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Etiological and Ecological Perspectives on Geographical Variations in Infant Mortality in British Columbia

by

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A Dissertation Submitted in Partial Fulfillment of the
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DOCTOR OF PHILOSOPHY

in the Department of Geography

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ABSTRACT

Infant mortality has been viewed widely as an important indicator of population health status. The infant mortality rate in British Columbia has fallen dramatically during the past three decades, and this province now has lowest rate in Canada. The infant mortality rate of Canada is the third lowest rate in the world, higher only than that of Japan and Sweden. Despite this general decline, however, geographical inequalities in infant mortality still exist in British Columbia at the Health Unit level. Reducing differences in health status amongst regions is a goal which has been addressed recently at the international level. "Health for All by year 2000" is a public health goal set by the World Health Organization. This dissertation seeks to investigate whether or not regional inequalities in infant mortality rates in British Columbia have fallen in the same way that the provincial mortality rate as a whole has declined. Secondly, it seeks to explore, etiologically and ecologically, any potential factors which may be responsible for existing geographical inequality in infant mortality at the Health Unit scale.

To achieve these goals an index of geographical inequality, essentially a weighted coefficient of variation, was first developed. This index was then compared to the provincial infant mortality rate to examine its temporal trend and to determine whether or not geographical inequalities in infant mortality have been declining in parallel to the mortality rate as a whole. Multi-variate analyses were then performed on selected etiological and ecological factors in order to identify significant factors responsible for Health Unit specific high infant mortality rates. They were used also to identify important ecological factors which may be responsible for the high prevalence rates of the more significant etiological factors leading to elevated infant mortality rates in specific Health Units. Using these results, interactive relationships amongst ecological determinants, etiological factors, and infant mortality rates were established.

These analyses established that regional variations in infant mortalities have not been reduced to the same degree as the provincial infant mortality rate. This is especially true of the post-neonatal mortality rate for which regional differences have increased during the past 10 years. This result leads to the conclusion that infant health status in specific Health Units has not improved in comparison to that in others. Multi-variate analysis suggests that the teenage birth rate is responsible for much of the regional inequality in post-neonatal mortality, and that family income level is the ecological factor which determines the prevalence of the teenage birth rate in specific Health Units. If this relationship is correct, it implies that the teenage birth rate should be reduced and the family economic condition should be improved, in order to mitigate regional inequalities in the infant mortality rate in British Columbia.

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DEDICATION

The author wishes to dedicate this dissertation to

his grandmother, Mrs. Lan-Zen Xiao,

a great woman, who by her sacrifices

permitted the completion of this work

CHAPTER ONE

INTRODUCTION

Infant mortality is an important issue in both public health and demography. This is because infant health status forms the base on which the health of the population is established. From a demographic point of view, it is important to determine the health status and productivity of each new generation, since this has significant impacts on all aspects of society. To illustrate, pre-term children are often delayed in perceptual, memory, and motor abilities, in comparison to full-term children (Siegel, 1982). Similarly, low birth weight infants have been found to experience increased special educational needs, because of a higher incidence of school learning difficulties. They are known also to have a greater rate of psychiatric referral for behavioural disorders (Nobel-Jamieson, et al., 1982). It seems likely that societies experiencing disproportionately high numbers of pre-term and low birth weight infants will suffer from a variety of social problems, some of which will impact on productivity. As a result, improving infant health status and reducing infant mortality have become major targets worldwide. Indeed, infant mortality began to emerge as a significant concern in Britain at the beginning of the 20th century (Armstrong, 1986), and has since been accepted as the most sensitive index of social welfare and public health (Yankauer, 1990).

The need to improve infant health status has been a great challenge to clinical medicine, epidemiology, and public health because of its complicated multi-factoral etiology and ecology. To illustrate, during the time period that a fertilized

egg develops into a newborn, the fetus can be influenced by a wide variety of risk factors that can affect its growth and development. These factors may result in abnormalities of organogenesis and in an unfavourable genetic constitution. Such defects, in turn, may reduce the infant's ability to cope with its environment. Such an impaired fetal status is associated, therefore, with elevated infant mortality. A better understanding of the pathogenesis and etiology of such phenomena will definitely assist in developing appropriate strategies to improve infant health status. It is extremely difficult, however, to achieve this goal.

During the intrauterine life period a fetus generally experiences two phases of growth and development: embryonic and fetal. The embryonic phase, that is the initial 12 weeks (first trimester) after conception, is the organogenesis period, during which time the ovum differentiates into an organism that displays most of the gross anatomic features of the human form (Källén, 1988). Mortality during this period is probably higher than at any other time during life. Causes of death include gene and chromosome abnormalities and related alternations of maternal health status. Advanced maternal age, for example, predisposes to certain chromosomal abnormalities. Maternal infections, or the administration of particular drugs to the mother, during the first trimester, may influence the differentiation of the fetus and can cause congenital anomalies. It is clear that intrauterine environmental factors, responsible for defects in differentiation, exert their effects principally within the first trimester (Källén, 1988).

The fetal period, which occurs between the 12th and 40th weeks of gestation, is marked by rapid growth and an elaboration of functions. It is not until

the 24th to 26th week, however, that the fetus becomes generally viable. A variety of intrauterine factors can cause fetal morbidity during this period. These include interference with oxygenation, resulting from disturbances of the placenta or umbilical cord; infections of bacterial, viral, or protozoan origin, injury by radiation, trauma, or noxious chemicals, and maternal nutritional disturbances. Such factors may result in retarded growth, low birth weight, premature birth, and congenital and/or perinatal conditions (Klaus & Fanaroff, 1988; Källén, 1988). Infants suffering from such problems are more likely to die in infancy than normal infants (Kierans & Hu, 1995; Hu, 1995).

