10 Reconsidering risk: Implications and future directions

The hypothesis that low birthweight and disrupted patterns of childhood growth increase risk of diabetes in an Aboriginal community has been tested and several themes of adult risk have been explored. The key findings from the present study are discussed and directions for further research are presented in this chapter.

10.1. Key findings

10.1.1. Diabetes prevalence and risk factors
Diagnosed diabetes prevalence within the Cherbourg community is approximately 20% among people aged over 18 years. If those with undiagnosed diabetes – or at the very least a significantly increased risk of diabetes (based on abnormal FBSL and clusters of risk factors) – are included, diabetes rates soar to 38.5% among women and 42.0% among men. For every diagnosed person in Cherbourg, there is likely to be someone with undiagnosed diabetes, which is similar to estimates for Australia as a whole. This high prevalence of undiagnosed diabetes persists despite routine hospital screening.

The prevalence of diabetes risk factors among the random sample of people who had never been diagnosed with diabetes is extremely high. Abnormal fasting glucose was recorded for 35% of women and 50% of men, obesity for 39% of women and 24% of men, central obesity for 73% of women and 29% of men, systolic hypertension for 11% of women and 27% of men, and diastolic hypertension for 8% of women and 35% of men. Forty percent of women and 25% of men had three or more independent diabetes risk factors (other than abnormal fasting glucose).

Do risk factor thresholds adequately reflect diabetes risk?
Conventional thresholds for diabetes risk may, however, be inappropriate for this population. Waist circumference increases linearly with BMI among never-diagnosed females and males (Figures 10.1 and 10.2), as might be expected. Only participants who had never been diagnosed were included in these analyses to reduce the likelihood of complications from insulin resistance causing fat to be centrally deposited, in addition to central adiposity promoting insulin resistance (Section 2.4.4). Furthermore, removing those with diagnosed diabetes from the
analyses reduces the chance of introducing confounds caused by medication, such as that to reduce blood sugar, improve insulin sensitivity or promote weight loss.

Even at BMIs that are generally considered underweight (<20), some women in Cherbourg have waist circumferences that are above the threshold for central obesity (Figure 10.1), indicating a pattern of centrally distributed fat among those of normal and less than normal bodyweight. This strongly suggests that the conventional definitions of overweight and obese are inappropriate for women in Cherbourg, given the relationships between waist circumference and other diabetes risk factors (Section 6.2.1). Furthermore, increased abdominal visceral fat indicates insulin resistance, and acts to increases risk of CVD through the release of FFAs (Section 2.4.4). If central obesity is correlated with diabetes risk, which it clearly is (see below), then the threshold for when BMI becomes ‘obese’ should reflect this risk. Under the conventional definition of obesity this is not the case for women in Cherbourg.

Figure 10.1. Scatter plot of waist circumference and BMI (never-diagnosed females) showing linear regression line (mean and 95% confidence interval). Horizontal reference line is set at 88cm for waist circumference, the threshold for female central obesity used in the present study. The vertical reference lines enclose the BMI range where presence of central obesity begins and where absence finishes (17.5 to 24).
Above a BMI of 25 kg/m², the conventional threshold for overweight, all randomly sampled women in the present study are centrally obese (Figure 10.1) and therefore at increased diabetes and CVD risk. Studies in other populations have also found a greater propensity for central patterned fat at lower than expected BMIs, for example among Aboriginal women (O’Dea et al. 1990; Rowley et al. 1997; Daniel et al. 2002) and in urban females and males in India (Yajnik 2002), where it was also noted that the non-obese are often centrally obese by accepted thresholds.

For males however, the BMI threshold of obesity at 30 kg/m² appears appropriate given the relationship between BMI and waist circumference and the range of where central obesity begins and absence of central obesity ends (Figure 10.2); the conventional range regarded as ‘overweight’ includes both those with and without central obesity.

![Figure 10.2. Scatter plot of waist circumference and BMI (never-diagnosed males) showing linear regression line (mean and 95% confidence interval). Horizontal reference line is set at 102 cm for waist circumference, the threshold for central obesity used in the present study. The vertical reference lines enclose the BMI range where presence of central obesity begins and where absence finishes (28 to 32).](image-url)
Would lowering the BMI threshold for obesity by 5kg/m\(^2\) more adequately reflect the health risks associated with diabetes among women? Waist circumference is positively and significantly associated with increasing blood pressure and FBSL among never-diagnosed women (linear regression: systolic p=0.018, diastolic p=0.002, FBSL p=0.003) (Figures 10.3 to 10.5). The waist circumference threshold of 88cm appears to be appropriate given that (with the exception of one woman with a high FBSL) the thresholds for blood pressure and FBSL are surpassed only by those who have a waist measurement above 88cm.

Figure 10.3. Scatter plot of systolic blood pressure and waist circumference (never-diagnosed females) showing linear regression line (mean and 95% confidence interval), p=0.018. Reference lines are at 88cm for waist and at 140mmHg for systolic pressure.
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Figure 10.4. Scatter plot of systolic blood pressure and waist circumference (never-diagnosed females) showing linear regression line (mean and 95% confidence interval), \( p=0.002 \). Reference lines are at 88cm for waist and at 90mmHg for diastolic pressure.

Figure 10.5. Scatter plot of FBSL and waist circumference (never-diagnosed females) showing linear regression line (mean and 95% confidence interval), \( p=0.003 \). Reference lines are at 88cm for waist and at 5.5mmol/l for BSL.
Among men the relationships appear to be more complex than among women. Despite an overall increase in both systolic and diastolic blood pressure with waist circumference, the only significant association is with FBSL (p=0.017), and even this association is fairly weak (Figure 10.6 to 10.9). High blood pressure and high FBSL are apparent at low waist circumferences, which may suggest that 102cm is too high as the definition of central obesity among this group of men, and a threshold of approximately 90cm may be more appropriate.

Figure 10.7. Scatter plot of systolic blood pressure and waist circumference (never-diagnosed males) 102cm for waist and at 140mmHg for systolic pressure.
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Figure 10.8. Scatter plot of diastolic blood pressure and waist circumference (never-diagnosed males) showing linear regression line (mean and 95% confidence interval), $p=0.275$. Reference lines are at 102cm for waist and at 90mmHg for diastolic pressure.

