to be examined further. Bhopal (2002) highlighted the need to carefully consider a number of factors to shift from “the probably not causal association to possibly causal one” (p.122). Questions of magnitude, dose response, and specificity (for mental health analyses) (Bhopal, 2002) have been addressed in the analyses chapters. Emphasizing the pattern of association when discussing the regression coefficients promoted a focus on the magnitude and dose response first and precision second.

The consistency of the associations across sub groups (Bhopal, 2002) has been specifically tested by looking for expected heterogeneity, and will be discussed further below. Experimental change is generally difficult to observe in neighbourhoods research and not possible here (but the use of natural experiments (Petticrew et al., 2005) could be an interesting avenue, for example looking at the role of neighbourhood social properties and natural disasters).

The remaining issues are temporality and biological plausibility (Bhopal, 2002). Because cross-sectional data has been used there is no means of assessing whether the exposure preceded the outcomes. However, as discussed a number of times above, the processes of selection and endogeneity are critical to neighbourhoods research. Issues of temporality are therefore discussed in detail below. The biological (or psychological) plausibility of the relationship between NeighFrag and health outcomes has been the basis for designing the analyses: this final criterion is therefore an important focus of the discussion below, asking if the data provides plausible support for the theory.



As well as the criteria for causal inference, the precision and validity of the associations need to be considered (Rothman and Greenland, 1998). The first question that has been considered here is the role of chance and systematic error. In other words, how certain can I be that the observed associations are likely to be a consequence of an effect of NeighFrag rather than error? Issues around precision and internal validity have primarily been addressed in the discussion following each analysis and are summarized below.

The major focus in the discussion below turns to the generalisability and external validity of the associations. External validity commonly refers to the extent to which a finding in one study can be generalized to whole or other populations. While that was certainly of interest here, the larger concern was if the observed relationships offered support for the theoretical hypotheses (Rothman and Greenland, 1998). The two are not unrelated. The purpose in developing hypotheses about the neighbourhood/health relationship was so that population level processes could be examined and understood. I would argue that there would be less value in asking, for example, could neighbourhood fragmentation modify the effect of neighbourhood smoking norms in other populations. Rather, the more relevant and enlightening questions were about the mechanisms: for example how plausible was it that the type of neighbourhood social group modified the transmission of smoking practices within neighbourhoods? What would then be generalizable would be the mechanism, not just the association.

The discussion will therefore concentrate on exploring alternative explanations for the observed associations between the neighbourhood fragmentation and health as a means of examining the causal evidence. Two possibilities are focused on. Firstly, unmeasured confounders may better explain the observed associations and either support or contradict the hypothesized mechanisms. A second alternative that will be explored is the differences in the measurement of the construct. The epidemiological confounding triangle (discussed in Chapter Five) will be used to examine possible alternative explanations.

As noted above, a key 'threat' to the validity of the associations is unmeasured confounding. In order to make causal inferences about the effect of an exposure on an outcome, ideally a person exposed to one level of an exposure would be equivalent or perfectly exchangeable, with a person exposed at another level (Rothman and Greenland, 1998). If this were the case then any change in the outcome between levels of the exposure could be attributed to their exposure. Regression analyses statistically attempt to approximate exchangeability. Adding variables which measure known confounders is a standard statistical means of 'making' observations more exchangeable. However there will always be questions over the extent to which regression analyses can actually achieve this, particularly once life course processes are taken into account.

In part this is due to the difficulties in reliably and validly measuring the complex confounding factors. Issues of measurement of the known confounders used have been discussed in relation to each chapter, and the acknowledged limitations of the mismeasurement have meant that the associations need to be interpreted conservatively. On the whole, though, the methods used here reflect reasonably standard techniques: the results need to be considered against the wider body of empirical evidence. More challenging (but to my mind informative) are the difficulties faced when there are possible unknown, or immeasurable, confounders. This will be the focus of the discussion below.

Adjusting for confounding to increase exchangeability becomes more difficult, and some say insurmountable (Oakes, 2004), when dealing with neighbourhood level exposures. Is a teenage smoker living in a highly fragmented neighbourhood actually exchangeable with a teenage smoker in a less fragmented neighbourhood? If there are any differences, the possibility of unmeasured confounding needs to be addressed. Of course, only factors which are also associated with the outcome need to be considered and even then only ones that are not on a mediating pathway. Nevertheless unmeasured confounding remains one of the real challenges faced by neighbourhoods' research.