Birth involves passage from the intrauterine to the extrauterine environment. This neonatal period is a highly vulnerable time for the infant, which must make many physiologic adjustments to survive in the extrauterine world. High neonatal morbidity and mortality rates attest to the fragility of life during this phase. As mentioned, the infant's intrauterine to extrauterine transition requires many biochemical and physiologic changes. For instance, the newborn's pulmonary function must be activated, so that there is a self-sufficient respiratory exchange of oxygen and carbon dioxide, because the infant is no longer dependent on maternal circulation of these gases via the placenta. The newborn infant also becomes dependent upon its gastrointestinal tract function for absorbing food, upon its renal function for excreting wastes and maintaining chemical homeostasis, upon its hepatic function for neutralizing and excreting toxic substances, and upon its immunologic system for protection against infections (Källén, 1988). No longer supported by the maternal placental system, the neonatal cardiovascular and

endocrine systems also become self-sufficient. Many of the health problems which are specific to newborns are related to a poor adaption to the extrauterine environment due to premature birth, congenital anomalies, or difficult deliveries (Källén, 1988; Hook, 1984).

Many internal and external factors influence whether a newborn infant can successfully adapt to the extrauterine environment. The internal factors involved are both genetic and biomedical. Genetic constitution, for example, varies by newborn, and determines the ability to adapt, as do biomedical factors, such as premature births and low birth weight. External factors include all potential medical approaches which enhance the ability to adapt. Advanced intensive care, for example, has saved the lives of millions of newborns who would have died had such technologies not been available (Yankauer, 1990).

Maternal conditions also are closely associated with fetal and infant health status. This association exists from the pre-conception period, throughout the whole gestational period, and continues after the baby is born. It has been reported elsewhere (Kierans & Hu, 1995) that infants born to older mothers (more than 35 years old) experience higher mortality than those born to younger mothers aged between 25 to 30 years. Källén has suggested that while exposure to teratogenic agents (e.g. diseases and drugs) may increase with maternal age, the maternal capacity to metabolize teratogenic agents declines with age (Källén, 1988). Mothers with certain health conditions, such as diabetes mellitus, hypertension and congenital heart disease also have high-risk pregnancies, which are associated with elevated infant mortality. In addition, infants born into low income families, or with

less educated mothers also experience higher mortality. Such maternal conditions directly and indirectly determine the health status of an infant, and hence the mortality within the infant population (Hook, 1984; United Nations, 1985; Puffer & Serrano, 1975; Puffer & Serrano, 1987).

It is clear, therefore, that there is a sound theoretical basis to support the hypothesis that multiple maternal and infant biomedical factors directly or indirectly impact on the probability of survival of an infant. A recognition and understanding of these factors, as well as improvements in medical technologies that seek to control them, will ultimately lead to improvements in infant health status and an associated reduction in infant mortality. To illustrate, the discovery and development of antibiotics and the development of vaccinations have played major roles in controlling and preventing infant deaths from infectious diseases, especially early neonatal deaths. Similarly, neonatal intensive care units and technological advances in neonatal care have prevented hundreds of thousands of infant deaths in the United States during the 1970s (Mason, 1991; Yankauer, 1990). Significant differences in infant mortality in the Developing and Developed World are due to the limited availability of these advanced medical technologies and facilities. Also a significant proportion of infant mortality in Developing countries is due to many treatable and avoidable causes of deaths, including infections and malnutrition (Koch-Weser & Yankauer, 1991).

However, differences in infant mortality still exist within Developed countries, even those countries with universal health care systems such as Sweden and Canada. One of the major characteristics of such care systems is that all citizens

have equal access to medical care. In this case, it might be expected that every infant would have the same chance of survival so that there would be no significant disparities in infant mortality. This is not the case, however, and spatial differences not only exist, but are also consistently present over time. It must be concluded, therefore, that access to health care does not guarantee health. While a universal health care system may provide equal opportunity to access services, actual utilization of this opportunity will be determined by a variety of factors, which together result in individual inequity in health care utilization. There appears to be a further set of factors or conditions outside the control of medical technology and the health care system that have significant influences on infant health. These factors form the ecological setting, the personal and social environments of the infant. Social groups both create and are influenced by their environments. A recent study of White and Black infants from Chicago neighbourhoods, for example, found that the proportion of low birth weight infants for both Blacks and Whites increased as the census tract median income fell, regardless of maternal age, education, or marital status. This suggested that the mothers' social and physical environments influenced low birth weight birth rates, and, therefore, infant mortality (Collins & David, 1990). Indeed, Black infant mortality, in neighbourhoods that had few Blacks, was substantially lower than the comparable mortality rate for White infants whose parents resided in the Negro ghetto. Moreover, according to census figures, the educational levels and occupational classifications of the two groups of Blacks were similar, suggesting that the ghetto environment itself was related to high Black infant mortality rates (Yankauer, 1990).

It is hypothesized, therefore, that environmental settings, that is ecological ghettoes, have a major impact on infant mortality. This fact has been recognized for decades. In Britain, for instance, since the beginning of the 20th century, infant mortality has been regarded as the most sensitive index of social welfare and sanitary administration (Newsholme, 1910). It is now recognized that about 60 percent of population health-related problems result from the ecological environment, which includes dimensions of the physical environment, such as water and air quality, and societal and cultural settings, including poverty and unemployment (Millar, 1994).

As a consequence health disparities will be largely determined by environmental factors, even within the same ecological setting. In other words, inequity in population health, including differences in infant mortality between and within countries, exists largely because of differences in the environment. In Spain, for example, despite an almost 50 percent reduction in national infant-related mortality since 1976, mortality differences at the provincial scale have continued and have actually been increasing (Vazquez-Viceoso, et al, 1993). A preliminary study by this author of rural and urban infant health in British Columbia, resulted in similar findings (Hu, 1995). It is clear that urban-rural differences in infant mortality continues to exist in the province, despite a universal health care system. Such spatial differences in infant mortality cannot be completely explained by commonly recognized biological factors such as maternal age, birth weight, or length of gestation.