Figure 10.9. Scatter plot of FBSL and waist circumference (never-diagnosed males) showing linear regression line (mean and 95% confidence interval), $p=0.017$. Reference lines are at 102cm for waist and at 5.5mmol/l for FBSL.
Thus the widely accepted BMI threshold for obesity at 30kg/m\(^2\) for females may not adequately reflect the risks associated with waist circumference. This suggests that the threshold at which BMI is considered to become a risk should be lowered among women in this population.

If the thresholds for BMI as an independent diabetes risk factor were lowered for women from 30kg/m\(^2\) to 25kg/m\(^2\), the level generally accepted as overweight, the number of never-diagnosed women categorised as high-risk would increase from 15 to 22, or by nearly 50%. Predicted diabetes in Cherbourg among women, using the same criteria as in Section 6.3 to estimate undiagnosed diabetes, could increase to 47.7%, with a ratio of 1.5 undiagnosed cases to every diagnosed case, very similar to that for men. An even lower threshold is advocated by Daniel et al. (2002), who suggest that a BMI threshold \(\geq 22\)kg/m\(^2\) would more adequately reflect diabetes risk among Aboriginal populations. Similar proposals for population-specific obesity thresholds have been put forward for other populations; lowering the threshold has been proposed for Japanese (Kanazawa et al. 2002) and Chinese (Cockram 2000; Bei-Fan 2002; Lee et al. 2002) populations, for example.

Waist circumference is much more strongly and significantly associated with other risk factors among women than among men. Given the strong positive linear relationship between waist circumference and BMI, reducing obesity among women especially could have a substantial impact on diabetes risk by reducing central adiposity.

Among men, the threshold of where risk begins according to waist circumference is not as clearly discernible as it is for BMI among women. A waist circumference of 90cm may be more appropriate than 102cm, but the relationships with other diabetes risk factors should be clarified with further investigation.

10.1.2. Patterns of child growth

Birthweight and child growth patterns among Cherbourg children have changed little over the half-century of IHC records (Section 7.1). Relative to international references, Cherbourg children typically show normal or rapid growth in the first few postnatal months. This is typically followed by a marked and prolonged period of growth faltering between 6 and 15 months, with a subsequent period of rapid catch-up growth from 15 to 36 months, and sustained growth from 36 to 60 months during which average weight growth is close to the international reference. There is little difference between the median growth patterns of girls and boys, although weight growth of boys tends to be more variable. Infection is probably the primary
cause of faltering growth in Cherbourg, with poor nutrition and psychosocial stress as likely contributing factors.

10.1.3. Programming hypothesis

IUGR, as determined by lower than median birthweight and higher than median weight growth velocity to the first or third postnatal month, does not appear to be consistently associated with adult diabetes risk (Section 7.3.1).

Low birthweight and low weights during the five growth periods to 60 months were not associated with any measure of diabetes risk (Section 0). Birthweight was associated with adult BMI among men, but the association was positive, rather than negative as expected under programming. Similarly, high weights at some ages and relatively rapid weight gain during childhood were associated with some increases in risk, especially the likelihood of obesity among women. Some of this association may be explained by maternal diabetes causing higher birthweights, but such infants would be expected to grow more slowly (catch-down) postnatally; instead it is those who gain more rapidly who are at increased subsequent diabetes risk.

This does not mean that in utero and/or early postnatal programming has not occurred, but that a greater role is played by more rapid weight gain during childhood. This rapid weight gain may be a response to earlier growth faltering. That rapid weight gain relative to the median was associated with adult obesity supports the role that catch-up growth might play, at least among females, in predisposing some individuals to diabetes.

The absence of consistent birthweight and weight growth effects may be because birthweight is mostly adequate, and does therefore not appear to affect adult risk of diabetes. It may also be that what is observed is the result of at least two types of processes, maternal diabetes creating higher weight babies, while those growing more rapidly may be the product of (limited) IUGR, with the fairly small sample reducing the likelihood that a result should be found significant.

In addition, prenatal ‘programming’ could still occur due to maternal diet that may be of poor quality during early gestation, but adequate in later gestation, producing newborns whose weights may not reflect early gestational nutritional deprivation. Maternal nutritional stores may have been inadequate before pregnancy, especially in past decades due to poor quality rations and working conditions (Section 4.3), and more recently because of inadequate understandings of healthy eating and reduced access to healthy food (Section 8.1).
The small size of the sample available to be studied is also likely to have reduced the significance and consistency of the results. Depending on the adult risk factor, detectable differences were in some cases found to be too large to be practical given the small sample (Table 7.8) and the estimated sample size required varied between approximately 50 to over 2000 participants, depending on the risk factor and exposure under analysis.

Unfortunately it was not possible for the most part to achieve the sample size necessary to demonstrate statistical significance, given the size of the community and the small differences found between groups. In similar future studies in Aboriginal communities, if statistical significance is sought it might be appropriate to expand the population to be sampled to include other communities. For example, increasing participant numbers to 1000 would have been sufficient to detect nearly all significant differences sought (Tables 7.9 and 8.14).

Even if associations had been found to be both consistent and strong, a genetic basis for patterns of growth, propensity towards central obesity and diabetes cannot be ruled out. Why, for example, do Aboriginal women have a tendency towards central obesity even at low BMIs? This could be due to a genetic predisposition for depositing fat more centrally, or it could be an outcome of rapid child weight gain – which could itself have its basis in either childhood environment or specific genes, or perhaps a combination. Furthermore, patterns of growth tend to run within families, and are particularly dependent on maternal growth (Alsop-Shields and Dugdale 1995). Although growth, to a degree, is therefore an inherited characteristic, inheritance may not depend on shared genes.

The relative homogeneity of exposure to risk factors that contribute to IUGR and reduced postnatal growth means that the entire community is potentially at risk. This could serve to mask any programming effects. Making comparisons with other populations would be useful, although difficult, given the additional confounds this would introduce.

**Over-consumption but inadequate nutrition**

Birthweight is a rather crude measure of maternal nutrition. Deficiency in specific nutrients, particularly protein, has been shown in experimental animal studies to affect glucose metabolism of offspring: it promotes glucose intolerance and insulin resistance, without major growth retardation or disproportion apparent at birth (Langley-Evans 2001). Historical evidence from Cherbourg detailed by Hegarty (1999), Kidd (1997) and Blake (1991), along with the recollections of the study participants, provides qualitative support to the probability that although diets may have been adequate in terms of energy, rations were heavily carbohydrate
laden, supplemented with occasional off-cuts of meat which would have been fatty rather than lean. Diets in Cherbourg today remain high in simple carbohydrates and fats as demonstrated by the results of the food frequency questionnaires (Section 8.1).