One of the very important factors that may be confounding the relationship is the combination of processes that lead to residence in a particular neighbourhood at a given stage in people's lives. As discussed in the review of the Neighbourhoods and Health research field, endogenous processes such as migration and health selection have become of substantive interest in recent times (Frank et al., 2007, Kawachi and Subramanian, 2007, Smith and Easterlow, 2005), although they are difficult to examine in cross-sectional secondary data such as here. The results from initial explorations of the NeighFrag index have suggested that people do not necessarily stay in one type of neighbourhood over their life course (otherwise where would all those younger adults in highly fragmented neighbourhoods come from and go to?). Therefore we need to consider how migration between types of neighbourhoods might be an important part of the explanation. To what extent could the associations be attributed to endogeneity? Can the associations be better explained by examining the relationship between the 'sorting and sifting' of people into neighbourhood types and the mental health and smoking outcomes?

### **8.3.1 Social Integration and Regulation**

Based on my reading of Durkheim's 'social group' framework, different types of neighbourhoods (as represented by NeighFrag) could offer varying levels of social support to residents. A highly fragmented neighbourhood would be more likely to provide a paucity of integration and regulation for individuals, contributing to a more stressful environment, perhaps by offering less social support (Andren and Rosenqvist, 1987). On the other hand, if a less fragmented neighbourhood had an excess of integration and regulation this might potentially contribute to stress levels, as the individual could feel overwhelmed by the strong collective nature of the social group.

The social integration/regulation mechanism was empirically tested using self-reported mental health and smoking outcomes. The mental health analyses offered the most scope for testing the mechanism with good sample size, and a good outcome measure. Unfortunately the mental health outcome used (SF36 scale) is not

one that has been used commonly in association with social fragmentation so there was less opportunity to directly compare evidence for other studies. However, there was support for its use in the theoretical model developed here and in the related social cohesion literature. In particular, an argument was developed from Durkheim's work for a direct effect of fragmentation on mental health (as opposed to illness).

The mental health analyses also provided the chance to observe whether the association of NeighFrag was more specific to mental health outcomes than, for example, physical health outcomes, again in support of my interpretation from a Durkheimian theoretical framework. The results observed here generally supported the specificity of association observed elsewhere (Stjarne et al., 2004, Smith et al., 2001). There was less support in the analyses for a relationship between NeighFrag and health measures that captured physical health constructs such as pain.

The smoking analyses were more exploratory and cannot therefore be directly compared to other social fragmentation studies. They have, however, provided further evidence of the potential effect of the neighborhood as a social setting for health-related practices.

#### ***8.3.1.1 Mental Health***

Neighbourhood fragmentation was inversely associated with self-reported mental health in the adult, urban population, as hypothesized. Poorer mental health in individuals, measured by the SF36 mental health scale, was predicted by increasing levels of fragmentation, indicating a cross-level effect. The association remained robust when controlling for known individual confounders, allowing for the non-independence of the individual observations, and when neighbourhood deprivation was included in the model.

The magnitude of the estimates and their linear nature is consistent with a causal effect (Bhopal, 2002). The two point difference for the NF1 and NF10 estimates compared to moderate fragmentation, and the three point difference between most

and least fragmentation were sufficiently large to detect population level differences (Ware et al., 2000). While the NF1 estimates were problematic to interpret reliably due to sparse data the pattern of the estimates, nor the comparison with the NeighFrag(continuous) estimate, did not contradict a dose response.

The association was modified by individual factors, indicating cross-level effect modification. When the study population was stratified into men and women an association was only observed for women; there was no empirical evidence of an association of NeighFrag with mental health for men. Statistical confirmation of this interaction was gained from pooled analyses using an interaction.

The associations support the theoretical hypothesis that types of neighbourhood vary in their levels of integration and regulation for their residents, with consequences for health. The evidence suggests that urban women living in neighbourhoods with increasing levels of fragmentation are exposed to lower levels of social integration and regulation, with adverse consequences for mental health.

The difference in the association by gender has two potential explanations. It could be argued that if neighbourhoods were social groups it would be expected that an association be present for the whole population. If so, a failure to find an association for men could weaken the evidence for the hypothesis that neighbourhoods act as social groups, and therefore as sources of integration and regulation.

On the other hand, the differential could also be argued as supportive by showing specificity in effect. Rather than assuming that the whole population is affected in the same way, the difference may reveal important ways in which men and women source social support. It may be that while men also benefit from integration and regulation in social groups, the neighbourhood is not as significant a source as it is for women.