A new perspective on health is provided in the World Health Organization's

(WHO) publication "Health for All by the Year 2000". This argues for a universal health level. To achieve this goal of health equity, national differences in health and spatial disparities between geographical areas within nations cannot be accepted, and must be minimized. From the literature just discussed, it is clear that differences in infant mortality, between geographical areas under the same administrative system, can have either etiological and/or ecological causes. It is apparent that the determinants that underlie these geographical inequalities in infant mortality must be understood. Only when this objective has been achieved is there the solid theoretical base that is needed to effectively and efficiently modify public health policy and administrative planning. For this reason the current study will investigate potential etiological and ecological factors in British Columbia that may be responsible for the geographical inequality present in infant mortality. Although infant mortality has been reduced significantly in this province during the past several decades, geographical differences at the Local Health Area (LHA) and Health Unit (HU) level consistently have remained (Cronin & Danderfer, 1996). In addition, access to the universal health care system has reduced the possibility that regional differences in infant health status and mortality simply reflect an inability to obtain health care. As a result British Columbia is an excellent region in which to test the mortality model that ascribes infant deaths to etiological factors, but argues that the prevalence of these etiological factors are determined by the characteristics of ecological settings (Bird & Bauman, 1995). If this is true, controlling or reducing etiological factors will reduce infant mortality as a whole, but will not address the root causes of these etiological factors, and therefore, will not affect regional

differences in infant mortality. A simple example may illustrate this model more clearly. It is known that low birth weight is one of the major etiological factors influencing infant mortality. Controlling deaths due to low birth weight, from an etiological point of view, involves provision of advanced technologies designed to increase the survival of low birth weight infants. However, such an approach does not address the question, "Why do certain mothers deliver low birth weight infants?" If those ecological conditions which determine the risk of low birth weight births can be understood better, they can be modified by changes in health policy, so reducing the number of low birth weight infants and lowering infant mortality.

CHAPTER TWO

THE ETIOLOGY OF INFANT MORTALITY

Etiology involves the study of the cause(s) of disease, illness or abnormalities (Procter, 1978; Mish et al, 1983). The etiology of infant mortality, therefore, requires research into all causes of infant death. The term "cause" is used here to mean any event, circumstance, or condition that brings about, or helps to bring the occurrence of that death. It may appear relatively simple to achieve this goal but in reality it is very difficult to determine definitely the cause(s) of infant death, since a wide variety of unhealthy conditions and inappropriate behaviours may combine directly, or indirectly, to ultimately kill an infant. In addition cause of death, or cause of disease are identified differently by distinct societal groups and professions. For instance in clinical medicine physicians identify the cause of death as an event, circumstance, or condition than, if controlled or stopped, could have prevented the infant's death. In contrast, pathologists see both morphological and functional changes (either reversible or irreversible), which lead up to an infant death, as the cause of that death. In epidemiology and medical geography researchers identify determinants or direct/indirect factors which are shared by a population or community with higher infant mortality as the causes of infant deaths, rather than describe possible causal agents within that population. In order to confirm cause and effect relationships in epidemiology and medical geography, laboratory tests and animal experiments have to be integrated into the process of investigation. The Bradford-Hill criteria are widely used in epidemiology and medical geography to establish cause and effect in such population-based studies. A

variable is generally agreed to be causal if it satisfies most or all of the following nine criteria: coherence, biological plausibility, the temporal relationship of the association, dose-response curve, experimental support, consistency of the association, strength of association, the specificity of the association, and analogy (Jones & Moon, 1987). Clinically or pathologically, it is the more immediate cause of infant death that is identified. For instance, maternal and infant malnutrition are usually seen as major causes of infant death in most Developing Countries. However, depressed economic development is rarely regarded as a cause of infant mortality, although it is well known that poor nutritional status in pregnant women and infants results from such stagnant economic conditions. Whether a factor is identified as a cause of infant mortality depends heavily on current knowledge about this variable. This means that presently accepted causal factors may be considered less relevant in the future, when more significant causal variables may have been identified. It is unnecessary, therefore, to be able to establish universal causes of infant mortality before undertaking research into strategies for controlling or reducing infant death. For the purpose of this study an operational definition of the etiological factors of infant mortality has been developed. Biological characteristics of mothers and their infants such as maternal age, parity, birthweight, and gestational age, for example, are viewed as etiological factors, or causal factors, or factors having an immediate impact on infant death. As a result, two groups of etiological factors have been identified as being significant in infant mortality. Some of these are physiological theory-based, while others have been derived from epidemiological studies (with or without animal or human experiments). In addition

others are based on logic, or drawn from folklore, and have no direct scientific support. Many of these factors overlap, or are mutually dependent. Generally, they are more individual-based than population-derived. Most are abnormal conditions or bio-physiological factors that are related to mothers and their infants. They will now be briefly reviewed.

2.1 Fetal and Infant Factors

Clearly, the survival probability of an infant is determined, to a large degree, by his/her own health status. Obviously, "healthy" infants are more likely to survive than "unhealthy" ones. Health status here is indicated not only by those measurable indices such as birthweight, gestational age, and birth complications, but also by an intrinsic ability or genetic constitution to cope with any environmental changes (Novak, 1987). From a geneticist's point of view an infant's genetic constitution determines to some extent its probability of surviving (Källén, 1988). To understand infant mortality, therefore, one must be able to establish quantitatively what distinguishes "healthy" from "unhealthy" infants. Clinically, a healthy infant is fully developed, that is it is mature, and possesses the maximum internal ability to resist external invasions. The developmental, or maturity status of an infant is normally described by its birth weight and/or its gestational age. Since birthweight usually indicates the number and size of an infant's cells, fetal birth weight is closely associated with maturity and development (Klaus & Fanaroff, 1988). Usually, the heavier a fetus is, the more mature it is, and the better developed its vital organs and internal systems are. As a result, heavier feta have a greater chance of survival.

In addition to birthweight, gestational age, the length of time between conception and time of delivery, can also be used to measure an infant's development and maturity. The less the gestational age, the shorter time the fetus has been developing, and the lower its chance of survival.