If protein is particularly important among some ethnic groups as espoused by the revised thrifty genotype hypothesis of O’Dea and colleagues (Section 2.3.2), then it is conceivable that this importance can be extended to critical periods of rapid development, such as during gestation and early childhood. Inadequate protein consumption during pregnancy may therefore have an exaggerated effect on diabetes and CVD risk outcome in some populations, especially if it is combined with an over-consumption of carbohydrates and fats. Birthweight may be reduced only slightly under such circumstances.

‘Thin fat’ babies and body composition

Inadequacies in some nutrients, such as protein, and over-consumption of carbohydrates and fats may be detrimental not only during pregnancy, but also during childhood through influences on the composition and metabolism of the developing body. The easily accessible calories in a diet rich in fats and refined carbohydrates would therefore not only induce problems of glucose metabolism, but independently promote rapid childhood weight gain. The continuation of such a diet into adulthood leading to adult obesity would only serve to exacerbate any effects of impaired glucose metabolism through programming, and any such ‘programmed’ metabolic disorders would further act to promote weight gain.

Birthweights therefore may not be restricted by inadequate calories, as energy itself is not scarce. Yajnik (2002) describes the ‘thin fat’ baby phenomenon in India, whereby babies are born at low weights relative to people of European descent in a Western setting, but with amounts of body fat that are nevertheless comparable. A similar pattern may be occurring among Aboriginal babies, in Cherbourg and elsewhere. One hypothesis is that babies are being born with the propensity to put on fat, which could be the result of genetics or programming. They are then exposed to a postnatal nutritional environment that encourages weight gain through high fat and carbohydrate consumption, which continues into adulthood. Children who are born heavier than their peers may have a greater proportion of body fat, rather than lean muscle tissue. This might explain why the only association between birthweight and diabetes risk was positive, rather than negative as expected.

Therefore the relationship between lean tissue and adipose tissue may be more important than birthweight per se. Analyses of mortality by geographic area as undertaken in the early Barker
group studies were probably appropriate, given the likely nutritional deprivation of the subjects. Absolute nutritional deprivation, however, is unlikely in Cherbourg, yet the intake of some nutrients may be inadequate. Even during rationing there was probably sufficient energy available even if the quality of food was poor. Perhaps it is such inadequacies that affect the development and growth of lean muscle, so that fat babies may develop. There is some evidence that consumption of foods containing iron has been inadequate, in that a study of obstetric outcomes found that 65% of women from Cherbourg who were delivering were anaemic, versus 13% of non-Aboriginal women from the surrounding region (Powell and Dugdale 1999). Protein deficiency may also occur under such circumstances; the results of the food frequency questionnaire, combined with anecdotal evidence, implies that meat is the primary source of both protein and iron in this community.

The children who had higher infant weights and more rapid growth, and possibly increased risk of diabetes as adults (Section 0), were not necessarily ‘overweight’. Although average body weight of children by the time they are five years old is similar to international references, it may be that lean body tissue mass is low relative to fat mass. Those children who grew rapidly may have put on even more fat in relation to lean tissue, and perhaps that fat tissue was distributed more centrally among the rapid growers.

Weaning practices may further contribute to increased childhood fat percentage. Wilson et al. (1998) found that children who were fed solids before 15 weeks of age had increased percentage of body fat at age seven years and increased weight. Infant feeding practices in Cherbourg should be investigated to determine how widespread early introduction of solid foods is. For example, I was informed by community health workers that some very young infants are fed ‘watery Weet-Bix’ rather than formula, and perhaps such practices, through promotion of weight gain and inadequacies of certain nutrients, are contributing to subsequent diabetes development.

**Nutrition security**

Low SES and the influences of the pay cycle on nutritional intake may directly affect obesity. Food insecurity arising from poverty may actually promote obesity, as has been shown in two studies from the United States – among children (Alaimo et al. 2001) and women (Townsend et al. 2001) – creating a cycling of overeating which contributes to weight gain. Insecurity may lead people to ‘overeat highly palatable and rich foods’, so that over-eating followed by short period of involuntary restriction results in gradual weight gain over time (Townsend et al. 2001,
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p. 1743). Thus uncertainty in food supply, rather than being a selective genetic pressure as proposed in the original thrifty genotype hypothesis, is selecting for a particular phenotype of obesity. The absence of regular physical activity contributes greatly to this risk.

Infection and stress

An additional possibility that warrants further investigation is the hypothesised link between infectious agents and non-communicable chronic disease. Yajnik (2002) suggested that overcrowded urban populations in India might contribute to diabetes and CVD risk though an increased exposure to pathogens, and that circulating cytokines, derived from adipose tissue and involved in the immune response, might be an important mediating factor in the development of insulin resistance. Exposure to cytokines has further been implicated in relation to stress response and the programming of the HPA axis (Section 2.4.4). Björntorp (1999) hypothesised that prenatal exposure to cytokines might be the mechanism behind programming of the HPA axis and susceptibility to adult environmental stress. That cytokines are implicated via two differing pathways, infection and stress, may be particularly pertinent given the overcrowded living conditions and psychosocial stress apparent in many Aboriginal communities.

In the present study, although levels of perceived stress were not found to differ significantly between diabetes risk groups, this may be due to similarly moderate to high levels of stress throughout the community. Stress associated with poverty and powerlessness may prove to be significant in predisposing Indigenous people and communities to diabetes and related metabolic disorders. Perhaps not unrelated, several women in the community had what appeared to be Cushing’s syndrome, an extreme manifestation of disruption to the HPA axis. Self-reported perceived levels of stress are generally high among Aboriginal people, and conditions conducive to stress are certainly apparent; low SES, sometimes crowded living conditions, breakdown of traditional social organisation, marginalisation, and the conflicting pressures of work and family obligations.

