1 Introduction

Type 2 diabetes is becoming an increasing health burden globally. Lifestyle factors which promote a positive energy balance through a combination of an energy-dense diet and physical inactivity are essential to diabetes aetiology. Certain population groups, however, are seemingly predisposed to develop diabetes under these conditions, with the fastest rising rates of diabetes occurring among various post-colonial indigenous groups.

The highest prevalence of diabetes has been recorded among the Pima Indians of Southern Arizona where diabetes occurs in up to 50% of all adults (Zimmet et al. 1997), or in more than 60% of adults aged between 45 and 65 (Zimmet et al. 1991). Type 2 diabetes is now appearing among Pima children and adolescents; by the mid 1990s, 8% of females and males aged 20-24, and 3% of 15-19 year-olds were affected (Bennett 1999).

Micronesians living in Nauru have the second highest documented rate for diabetes prevalence at just over 40% of the adult population (Zimmet et al. 1997). Phosphate mining was introduced to the island in 1906, resulting in rapid and radical lifestyle change; foods were imported and purchased rather than locally derived and physical activity levels declined as more people were employed in sedentary jobs, resulting in widespread obesity (Rubenstein and Zimmet 1993).

The third highest rate (22%) occurs among Indian Fijians living in urban centres (Zimmet et al. 1997).

In Aboriginal Australia, rates of diabetes prevalence vary greatly between communities with degree of acculturation. Approximately 20% of those living in urban areas are estimated to have diabetes, the fourth highest rate in the world (Zimmet et al. 1997), while those maintaining more traditional lifestyles are less affected. Carter and Bartley (1996) predict that up to 40% in some communities might be affected, which is overall approximately six times the rate for the total Australian population (Dunstan et al. 2002). Diabetes therefore places a significant burden on Aboriginal health, in particular through its contribution to cardiovascular diseases and renal failure, which contribute greatly to excess Aboriginal mortality and disability (AIHW 2002a).

Lifestyle factors are important. Western modes of living characterised by overnutrition and under-activity are pervasive across many countries and populations, but diabetes prevalence is
less uniform than implied by the distribution of these lifestyle characteristics. The underlying causes of the differences in diabetes prevalence have been the subject of much conjecture, experimentation and debate. Two main mechanisms have been proposed, the first based on genetic adaptation and the second on environmental conditions during development, and these have become increasingly intertwined as the literature has developed.

In 1962, James Neel hypothesised that there was a genetic basis for diabetes predisposition within certain ethnic groups, where seasonal variation in food supply acted as a selective pressure, favouring those who were more efficient at storing excess energy when it was available to call upon in leaner times. This is the ‘thrifty genotype’ hypothesis and in recent decades it has been considerably refined (O'Dea 1984, 1991, 1992; O'Dea et al. 1993; Neel 1999b) (Section 2.3). The types of foods eaten rather than the quantity and seasonality are now emphasised, and the paradigm has shifted from the efficient storage of energy to its efficient utilisation. The theme of the hypothesis, however, remains the same; genetic adaptation favoured by one environment becomes detrimental when the environment changes, particularly when a transition is made to a constant supply of energy-dense foods, with a concomitant reduction in physical activity, as associated with modernisation processes.

In the late 1980s, David Barker and colleagues proposed what was perceived as an alternative to Neel’s thrifty genotype, that adaptation to the developmental rather than evolutionary environment predisposes some individuals and groups to diabetes (Barker and Osmond 1986; Barker 1991; Hales and Barker 1992; Barker et al. 1993). The roots of this hypothesis date back to Forsdahl’s (1977) study of heart disease, and to an even earlier study of altered adult phenotypes of animals with abnormal growth (Leitch 1951). Nutritional environment, according to the programming (or ‘thrifty phenotype’) hypothesis, programs the organism through permanent physiological change at certain critical stages in development. If the nutritional environment of the organism later changes, this developmental adaptation becomes detrimental. Prenatal nutritional deprivation programs the fetus for survival in a nutritionally deprived environment. If, however, the environment later becomes one of adequate nutrition or overnutrition, metabolic disorders, such as Type 2 diabetes, can develop. Studies by Barker and others have found consistent relationships between low birthweight (<2500g) and risk of developing diabetes, and related disorders, as an adult (for example, Hales et al. 1992 Law et al. 1992b; Robinson et al. 1992; Barker et al. 1993; Fall et al. 1995a; Stein et al. 1996). Findings from more recent studies suggest that ‘catch-up’ growth following growth faltering in children is similarly related to subsequent metabolic disease (for example, Eriksson et al. 1999; Victora
and Barros 2001). The programming hypothesis thus also attempts to explain burgeoning diabetes rates in populations that have undergone recent and rapid transition, and how there may be familial clustering and intergenerational effects, but without any implied genetic differences.