Birthweight and gestational age also represent the internal ability of an infant to cope with environmental threat to some degree. A mature and well-developed infant would normally have a rational birthweight and gestational age associated with well-functioning physiological and immunological systems, enabling it to cope effectively with its environment. However, depressed birth weight and gestational age ought not to be viewed as the primary causal factors of infant death. They are intermediate factors, which themselves are determined by other internal characteristics of the infant due to pathological conditions occurring during the gestational period, and/or during the course of delivery and/or after delivery. Indeed, the causal variables may occur even before conception. In other words, both abnormal birthweight and gestational age are expressions of interactions between genetic and environmental causal factors that may take place before conception, during the gestational period, in the process of delivery or even after delivery. Differences in mortality rates for infants with comparable birthweights or gestational ages imply that other characteristics also influence the probability of infant survival. From an etiological point of view, genetic constitution is the original base upon which physiological, biochemical, immunological, and other vital systems rest, and which physiologically determines infant health status. The etiology of infant mortality should be reviewed, therefore, from its origins, that is from infant genetic

characteristics.

2.1.1 Genetic Characteristics as Causes of Infant Mortality

Maldevelopment of an embryo can occur because of errors in the genetic or chromosomal constitution, hazards in the environment, or a combination of both. This impacts on DNA, or the chromosome or embryonic development may directly cause fetal or infant death. It may result in the birth of an infant with congenital abnormalities that has a reduced chance of survival (Källén, 1988). It has been estimated that at the time when pregnancy is first recognizable clinically (about five weeks after the onset of the last menstrual period), the proportion of human conceptions with cytogenetic abnormalities is some 5 per cent (Hook, 1981a; Hook, 1983a). Almost all of these abnormalities are clinically significant because they are likely to either cause embryonic or fetal death, or alternatively to carry a high risk of retardation, or congenital defects in conceptuses surviving to a live birth (Hook, 1984). In live births the proportion with cytogenetic abnormalities has dropped to 0.6 percent, about half of which are clinically significant. The distribution of types of cytogenetic abnormalities in embryonic and fetal deaths decreases as gestational age increases.

The causes of such genetic abnormalities generally include biologic and environmental factors. It has been reported that about 9 percent of human sperm has chromosomal abnormalities (Martin et al, 1982). These data would imply, assuming that chromosomal composition of gametes does not influence zygote formation and that there is at least as high a proportion of cytogenetically abnormal

ova as sperm, that as many as 15 percent of all zygotes have some chromosomal aberration. Based on this figure and the 5 percent abnormality estimate at gestational age five weeks, at least 10 percent of all zygotes must be lost in the first three weeks after fertilization (Hook, 1984). It is uncertain, however, whether such abnormalities in human sexual cells is a natural phenomenon or is the result of external factors. In the latter case, the occurrence of such abnormalities would be increased by both additional male and /or female exposure. It may be that interaction of one or more genetic factors with one or more environmental factors is necessary to cause enough disturbance to normal development to result in a birth defect, that is many defects have a multifactorial background (Källén, 1988). To illustrate, it has been estimated that 20 percent of all congenital defects are caused exclusively by faulty genes (Wilson, 1987), while an unequivocal association between maternal age and chromosomal disorders has been reported elsewhere (Källén, 1988). The explanation of such an association is that exposure to environmental hazards increases with maternal age. For instance, the greater risk of a mutagenic event, as the age of both mother and father increases, may explain the high incidence of chondrodystrophies in the infants of elderly parents and may be at lease part of the explanation for the well-known increase in risk for chromosomal abnormalities, notably Down's syndrome, in the infants of older women (Källén, 1988).

Genetic factors may contribute less to infant mortality than to embryonic and fetal deaths, prior to the time of delivery. This is because the majority of feta with congenital defects die before birth and such greater defects are, therefore,

responsible for a large proportion of stillbirths and fetal deaths. As a result, they contribute significantly to perinatal mortality, rather than to infant mortality. The proportion of livebirths with cytogenetic abnormality is only 0.6 percent (Hook, 1981b, 1983b). Survival probability for the infants with such congenital anomalies depends largely on the severeness of such defects. As a consequence, the total contribution to infant mortality made by genetic factors is even less, although mortality is significantly higher for infants with congenital anomalies than those without (Kierans & Hu, 1995; Hu, 1995).

From an ecological point of view, geographical variation in infant mortality due to genetic anomalies can be established in two ways. Firstly, different racial genetic characteristics may determine the number of infants inheriting certain genetic abnormalities. As a result, a particular genetic constitution (genotype), resulting from weakness in chromosomes or genes, may increase the probability of infant death. This may explain in part why infant mortality differs from race to race. Secondly, variations in environmental quality, including its physical, socio-economic, religious, cultural, and life-style dimensions, also may affect the chromosomes of feta resulting in mutations during gestation. Such maldevelopment also may eventually lead to infant death. Clearly, such a death is not simply either a genetically-caused infant mortality, or an environmental-related infant death. While simple cause and effect relationships between genetic weaknesses and infant mortality has not yet been established, research has confirmed that certain races and ethnic groups possess particular genetic characteristics which favour certain congenital anomalies. It is clear, for example, that Black livebirths appear to display

a higher rate of the XXY genotype than White livebirths (Hara et al. 1976). The XXY genotype, that is Klinefelter syndrome, affects males but is not life threatening (Ritchie, 1990). Postaxial polydactyly is also reported to be more prevalent in Blacks than Whites (Myrianthopoulos, 1985). Rates of neural tube defects differ within Caucasians according to ethnicity, being particularly common in infants with Irish, Welsh or Scottish ancestry (Wilcox & Russell, 1986). It has been found also that Caucasians, as a whole, have a genetic predisposition to malformations of central nervous system (CNS) which is not present in other racial groups (Baird, 1977).