Of course, neuroendocrine response and metabolism are not the only potential contributing factors which can be ‘programmed’. Food preferences are developed during childhood, and are influenced by maternal food consumption perhaps as early as gestation and the neonatal period (Birch 1999); a cycle of preference for refined carbohydrates and fats probably began with the community’s history as a settlement, and is promoted by their ready availability and affordability in the current environment. Such availability may serve to foster unhealthy preferences (Birch 1999). Parents influence their children’s eating behaviours also through the
food made available to children, with parents who focus on health providing more fruit and vegetables (Davison and Birch 2001). Restricting food available to children may serve to increase the percentage of energy intake which is fat (Davison and Birch 2001), and is a further aspect of food insecurity. Any genetic component of food preferences is likely to be minimal (Rozin and Millman 1987) or at least exhibits little variation between individuals and groups (Reed et al. 1997), but may affect preferences for fat and sweet-tasting carbohydrates (Reed et al. 1997), and how bitterness is perceived (Falciglia and Norton 1994).

**Nutrition and lifestyle transition**

That rapid lifestyle change has left some populations particularly vulnerable does not necessarily support the thrifty genotype argument that selective pressure in some populations has had no time to operate at a genetic level. Nutrition transition in any community, where there is a discrepancy between early life nutrition and later nutrition (or nutrition and physical activity) has the potential to predispose a generation to diabetes under the programming hypothesis (Yajnik 2002). That the rates continue to increase beyond a single generation may be due to the intergenerational effects on birthweight, so that a mother who has experienced poor growth passes this on to her children even in a well-nourished environment (Section 2.4.3). Eventually these effects should become attenuated, but it could take several generations for this to be appreciable. Meanwhile, prevalence of diabetes and related metabolic disorders continues to rise. Addressing the positive energy balance in adulthood is essential to diminishing the risk.

Changing habitual intakes of foods may be difficult. Cooling and Blundell (1998) found that people who habitually consume high-fat foods may be unable to distinguish between high-fat and low-fat foods, and they rate the intensity of their hunger as greater than those who habitually consume low-fat foods, further influencing them to over-eat.

### 10.2. A modified model

A new model of diabetes aetiology is required that considers contributions beyond the possible inputs from genes, early developmental processes and adult behaviours, and one that treats these factors as synergistic rather than discrete. The conceptual model should also emphasise a life-span approach. For example, physical inactivity and high carbohydrate consumption during adulthood probably contribute to, or act in synergy with, a predisposing mechanism that may have its origin in infancy. Figure 10.10 illustrates such a model, demonstrating possible pathways involved in the development of Type 2 diabetes in Cherbourg. This explanatory
framework is also applicable to other communities which have experienced similar circumstances. Included in the model is a genetic component, i.e. ethnically specific genes or ‘thrifty genotype’, and the recently hypothesised contributions from infection and environmental stress. These are not necessary for the model to work, but may enhance its ability to address the effects caused by biological and social environments.

The behavioural effects on diabetes are not restricted to adulthood, as has often been the focus for ‘lifestyle’ diseases, but these may have diverse effects at different stages of the life-span. The inter-generational health effects of lifestyle are also demonstrated.
Environmental stress promotes rapid weight gain and further programs glucose metabolism. Altered metabolism may enhance infant and childhood weight gain. Possible genetic factors increase the importance of protein vs. carbohydrates and fats in diet. Diet low in protein, high in refined carbohydrate and fat in pregnancy programs glucose metabolism, which may further exacerbate weight gain. Diet high in refined carbohydrate and fat continues into adulthood, promoting continued weight gain which is exacerbated by programmed metabolism. Altered glucose metabolism, but birthweight may be normal (thin-fat babies). Low levels of physical activity fail to moderate the effects of surplus energy. May disrupt the HPA axis (possibly further programmed by prenatal exposure to cytokines) and promote insulin resistance. Diets low in protein, high in refined carbohydrate and fat in pregnancy increase in circulating cytokines may mediate development of insulin resistance. Diet high in refined carbohydrate and fat continues into adulthood.

Figure 10.10. Hypothesised model of diabetes aetiology, illustrating possible pathways of diabetes development through contributions from genes, diet and physical activity. The influences of environmental stress and high rate of infection on diabetes development remain conjecture at this stage.

10.3. Sex and social barriers to better health

Adult lifestyle factors which promote a positive energy balance and contribute to diabetes risk are socially mediated. One of the most salient social factors affecting diabetes risk is gender, the social meanings ascribed to being female or male.

Differences between women and men are apparent for many diabetes risk factors. These differences could be a result of differential exposure to risk or differential physiological response to risk, or a combination of these factors acting synergistically. For example, differences in prenatal growth patterns could be the result of sex-specific sensitivity to
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nutritional deprivation in utero. Postnatally however, separating biological from social influences becomes more difficult, as gendered expectations and practices come into play. This study uncovered some gender-mediated barriers to better health and good diabetes care within the Cherbourg community.

Sex-specific risk

Males are more likely to have undiagnosed diabetes, as defined by the presence of three or more risk factors in addition to abnormal fasting blood glucose (Section 6.3.2). This may be due to differences in health-related behaviours, such as propensity to seek health advice. It may also be because pregnant women are tested routinely for gestational diabetes, whereas men have no such routine testing. If they make fewer visits to the community hospital than women they are also missing out on casual screening which, although imperfect, may lead to diagnosis in some instances. Although men are more likely to have three or more concurrent risk factors than women and thus be classified as high-risk, women’s levels of some risks, such as obesity, are greater.

Central obesity was much more common among women than men, especially among those who had never been diagnosed, while overweight and obesity were only slightly more common. It was also demonstrated that a lowering of the BMI thresholds for overweight and obesity by 5kg/m\(^2\) might be appropriate for Aboriginal women, while for men the internationally accepted thresholds appear appropriate. Central obesity was positively associated with childhood weight among females and rapid weight gain in childhood, in a similar manner as high BMI (Section 7.3).

Girls are more likely than boys to undergo catch-up growth (McCowan et al. 1999) (Section 3.2.4), and this may be why there were differences in diabetes risk according to relative changes in body size in childhood among women but not among men. Women who are heavier than average as infants and children are more likely to exhibit diabetes risk factors as adults, which is the opposite of what is expected under programming. However, those who gain weight at a greater rate than average during childhood are especially at risk of diabetes, meaning that rate of weight gain is important and not just absolute size at a given age. Dysregulation of the HPA axis may occur differentially among females and males, and this may explain women’s greater propensity, even at low weights, to deposit fat abdominally.