Initially, these two hypotheses have been perceived and positioned as diametrically opposed and mutually exclusive. Through intensive research on both sides of the debate considerable overlap and interweaving of themes has more recently developed, so that each of the original hypotheses could be treated as extremes on a spectrum; explorations of each have made valuable contributions to furthering our understanding of diabetes. The development and dynamism of the debate is explored more fully in Chapter Two.

In Australia, not only is the prevalence of diabetes much higher in the Indigenous population, but birthweights for Indigenous babies are on average much lower than for the rest of Australia (Day et al. 1999). In addition to lower birthweight, Indigenous child growth typically shows a disrupted pattern in comparison to international reference data, such that growth faltering occurs at weaning followed by a period of catch-up growth (Gracey 1998).

The present study reviews the contributions made by both the thrifty genotype and programming hypotheses in furthering understanding of patterns of diabetes in Aboriginal Australia. It explores nutrition and lifestyle, within the socioeconomic context of a particular community, and analyses adult diabetes risk in relation to birthweight and child growth parameters, thus testing the main theme of the programming hypothesis. The study is situated in Cherbourg, an Australian Aboriginal community in southeast Queensland, which suffers from a high prevalence of diabetes.

Testing the programming hypothesis in an Aboriginal community was attempted for two key reasons. Firstly, although there have been a great number of studies in a variety of populations globally (albeit with a Eurocentric focus) there have not been any studies to date among Indigenous Australians which have specifically investigated the programming hypothesis. Given the high rates of diabetes, and low birthweight and disrupted child growth, this presents a major gap in the literature. Secondly, the past emphasis on ethnically specific genes has been unhelpful with regard to improving the diabetes situation in Aboriginal Australia, with perceptions that if genes are involved then social, political and personal responsibility is diminished. For example, an opinion commonly expressed to me during fieldwork was ‘of course I’ve got diabetes, I’m a blackfella, my whole family’s got it’. Trying to live a healthy lifestyle can be expensive, both economically and socially, in certain contexts. The term
‘lifestyle’ itself is problematic, as it implies that some level of choice is involved. Many ‘lifestyle diseases’ disproportionately affect those at the lowest socioeconomic levels, who may have little choice in terms of health-related behaviours. Being motivated to be healthy becomes even more difficult when it is believed that no amount of effort will make a difference because one’s fate is genetically sealed.

The programming hypothesis places a greater emphasis on the social origins of diabetes. Past environments, and therefore the nutritional and microbiological history of the community, may be at least as important in determining adult diabetes risk as adult lifestyle factors, with some possible contribution from population-specific genes. Considering the likely historical contributions as well as the current context of diabetes prevalence is therefore essential. For example, for decades many Cherbourg children were housed in dormitories where food quality was poor and infection rife; both poor nutrition and infection have thus influenced growth patterns in the community.

The present study consists of three parts: a clinical survey of risk factors for diabetes, a questionnaire of current lifestyle factors, and analysis of the archival birthweight and weight growth data in relation to adult diabetes risk.

The following chapter introduces the concept of health transition from infectious disease to chronic degenerative disease as most important in determining patterns of mortality, and describes the development of both the thrifty genotype and programming hypotheses. Chapter Three profiles Indigenous health in Australia across the life-span, from poorer infant health to the impact of diabetes on adult morbidity and mortality. Chapter Four introduces the study community in its historical context and the present day. The design and methods of the study and the development and implementation of the surveys are described in Chapter Five. Chapter Six presents the results from the risk factor survey, and provides estimates of both diagnosed and undiagnosed diabetes prevalence in the community, and relationships between the various risk factors. The archival growth data are described in Chapter Seven, and the means for quantifying growth variability. Child weight growth data are then analysed in relation to the surveyed risk factors to test the programming hypothesis in this community. Chapter Eight presents the results from the questionnaire regarding the well-recognised proximal causal factors of diabetes: nutrition, physical activity and lifestyle, while Chapter Nine explores further social variables that may influence diabetes risk less directly. The conclusions of the study are
presented in Chapter Ten, with discussion of barriers to better health within the Cherbourg community, the implications of the findings and directions for further research.