In summary, genetic factors can cause infant death, either through weakness of certain racial genotypes or through externally/environmentally-caused chromosomal abnormalities. From an etiological point of view, the former is normally recognized as an initial cause of infant mortality while the latter is categorized as an external cause. Since a larger proportion of feta with such abnormal genotypes are spontaneously aborted, the actual contribution to infant mortality made by such genotypes is probably relatively low, especially when examined at provincial or Health Unit scales. At these scales, racial variations are generally fairly insignificant. In addition, it is difficult to distinguish which life threatening genetic effects are related to genotypes and which are environmentally-caused chromosomal abnormalities. It is reasonable, therefore, to treat them as "background noise" when evaluating regional inequity in infant mortality at the provincial level. Even in British Columbia where significant variation in distribution of Aboriginal population by Health Unit appears to be responsible for regional differences in mortality rates, the death rates due to perinatal and congenital conditions between aboriginal and the

rest of BC population do not seem to be significantly different, supporting the above statements (Foster, 1995).

2.1.2 Externally-Induced Chromosome Abnormalities as Causes of Infant Mortality

As discussed in Section 2.1.1 of this thesis, external or environmental factors can cause fetal chromosomal abnormalities leading to infant death. From an etiological point of view, such infant mortalities are viewed as deaths caused by environmental factors rather than by genetic characteristics, because initial causal factors are environmental. However, not all infants or feta exposed to the same external factors eventually develop chromosomal anomalies that lead to death. Original genetic characteristics still determine, to some degree, fetal or infant fate when exposed to such environmental hazards. As a result, genotype or genetic constitution are at least contributing causal factors in such deaths.

Humans are continuously exposed to environmental hazards, including background radiation, air and water pollutants and certain potentially threatening food components. Some of these harmful agents can reach the embryo easily and theoretically damage it. There is some indirect evidence suggesting that the environment plays such a role in some birth defects. The well-known spatial variations in the distribution of neural tube defects is, to a large extent, due to ethnic and/or racial genotype characteristics, but environmental factors may also play a role (Källén, 1988). It has been now proved that shortage of folic acid can cause spinal bifida. There have been many other documented instances of infant deaths caused by chromosomal abnormalities which have resulted from exposure to

environmental hazards. While it is still uncertain whether ionizing radiation is associated with Down's syndrome (Alberman et al. 1972; Uchida, 1977), the thalidomide tragedy provided conclusive evidence that externally-induced abnormalities can cause infant deaths (Källén, 1988).

Pollutants in drinking water can reach the embryo or fetus and may genetically influence the developments of the feta. Several published studies have sought to examine whether water quality affects the frequency of birth defects. These have suggested positive associations between water softness and the rate of anencephaly (Lowe, 1971), the nitrate content of the water and neutral tube defects, and water fluoridation and malformations such as Down's syndrome (Needleman, et al., 1974; Erickson et al., 1976). Possible links between fluoridation and Down's syndrome have been studied in various countries, with controversial results. As yet there are no scientifically valid data proving that the fluoridation of the drinking water increases the risk of Down's syndrome. However, neither has the reverse been established (Källén, 1988). As a consequence, there are no valid scientific studies that prove that there is absolutely no relationship between the fluoridation of drinking water and Down's syndrome.

Air pollution has been suggested repeatedly to have adverse effects on human health, including an increased risk of reproductive failure (Källén, 1988). Since the exposure of a specific individual to air pollution is difficult to measure, estimates of impacts have been made based on comparisons of reproductive outcomes according to air quality at the place of residence. However, there are also strong correlations between place of residency and many other factors, including

socioeconomic conditions. Furthermore, place of residence only captures part of the daily air pollution exposure of an individual, since a substantial part of the day is often spent in other areas, for example, at work. Such methodological issues and associated study design variations may be responsible for the current controversy. One interesting Swedish study, with case-control design, investigated possible links between environmental hazards and the risk of giving birth to an infant with neural tube defects. This research, however, found no significant association between these two events (Ericson et al, 1989).

Links between workplace hazard exposures and congenital malformations have been more extensively studied, because of relatively higher exposure to probable hazardous materials. It was firstly reported, in Russia, in 1967, that exposures to volatile anaesthetics might be associated with reproductive failure, including miscarriages, premature delivery, or congenital malformations (Vaisman, 1967). Similar results were reported in a small Danish research project (Askrog & Harvald, 1970). Subsequently, many researchers have studied associations between workplace exposures and the possible genetic malformation of infants. Workplaces examined have included bio-medical laboratories (hospital and university research laboratories) (Funes-Cravioto, et al. 1977; Meirik, et al. 1979; Zeuthen Heidam, 1984), and offices with video display terminals (VDTs) (Delgado, et al. 1982). This literature tends to support the hypothesis that there is an association between workplace and pregnancy outcome. Work during pregnancy increases the likelihood of all types of fetal damage and in some cases may result in very specific relationships, such as that between work in printing offices and the

birth of an infant with gastroschisis (Erickson, Cochran, & Anderson, 1978). Such relationships may involve two types of causal paths. In one, chemical, or other work environment exposures may play roles in a multifactoral model, slightly increasing the risk of a disturbance in the development of the embryo. The same environment may influence the incidence of a number of different conditions. As Källen pointed out, such effects are difficult to demonstrate and impossible to disprove (Källén, 1988). It is always possible, therefore, that any factor which reaches the embryo from the woman's working environment can have such an effect. The only effective prevention is reduction of exposure to agents which can reach the embryo. A second possibility is that an occupational exposure may carry a high risk of a specific malformation. Although no such links have been scientifically proved, this does not mean that no such relationships exist.

From the preceding literature review, it is difficult to prove that environmental factors or conditions can cause genetic anomalies which, in turn, result in the birth of a maldeveloped infant with a higher than normal probability of dying. However, it is also difficult to conclude that such environmental factors have no effects on infant mortality. Part of this uncertainty may be attributed to methodological limitations, since the isolation and measurement of specific individual environmental exposures seem impossible. Studies of environmentally-induced infant mortality, whether negative or positive, may be confounded by a series of factors which have not been, and may never be, controlled. From an etiological point of view, significant differences in infant mortality experienced by populations with similar socio-demographic characteristics living in distinct geographical areas, should be

attributed largely to the environment rather than to intrinsic factors such as genotype, age, ethnicity, and social class. These latter factors are often used as the most basic descriptors of a population's risk status (Gordon, 1984). it is clear, therefore, that location must to be considered when studying geographical inequalities in infant mortality at relatively large scales.