The focus of this study was on adults rather than children, as all participants were over 18 years. Discussion regarding gender differences is thus primarily restricted to those occurring among
adults. A few possibilities of differences among children deserve a brief mention. Higher levels of physical activity among boys may be generally expected and encouraged, and it may be that lower activity levels among girls who are putting on weight rapidly, rather than simply weight at a given age, are influencing their later diabetes risk.

Women in low socioeconomic groups tend to be doubly disadvantaged, suffering from both the health effects of low SES and lower status assigned to their gender (Ball et al. 2002). Women in lower SES groups tend to have more ‘traditional’ roles, i.e. they are less likely to be in paid employment, and this was apparent in this study (Section 9.1) where the main occupation of women was ‘home duties’. What this means in practical terms may include reduced financial autonomy and disposable income, and increased responsibility for the running of households and the well-being of their families and relationships.

10.3.1. Women’s busyness

A recurring pattern observed during fieldwork is that women are extremely busy taking care of others and thus less able to take care of themselves. For example, in heterosexual partnerships where both partners have diabetes, it was brought to my attention several times by the women that they ensure their partners take their medication and eat properly, while they themselves do not; they are ‘too busy’ to provide the same level of care for themselves. Previous studies of marital status and health in other populations suggest that men receive greater health benefits from marriage than women (for example, Ebrahim et al. 1995; Cheung 2000; Prior and Hayes 2001), and it may be that this type of one-way relationship in fact is detrimental to women’s health. Women, at least in Cherbourg, are the carers, men are the cared for. Women expend time and energy looking after others and reserve little for themselves.

In the present study, women in general reported poorer health than men, and diagnosed diabetic women reported the worst level (Section 9.2). Women were more likely to have seen a doctor in the last twelve months than men, which corresponds to previous studies elsewhere (for example, Fitzgerald et al. 1995). As women are more likely to seek medical or health advice, perhaps they perceive their health concerns as more serious than men do, or perhaps they are simply more willing to seek advice. This is consistent with findings from Hibbard and Pope (1983), who found that women were more likely than men to perceive their symptoms and to take action.
Bird and Fremont (1991) found that the higher morbidity that women perceive is real, a reflection of undertaking less paid work (and for lower wages) and having a greater number of caring roles and fewer hours of leisure and sleep than men. The additional roles of women, and the absence of defined hours in which to perform associated tasks, means that finding time for leisure and thus physical activity could be more difficult – there is no ‘downtime’. Even finding the energy to be more active could be difficult. Many participants, women and men, complained of always feeling tired; finding the motivation to become more active may therefore be extremely difficult.

Women who have had an acute illness event, such as a heart attack, perceive their health in terms of their ability to meet their role expectations (Abbey and Stewart 2000). This could perhaps be extended to include women with chronic illness, such as diabetes, and their perceived ability to carry on with the tasks of everyday living, which may explain why diagnosed women had the poorest health rating.

**Nutrition**

There was little reported difference in what women and men were eating, but women were less likely to think their diet was healthy, meaning that on the whole they probably had a better idea of the nutritional value of their food. This may be in part because women take more interest in their health overall.

The deficit of healthy diets in the community is only partially the result of a lack of nutrition awareness. Many participants were aware that their diet was probably unhealthy (although a large proportion said they did not know). Instead, it may be that alternatives are rarely perceived, for example, one man in his thirties explained that he drank a lot of soft drink (non-diet) because he didn’t drink alcohol, and ‘what else is there to drink?’ Food preferences are established to a degree early in life (Birch and Fisher 2000), and if a person’s diet is similar to that of their social circle, then they are unlikely to seek opportunities to stray beyond the familiar. Perhaps today’s preference for the ‘meat and potatoes’ diet is even a direct result of the types of settlement food rations; starch and meat were staples, and diet was not varied. That which had been necessity perhaps with time became preference.

There is also little opportunity to sample other types of cuisine through eating out at restaurants or cafés. Other than the take-away in Cherbourg and one in Murgon, the two pubs in Murgon serve traditional pub food (fish and chips, roast meat), and there is also a Chinese restaurant in Murgon and a restaurant at the motor inn. Eating out can also be very expensive and limited by
low income. Take-away food on the other hand is perceived to be cheap, especially given the convenience.

Even with the time and the inclination to cook healthy food, attaining fresh ingredients may be difficult. Fresh fruit and vegetables are available in Murgon although the choice is somewhat limited and prices are much higher than elsewhere, reflecting the costs of transportation and the small market. Deliveries, at the largest supermarket at least, occur only weekly. Coordinating the best time to shop with available transport can be a chore, and it is a task that usually falls to women. Men who shop are more likely to have their own transport, while for women the need to organise transport increases the necessary planning and arduousness of the task.

Women do most of the food preparation, which could provide the opportunity for more control over what they and their families eat. However, male partners often dictate the kinds of foods that they are willing to eat. A number of women explained that they would change what they were eating, but that their partners were firm about their own preferences and as they did not want to prepare two different meals, they chose to keep to them to maintain harmony. One woman told me that she ‘snuck vegetables into the stew’ because it was the only way her husband would eat them. This is consistent with findings from Baturka et al. (2000), that women feel bound to the dietary habits of their families. Thompson et al. (2000) explain the situation in another Aboriginal community thus:

…it is often difficult to meet the specific dietary needs of an individual within the family as the family meal is for the social group as a whole. A meal that is prepared specifically for one’s physical health is seen as a very different meal, cold and vacuous, compared to the warm and nourishing meal that the family consumes. (Thompson et al. 2000, p. 732)

The facilities to keep fresh food and to prepare meals are also often basic or inadequate. Some houses do not have refrigerators for example, or have a stove top but no oven, or are lacking in a suitable workspace. Availability of cooking pots or pans and utensils may also be a limiting factor in meal preparation.

Physical activity

Consuming large quantities of carbohydrates and fat is less damaging in individuals undertaking intense physical activity, but activity levels in the community are extremely low (Section 8.2.1). Increased physical activity would serve to reduce metabolic impairment through decreasing insulin resistance (Gutin and Owens 1999). Even if carbohydrate and fat intake were reduced,
intake may still be too much in relation to energy expended during physical activity, leading to an energy surplus. Fats which are consumed are more easily stored as fat than other macronutrients, and higher intake of fat is associated with increased body fat percentage and skinfolds (Davison and Birch 2001).