2.1.3 Birth Weight

Birth weight is defined as the body weight of an infant at the time of delivery (Cronin & Danderfer, 1996). Infant body weight is a measure of both water and cells. That portion of this weight, contributed by cells, is determined by their size and number. During early fetal life, virtually all growth is due to an increase in cell number (hyperplasia). Increases in cell size (hypertrophy) become dominant during the latter part of gestation (Klaus & Fanaroff, 1986). It is not clear, however, how long the increase in cell number continues, or what variations occur in different organs (Sweet, 1986). Interference with the growth of the fetus during the period of hyperplasia results in organs that contain fewer cells than normal, but cells of normal size. If the insult occurs during the period of hypertrophy, the cells would be normal in number but small in size. An intrauterine insult throughout the periods of both hyperplastic and hypertrophic growth will result in fewer, smaller cells. The classic example of this is the infant with the rubella syndrome which is intrauterine infection, causing diminished fetal growth (Sweet, 1986). It was reported that 60 percent of infants with rubella syndrome fell below the tenth percentile for weight, and 90 percent fell below the fiftieth percentile (Cooper et al, 1965).

It is clear then that birth weight can indicate the degree of maturity of development of an infant during its intrauterine lifetime, that is during its gestational period. In comparison with normal infants, lighter birth weight babies may be under-developed or immature, or their development may have been retarded. In either case they will have a higher risk of dying and, therefore, cause increased infant mortality. As a result, infant birth-weight is regarded in both Developed and Developing countries as one of the most important determinants of chance for survival and of future healthy growth and development. (Puffer & Serrano, 1975; Kramer, 1987). As compared with normal-birth-weight infants (birth weight greater than 2,500 grams), those with low-birth-weight (birth weight less than 2,500 grams) are almost 40 times more likely to die during the neonatal period. Infants with very low weight (birth weight less than 1,500 grams) have a relative risk of neonatal death that is almost 200 times greater thant that of normal-birth-weight infants (McCormick, 1985). Therefore, for the past 20 to 30 years reducing the prevalence of low-birth-weight births has been the prime object of worldwide programs designed to reduce infant mortality and improve infant health status, especially in Developed countries (Piper, 1991; World Health Organization, 1985).

Birth weight, like infant death, is one of the health status measures for newborns. All factors which are associated with the development of the fetus during gestational period, therefore, have impacts on infants' birth weight. In reality, low birth weight itself has a complicated etiology which has been under investigation by researchers on a global scale. Based on the recognition of the important role of low birth weight in infant mortality, the focus in improving infant health status has shifted

recently from directly reducing infant deaths to studying and controlling the etiological causes of low birth weight. This is because LBW births contribute a significant proportion of infant mortality in almost every country. Updated knowledge about low birth weights, however, has revealed that they display similar etiological and ecological patterns to infant mortality. As Kramer (1987) summarized in his comprehensive review of the determinants of low birth weight, factors caused or associated with low birth weight can be divided generally into seven groups, namely genetic and constitutional, demographic and psychosocial, obstetric, nutritional, maternal morbidity during pregnancy, toxic exposures, and antenatal care. This multifactorial etiology and ecology, however, make it practically very difficult to identify specific causal factors of low birth weight (Källén, 1988).

Infant gender has been reported to have a rather controversial association with low birth weight. Some large-sample-sized studies have demonstrated that male infants are heavier than females, a phenomenon that exists in both Developed and Developing countries (Hingson, et al. 1982; Zuckerman, et al. 1983). However, the gender of an infant does not seem to impact on gestational age and risk of prematurity. The relative risk that female infants will experience intrauterine growth retardation (IUGR) appears to be about 1.19, in comparison with males. There is also a relative risk of 1.08 to 1.09 that females will be low birth weight infants (Kramer, 1987). The genetic basis for such gender difference in birth weight does not seem to be clear. However, the Y chromosome, which is specific to the male gender, has been reported to increase the rate of fetal growth (Alberman, 1984).

Parity is closely related to infant birthweight, although the physiological basis

for such a relationship has not yet been fully established. Nevertheless, it is well known that the birth weights of primiparous infants are significantly lower than those of multiparous infants. Such a tendency exists consistently, no matter what the mother's age at delivery. In other words, any association between birth weight and maternal age does not confound the relationship between parity and birth weight. For instance, results from a large sample, prospective study carried out in the U.S. in 1970s clearly demonstrated that the mean birthweights for both Black and White infants rose with increased parity up to 3 children, indicating a U-shaped curve for the relationship between LBW rate and parity (Hardy & Mellits, 1977). A similar relationship has also been found in Sweden (Källen, 1988) and Britain (Carr-Hill & Pritchard, 1985). However, many factors could confound this effect of parity on birthweight, making it difficult to quantify the "pure" effect of parity. As Kramer (1987) pointed out, since maternal parity is associated, to a large extent, with the socio-economic status of the family, racial and/or ethnic origin, birth interval, and maternal morbidity during pregnancy, the "pure" effect of parity on birthweight may, therefore, be small, or even insignificant. Kramer (1987) estimated that the average difference in birth weight between multiparae and primiparae was about 80 grams, and relative risk for primiparae infants to have intrauterine growth retardation (IUGR) was about 1.23. In addition, the etiologic fraction of LBW due to primiparae was between 7% to 10%, based on 30 to 50 percent of prevalence of primiparae among total live births (Kramer, 1987).

Malformation is defined as, "a morphologic defect resulting from an intrinsically abnormal developmental process" (Källen, 1988). Infants with

malformations usually also have a low birth weight. Indeed, some congenital disorders, for example anaencephaly, have a direct effect on birthweight (Carr-Hill & Pritchard, 1985). Annual birth-related information in British Columbia shows that there is a significantly high percentage of infants with malformations among LBW births, in comparison to that found amongst infants with normal birth weights (Cronin & Danderfer, 1996; Kierans & Hu, 1995). As a result, the exclusion of cases of gross fetal abnormality is common practice in birthweight surveys, in order to reduce the confounding effect of malformation on birthweight.