Physical inactivity is a major barrier to better health in Cherbourg, especially among women. Many men work as heavy labourers while women tend to work in offices or in the home. Leisure time physical activity, however, is rarely pursued, other than through weekly football or netball competitions by the minority (Section 8.2.1). Most people prefer to watch sport, being very supportive of the local teams, rather than play it. The exception to this is golf. Golf is becoming extremely popular among Cherbourg men, especially those who are older. Golf should be classified as a light rather than vigorous activity, much like walking, although it is viewed by those who play it as a fairly vigorous form of exercise. Golf can be expensive (equipment, club membership, green fees), and so it is limited to those who are employed. Women in Cherbourg do not play golf, perhaps partly through limited disposable income, but also perhaps because it is perceived as a man’s sport. Women’s golf (and sport in general) does not receive the same level of media coverage as men’s, and does not have the same level of sponsorship and prestige. This gender division does not hold for all sport – a few women in Cherbourg do play rugby.

Physical activity that is considered only light was reported by most people, especially women, and this generally takes the form of walking: to the shops, for work or for social visits. Walking is a primary form of transport, but the pace is generally slow and relaxed, and so the effects on improving cardiovascular fitness would be minimal. Aside from walking, so-called ‘active transport’, such as cycling, running, skateboarding and roller-blading, does not occur in Cherbourg. Only one person I was aware of (a young male) rode a bicycle.

Social norms for physical activity in Cherbourg are for low levels of activity. In addition to dietary practices, children learn from their family environment preferences for physical activity (Davison and Birch 2001). There is a general aversion to intense physical activity, and this is especially apparent among women, as demonstrated by participants’ responses to whether they might consider changing the amount of exercise they do if it made them healthier, whereas more said they would be willing to change their diet. Part of this may stem from an unwillingness to stand out from the crowd or seem as if they were trying to put themselves above others in some way. Anecdotal support comes from the story of one woman with diabetes who had begun...
using the gym at the sports centre quite regularly. Others in the community began to comment that she had lost weight. She then stopped going to the gym because she didn’t want to seem to be trying to be better than others. This embarrassment or ‘shame’ is unlikely to be restricted to women. Thompson et al. (2000) found that a major barrier to exercise in Aboriginal communities was that people might worry that they would start to look ‘too good’, and were more willing to undertake exercise in response to an acute illness but not when they were ‘healthy’. Lack of confidence in oneself and an unwillingness to stand out have no doubt been contributed to by the decades of discriminatory government policy and white administrative control of the settlement, and are connected with feelings of helplessness and external locus of control, influencing stress levels and participation in exercise. This unwillingness to risk appearing overly confident in oneself was illustrated in responses to questions of personal attractiveness (Section 9.3).

10.3.2. Is big beautiful? Attitudes to obesity

Overweight has become a social norm. Nearly 40% of never-diagnosed women are obese (60% are overweight or obese) and nearly a quarter of never-diagnosed men are obese (more than half are overweight or obese) (Section 6.2.1). Obesity is prevalent even among young adults. Duration of overweight and obesity is important in increasing diabetes risk; as these are apparent even among young adults, ‘exposure’ to this particular risk starts early.

In some communities internationally where a high prevalence of overweight is recorded, it is associated with a preference for larger body size (Craig et al. 1999). In Cherbourg, however, this does not seem to be the case, with a large proportion of participants expressing a preference for being thinner (approximately 30% among women – slightly higher in the diagnosed group, but only 10% among never-diagnosed men, and 35% for diagnosed men, (Section 9.3)). A desire for greater muscularity was expressed more frequently by men, especially those who had never been diagnosed, about twice as many of whom wanted to be more muscular rather than thinner.

This mirrors desire in the wider community, that women in general desire to be thin, while men desire muscularity (for example, in Australia: Crawford and Campbell 1999; in the United States: Leit et al. 2001). Perhaps not as many women and men as might be expected wish to be thinner, given the prevalence of overweight and obesity.
Despite the high BMIs and the relatively high levels of body satisfaction, there did not seem to be an overall cultural preference for larger bodies, no ‘big is beautiful’ theme that has been documented in some other cultures (Brewis et al. 1998; Craig et al. 1999). Nor is there any strong negative view of obesity. At present, there is a greater level of body confidence among women and men than may be expected, given the mean BMIs and the ideals in the wider community towards thinness and the anxiety this can promote (for example, see Harris 1983). While this apparent lack of anxiety over body image may be psychologically healthy, it could be contributing to the risk of diabetes and other chronic disease. Baturka et al. (2000), for instance, perceive a need to address perceptions of body image if motivation to lose weight is to be increased. Creating greater body dissatisfaction is not, however, a desirable means to improve health in Cherbourg.

The threshold for healthy weight perceived by participants in another study was higher than that which is medically recommended (Crawford and Campbell 1999), and this also appears to be the case in the present study with a number of participants considering that their body size and shape were healthy although they were well over the threshold for overweight. As perceptions of obesity may be correlated with functional capacity (Ferraro and Yu 1995), a better option, in terms of maintaining satisfaction with bodies, would be to work towards increasing incidental activity through emphasising how it makes people feel better and more energetic, improving capacity for achieving everyday tasks. Qualitative evidence from Baturka et al. (2000) suggests this would have greater effect on health improvement than a primary body focus; women in their study, who were also from a social environment where overweight was common, said that they wanted to ‘keep their own bodies’ but to feel healthier. Given the relative willingness of participants to change their dietary and exercise practices, implementing changes to an exercise regime may be more difficult than making dietary changes.

10.3.3. Alcohol and tobacco

More women than men in all groups reported that they never drank alcohol (75% versus 40% among diagnosed participants, 30% versus less than 20% among never-diagnosed participants). Alcohol consumption, when it does occur, is heavy, and therefore it can be considered a risk-taking behaviour in this context, as very few people drink at what might be considered ‘moderate’ levels. Other research notes that men are generally more likely to engage in risk behaviours (Chisholm 1996). In Cherbourg however, patterns of alcohol consumption are more complicated. Fewer women than men reported drinking alcohol, but the levels of risk that
women were consuming alcohol at were slightly higher than among men, and a greater proportion of women than men who drank reported consuming more than 20 drinks on a drinking day. A similar picture occurs with smoking: approximately 1.5 times as many women as men are regular smokers.