2.1.4 Gestational Age

Gestational age is defined as fetal age or duration of pregnancy, measured from the first day of the last normal menstrual period (Bracken, 1984; Cronin & Danderfer, 1996). It is normally expressed in completed days or completed weeks, so that events occurring 280 to 286 days after the onset of the last normal menstrual period are considered to have occurred during the 40th week of gestation. As a measurement of fetal growth, gestational age generally is divided into five groups that reflect infant maturity. These groups are extremely premature (gestational age of less than 28 weeks), moderately premature (gestation age of 28 to 36 weeks), pre-term/premature (gestational age less than 37 weeks), term or mature (gestational age between 37 to 41 weeks), and post-term or postmature (gestational age of 42 weeks or more) (Cronin & Danderfer, 1996). Obviously, gestational age measures the length of time the fetus occupied the maternal uterus, the only physiological environment for its development. The shorter the gestational

age, the less mature the fetus.

Premature infants normally have less developed morphologies and more immature physiological functions, which impair respiratory, cardiovascular, and immune systems. As a result, thermal instability, metabolic disorders, decreased oxygen delivery, infection and central nervous system problems are often seen in immature infants, increasing their probability of mortality (Claus & Fanaroff, 1986). Vital statistics related to premature infants, published elsewhere, have consistently shown that premature infants (born with a gestational age of less than 37 weeks) are significantly more at risk of dying than term births. Gestational age, like birthweight, however, is not a fundamental cause of infant mortality but rather an intermediate characteristic which is associated with more basic factors, such as the genetic constitution of the fetus and various maternal biological and demographic characteristics. However, it can be used as an indicator of risk when studying the etiology of infant mortality.

There are many arguments for and against using both birthweight and gestational age as causal indicators of infant mortality. A major theoretical concern with birthweight is that the cutoff point for defining low birth weight (<2,500 grams) was defined some 100 years ago (Rooth, 1980; Claus & Fanaroff, 1986; Kramer, 1987). It is now obvious, from interracial comparison research and immigration studies, that it is inappropriate to apply a universal standard to assess all infant birthweights. The average birthweights of Caucasian and Mongoloid newborns, for example, are significantly different. In British Columbia, those infants of Chinese origin, for instance, are on average about 200 grams lighter than the provincial

norm. As a result, based on the mortality distribution by birthweight amongst such newborns, the standard for classifying low birth weight Chinese infants should be 2,300 rather 2,500 grams (Kierans & Hu, 1995). Similar racial/immigrant difference have also been reported in the United States (Singh & Yu, 1995; Wang, Strobino & Guyer, 1992), and in the United Kingdom (Carr-Hill & Pritchard, 1985).

Concern over the accuracy of gestational age rests on the way it is recorded. Since it involves recall of the date of the start of the last normal mensural period, it rests to a large degree on the accuracy of maternal memory. In many cases the date is simply an estimate so creating significant discrepancies between recorded and actual gestational age, reducing data quality. In addition, infants with short gestational ages may, or may not, be normally developed. These two different types of premature newborns, however, experience significantly different mortality risk after birth (Kramer, 1987).

2.2 Maternal Characteristics

There is no doubt that there is very close relationship between maternal characteristics and the health status of infants. Of course, physiologically and physically infants spend their entire intrauterine life within their mothers' bodies. During this period of time they develop the body systems needed to cope with their future external environment. Necessities required for fetal growth and development are provided completely by the mother's body via the umbilical cord. As a result, the internal environment of the mother has an immediate influence on fetal development and growth including organogenesis, structural refinement, function

formation, and physical enlargement. Every change in the mother's body, therefore, has a greater or lesser impact on the fetus. For this reason, a wide variety of maternal characteristics appear to be associated with infant mortality. These include maternal demo-biologic features including age, height and weight, previous maternal pregnancy history, ethnic and racial characteristics, maternal morbidity conditions (including diseases during pregnancy) and maternal exposure experience (including personal behaviour such as smoking, alcohol consumption, and drug use) (Kramer, 1987, Källén, 1988). In addition, other variables which impact on mothers can indirectly affect an infant's survival probability. Socio-economic status and maternal education, for example, have been reported to contribute significantly to infant health status (United Nations, 1985; Bracken, 1984). Such factors will be further reviewed in the chapter discussing the ecology of infant mortality.

2.2.1 Age

The association between maternal age and infant mortality generally can be described by a U-shaped curve. Infants born to younger and older mothers generally have higher mortality rates than those born to moderately aged mothers. In British Columbia in 1995, for instance, the mortality rates of infants born to mothers aged less than 20 and over 40 years were about 9 per 1,000 live births, almost twice the mortality rate of 5 per 1,000 live births for infants born to mothers aged between 20 to 24 years (Cronin & Danderfer, 1996). This relationship has existed consistently for at least several decades in British Columbia (Cronin & Danderfer, 1994; Kierans & Hu, 1995; Hu, 1995). In addition, maternal age also

displays a close relationship with several conditions which increase the risk of dying in infants (Källén, 1988). According to Swedish data, mothers younger than 20 years or older than 35 years have a higher risk of giving birth to infants with birthweight less than 1,500 kg. Such extremely low birthweight infants have significantly higher mortality than normal birthweight babies. Infants born to adolescent (less than 20 years of age) and older mothers (over 35 years) have higher perinatal mortality and stillbirth rates, paralleling increased infant mortality. Younger and older mothers also have a higher probability of giving births to infants with certain severe congenital anomalies, including Down's Syndrome, Gastroschisis, Chondrodystrophy, and neural tube defects (NTD). According to Hook (1985), Down's syndrome possesses both a maternal age-dependent and independent etiology. At maternal age 20, the bulk of cases are maternal age independent, at about age 30 approximately half are age dependent, and by the 40s, the vast majority of cases are age dependent. There is no doubt, therefore, that maternal age is an important factor, or determinant, in the etiology of infant mortality.