Risk-taking behaviours are usually associated with socioeconomic marginalisation and perceived lack of personal control. Young women are potentially at greater risk from these behaviours, given even fewer employment opportunities than for men and the rigid expectations of their roles within the community. With smoking especially, there has historically been a recurring theme that smoking is first taken up among the upper classes, and then trickles down to lower socioeconomic groups (for example, Ravenholt 1990). Prevalence then declines among higher socioeconomic groups but increases among lower socioeconomic groups. Perhaps this is what is happening in Cherbourg, on a small scale. Although there are no historical data for Cherbourg, smoking uptake in Australia is more prevalent among young women than young men (AIHW 2002a). Both alcohol and tobacco are significant physiological stressors, and contribute to CVD risk if not directly to diabetes risk, although they may play some role in diabetes aetiology though the pathway of dysregulating the HPA axis (Björntorp 1999).

Understanding of gender-based barriers to improving health will benefit from further in-depth qualitative research, among those participating in the Cherbourg diabetes support group for example.

10.4. Further research and recommendations

The findings from the present study lend some tentative support to the programming hypothesis, but not in its classic form. The importance of considering the life-span and risk in its social context has been emphasised through a relationship between more rapid infant and child growth, relative to the median gradient, and increased risk among women of becoming obese. An exaggerated tendency to put on fat is probably the root cause of all three modifiable risk factors – obesity, central obesity and high blood pressure - that were examined in this study. Insulin resistance may develop very early on in life, through nutritional and psychosocial stress during growth faltering causing dysregulation of the HPA axis and a propensity to deposit fat abdominally. Rapid childhood weight gain may be facilitated by insulin resistance, which may in turn be a factor in promoting catch-up growth. In a nutritional environment that is high in fat, with little physical activity, there is more dietary fat available to be deposited. With insulin resistance facilitating lipolysis, this abdominal fat becomes dangerous for CVD (Section 2.4.4).
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Stress and/or infection could therefore be significant factors underlying both rapid childhood weight gain and heightened diabetes risk. There could also be some genetic factors involved in predisposing the HPA axis to dysregulation under certain conditions. None of the risk is independent of lifestyle, but factors contributing to obesity have their origins in early life.

10.4.1. Research opportunities

There are several areas of interest for further research that arise from the present study.

1. Measure saliva cortisol and concentration of circulating cytokines to determine whether there is a relationship with risk of developing diabetes. Such a study could be structured in three parts:

   a. Women – at different stages during pregnancy, to determine if a developing fetus is exposed to high stress levels, and at which stage of pregnancy this might be most critical;

   b. Infants and children – a longitudinal study would be most valuable to determine whether periods of faltering and catch-up are associated with cortisol and/or cytokines, and how these relate to risk factors for diabetes among children (adiposity and fat distribution, blood pressure, FBSL, insulin response to glucose challenge);

   c. Adults, those with and without diabetes – to determine whether concentrations of cortisol and/or cytokines vary between groups and with risk factors.

2. Investigate dietary intake more intensively, both macro and micronutrients to establish if there are particular deficiencies or excesses which may be contributing to diabetes risk within the community. Again this could be structured as three parts:

   a. Women – examine components of maternal nutrition during pregnancy and relationship to birthweight;

   b. Infants and children – to determine which aspects of diet may inhibit growth, even if calories are sufficient, and which promote rapid fat gain over lean muscle gain.

   c. Adults, to determine in detail the mediating factors that enhance adult diabetes risk.
3. Use objective measures of physical activity, for example participants wearing a pedometer, and compare activity across groups within the community. This would also enhance opportunities for participants to increase their incidental activity.

4. Study family influences within the community on risk enhancing/moderating behaviours, especially nutrition socialisation. This would lead to a greater understanding of the social context in which nutritional risk occurs.

5. Conduct in-depth qualitative research into barriers to better health, especially as they relate to gender and other social constraints. This could be undertaken with the participants in the Diabetes Support Group, and specific intervention measures could be trialled within their own homes. Outcomes, both social and physiological, should be measured against baseline. This should be undertaken over a period of at least six months.

6. A much larger study would be more robust and have greater statistical power. Conducting a similar study to the one described here across a number of communities throughout Queensland, and even nationally, has potential to yield less ambiguous results than was possible here.

10.4.2. Community and policy implications
Community-wide dietary intervention strategies have been shown to improve indicators of health status (for example O’Dea et al. 1984; Lee et al. 1994a; Rowley et al. 1997), and such intensive forms of intervention may be feasible to a degree in the setting of the study community. Cherbourg is not an isolated community and residents have some choice, however limited, about where they do their grocery shopping for example. Preferable to an intensive intervention would be implementing long-term change in dietary and exercise habits and preferences. Changing diet may be difficult as some patterns are passed through generations and are resistant to change (Thompson et al. 2000). Thompson et al. (2000) also suggest that as connections to subsequent generations as well as past generations are important, successful programs need to focus on the future rather than on the individual in the present.

Any attempt to modifying both food consumption patterns and physical activity needs to be undertaken within the social context. For instance, Thompson et al. (2000) found in a study of Aboriginal people in Melbourne that types of foods can be categorised and given meanings that may not have such strong significance in the wider population. They also concluded that ‘diet’ foods were perceived as unsocial and isolating, disconnecting an individual from family and
community. Finding ways to imbue such healthy foods and healthy eating behaviours with positive associations so that they have resonance within the community could be a valuable step in improving community nutrition. Emphasising that healthy food is not just for those who are sick or at risk, but for everybody, may go some way towards reducing its isolating quality. Furthermore, of greater importance than an individual’s physical health is the health of relationships to family and community, and family meals contribute to maintaining such relationships (Thompson et al. 2000). Linking perceptions of the individual to family health may therefore be an extremely beneficial method of improving individual health.

Physical activity may also be seen as isolating as diet foods (Thompson et al. 2000) so that building community support and participation is essential. While the Cherbourg rugby and netball teams do reinforce a sense of community in a context of physical activity, participation in such sport is minimal.

The following items need to be addressed to reduce the risk and impact of diabetes and related complications, not only in Cherbourg, but also in other communities. The first two items in particular are intended for national and international attention.

1. Current thresholds for independent diabetes risk factors may not be appropriate for all populations. It is recommended that the threshold for obesity as measured by BMI is lowered by 5kg/m$^2$ when assessing the risk of Aboriginal women, to reflect more adequately the health risks associated with increased BMI through related central obesity. Such increased propensity for centrally deposited fat even at low BMIs has been found in previous studies. No such change in thresholds is recommended for men, although further investigation of risks associated with waist circumference is required.

2. Prevention of obesity is easier than reducing weight once obesity is established. The critical period for obesity prevention appears to be during childhood, especially among females, as rapid childhood weight gain is associated with higher adult BMI.

3. Promotion of rapid weight gain among growth-faltering infants may contribute to later diabetes risk. The widespread practice of promoting rapid short-term weight gain among infants whose growth has slowed should perhaps be reconsidered. This is a difficult issue, as the short-term benefit of survival, especially among severely faltering or low weight infants, is more pressing than possible long-term harm such rapid growth may promote. As it is inappropriate to make changes to health policy
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and practice on the basis of a retrospective longitudinal study rather than on clinical trials, addressing the cause of faltering (stress, infection) could be more appropriate than potentially overfeeding an infant.

4. Peer education within the community, emphasising the benefits of being more active and eating healthier foods, could prove an effective strategy for diabetes prevention and management. Although few diagnosed people had made changes to their diet and exercise regime, almost all those that had made changes reported benefiting from making the changes. Such people would be well-placed to be peer educators, especially if they were able to make these changes while maintaining connection with family and community. The wider social benefits of such change would need to be highlighted. That a number of participants did not mention lifestyle as a possible cause of diabetes means that lifestyle behaviours would be a good topic to target with education, to remove some of the overemphasis on genetic determinism, and put some control and responsibilities for one’s own health back into people’s lives. Measuring health at baseline so outcomes can be evaluated is essential.

5. Cultivating an internal locus of control may serve to increase physical activity, given the links between locus of control and levels of physical activity (Gregg et al. 1996). This may, however, be perceived to conflict with social and familial relationships. Methods of internalising perceived control should therefore focus on programs which are aimed at broader social concerns, in addition to individual concerns. For example, community members could undertake a short course in how to manage food buying on a low income, so there is greater nutrition security and stability throughout the pay cycle (food insecurity contributes to obesity among adults and children). Reducing perceived stress and feelings of helplessness by shifting dominant locus of control may also reduce diabetes risk that is facilitated by hypersensitivity in the HPA axis.

6. The importance of a healthy lifestyle throughout the life-span should be advocated. In particular, a focus should be placed on increasing physical activity for all. This does not have to be organised sport. One very successful program appears to be the ‘10 000 steps’ initiative (for example, Iwane et al. 2000; Lindberg 2000), where participants in the program wear a pedometer, and are encouraged to aim for taking 10 000 steps every day. This could work to increase incidental activity, such as walking to visit friends rather than driving, and walking to work. This incorporation
of activity into everyday life would be beneficial especially to young people, in prevention of obesity. As it is also more difficult to increase physical activity among older people (Samara et al. 1997), targeting such a program at young people may have more of a benefit on the scale of community.

7. The continuing low SES of Aboriginal people should be addressed. Improving SES could markedly improve health by reducing risk of diabetes via a number of pathways: reduced stress and infection, improved diet and increased physical activity through the widening of lifestyle choices that are available. Low SES is an important predictor of increasing weight over time (Ball et al. 2002). Bird and Fremont (1991) found that people who participate in productive and fulfilling roles have better health than those who are unemployed. Increasing employment within the community, or placing more value on informal roles, and promoting opportunities outside the community, may improve health and reduce diabetes risk.

10.5. Conclusions

Diabetes prevalence in Cherbourg rivals some of the highest estimates in the world. The hypothesis that high rates of diabetes in Aboriginal communities are related to high rates of Aboriginal low birthweight was not supported in this study.

No relationships were found between low birthweight and subsequent increased diabetes risk, nor was reduced postnatal growth implicated. On the contrary, where relationships were found between infant or childhood weight and diabetes risk, these were positively associated, and only occurred among females. Rapid childhood weight gain was also associated with adult obesity among females but not males. The results from this study therefore lend some qualified support to the programming hypothesis, mediated through catch-up growth, although it may simply be that a trajectory of weight gain, mediated by behaviour, commences in childhood and is continued into adulthood.

Adult lifestyle factors and environment remain important in diabetes aetiology, and at some level genetic factors may also contribute. Nutritional factors, such as a diet high in simple carbohydrates and fats, combine with physical inactivity, and physical activity may be more important than diet in moderating diabetes risk. Additional environmental factors contributing to infection and psychosocial stress may prove to be important in the high rates of diabetes in Aboriginal communities. Further study is required to refine and test these hypotheses.
Regardless of whether programming makes a significant contribution to diabetes risk in Aboriginal communities, this study provides strong evidence that diabetes should be perceived within the context of the life-span, and that risk is mediated by the social environment. Higher rates of diabetes and occurrence at earlier ages among some groups may be due to both poorer quality diet over the life-span, beginning with infancy or perhaps prenatally, and its interaction with some genetic bases. The programmed metabolic effects from poor prenatal and infant nutrition promote further weight gain during childhood, at least among females, and weight gain increases the metabolic effects. Poor health in Cherbourg is also the legacy of its history as a settlement, and in particular its residents’ continuing poverty and marginalisation.

Risk of diabetes may therefore involve prenatal responses to nutritional stress, patterns of child growth and development, and expectations and behaviours in early and later adulthood. Diabetes risk may be transmitted between generations through learned behaviours, possibly acting in synergy with some predisposing mechanism. There are sex differences apparent in many measures of diabetes risk; how these might be mitigated or reinforced through gendered attitudes and behaviours and implications for diabetes prevention and control need to be considered. At the risk of promoting an excess of individualism and a loss of community, individuals taking a certain level of personal responsibility for their lives could improve health and reduce diabetes risk significantly. Further research with larger study samples is required, especially on the potential roles of environmental stress and exposure to infection.

For as long as Australia’s Indigenous people remain socially and economically marginalised, health gaps will remain. At present, Indigenous people continue to suffer both high rates of infection and an increasing burden of chronic degenerative disease. Reductions in mortality due to infectious disease have already occurred, and rates of mortality and morbidity due to degenerative diseases may improve eventually, as has occurred more widely in the general population. Although health may slowly improve with overall economic growth, wealth gaps continue to widen. Therefore specific policies must be put in place to deal with the excessive disease burden. In Cherbourg, a funeral a fortnight is far too many.