There has been considerable controversy, however, over whether maternal age itself is an independent causal factor in the etiology of infant mortality (Kramer, 1987; Källén, 1988). Age is closely associated with parity. Measurement of the "pure" effects of maternal age on infant mortality, therefore, requires control for parity. Furthermore, young or adolescent mothers (those within 1 or 2 years of menarche) have not yet finished growing and are, therefore, likely to have a lower weight-for-height than older women. They frequently tend to consume fewer calories

and other nutrients. Therefore, infants born to these mothers tend to be lighter, and so for this reason have a higher probability of dying. Since such pregnancies are often unwanted or unplanned, young mothers are often late in seeking antenatal care. In addition, many younger mothers are often from lower socioeconomic classes, and, therefore, tend to have a greater use of cigarette, alcohol and illegal drugs. These substances put infants born to such teenagers at higher than normal risk. As a result, disparity in infant mortality by maternal age cannot be attributed solely to the effects of age itself (Kramer, 1987). However, there is a sound physiological basis for claiming that maternal age has an independent effect on infant health status. For instance, the risk of a mutagenic event may rise with increasing age in both men and women. Such a tendency may explain the increasing risk of chromosomal abnormalities, notably Down's syndrome in the infants of elderly parents. In addition, actual exposure to teratogenic agents, including diseases and drugs, may increase with maternal age. It is theoretically possible also that with increasing age there is a decline in the capacity of mothers to metabolize teratogenic agents. The uterine environment, endocrine mechanism, and birth process may all become less than optimal with increasing age, resulting in an increased risk of preterm birth, fetal growth retardation, birth damage, and hence infant death. In all these above examples, increased risk is a direct effect of maternal age (Källén, 1988).

2.2.2 Race/Ethnicity

From an etiological point of view, the effects of maternal race and ethnicity

on infant mortality, if any, will be exerted through a genetic-determination mechanism. Simply put, the causal association between maternal race/ethnicity and infant mortality is that unique genetic characteristics possessed by certain ethnic/racial mothers can cause higher mortality amongst their infants. For instance, neural tube defects (NTD) have been reported to have a higher prevalence amongst infants born to Caucasian mothers of Irish, Welsh or Scottish ancestry (Källén, 1988). In practice, however, such maternal race/ethnic characteristics are always interrelated with socio-economic status, which makes it difficulty to identify any independent role of maternal race/ethnicity in the etiology of infant mortality. To illustrate, the prevalence rate of postaxial polydactyly in Blacks is higher than in Whites. There are also a number of other malformations that show the same racial differences (Myrianthopoulas, 1985). If these genetic malformations are vital, their differences in prevalence will definitely cause increased Black infant mortality. However, since it is well-known that Blacks usually occupy a lower socio-economic class than Whites, at least in the US, elevated Black infant mortality cannot be explained completely by genetic characteristics. To isolate the purely genetic effects of racial/ethnic origin, studies must control for several confounding factors, including maternal age, parity, height, weight, socioeconomic status (education, occupation, and income), nutrition, potentially harmful lifestyle habits (smoking, drinking or drug use), antenatal care, birth interval, and infections. This is necessary because all or any of these factors could result in distinct infant mortality, in different racial/ethnic groups (Kramer, 1987).

Infant mortality may also be linked to racial or ethnic origin through the

racial/ethnic-birthweight-mortality path. In the United Kingdom, for example, the birth weights of infants born to mothers of Indian or Pakistani origin have been compared with those of Caucasians. It has been demonstrated that the average birthweight of ethnic infants was about 100 to 300 grams lower than that of Caucasian infants, and as a result, the incidence of technically LBW ethnic infants was higher. Similar differences have been reported in British Columbia (Kierans & Hu, 1995) and the United States (Singh & Yu, 1995). However, such studies did not find a significantly higher infant mortality amongst ethnic infants, despite the higher incidence of LBW. This suggests it may be incorrect to apply the 2,500 gram LBW standard to infants whose mothers are of Asian origin. Asian mothers are generally smaller in stature, and therefore, their infants are normally smaller than those of Caucasians. As a result, the LBW standard for Asian infants should probably be some 2,200 to 2,300 grams, similar to that proposed earlier for Asian mothers (see section 2.1.4). In this case, applying the 2,500 gram LBW standard to assess Asian infants incorrectly classifies some "normal weight" ethnic infants as being of LBW. This tends to cause an underestimate of the mortality rate amongst truly LBW infants.

2.2.3 Height/Weight

A mother's height during pregnancy is determined by three factors: her genetic potential for growth; her state of skeletal maturity; and the effect of environmental influences during the period of skeletal immaturity. These factors differ in their modifiability. Genetic potential is presumably fixed, but delayed child-bearing amongst young adolescents and, over the long term, general improvements

in nutrition, might be achieved by intervention. Maternal height can affect intrauterine growth through either genetic, or environmental (physical) mechanisms. Some of the mother's genetic potential is passed on to the fetus, and any deficit in her stature, regardless of its etiology, could impose physical limitations on the growth of the uterus, placenta, and indeed the fetus. Therefore, diminished maternal height may be one of the causes of the increased rate of LBW, and subsequent increased infant mortality (Kramer, 1987).

As with maternal height, maternal pre-pregnancy weight is influenced by both genetic and environmental factors. Even after correcting for stature, body weight is, in part, genetically determined, and genes that control adiposity or lean body mass could, theoretically, be expressed in the newborn. Even in the absence of such expression, however, maternal weight prior to conception reflects nutritional stores potentially available to the growing fetus (Kramer, 1987).

The link between maternal weight/height and infant mortality is through weight/height-birthweight-mortality. Both mothers with short stature and thin mothers tend to give birth to lighter infants. As a result, the proportion of LBW infants born to such women, when compared to those infants born to relatively heavier and taller women, is high. The mortality rate for the infants of such small, lighter women, therefore, can be expected to be above normal. However, a fundamental point must be addressed here with regards to the roles of maternal weight and height in the etiology of infant mortality, that is the racial or ethnic origins of mothers. As previously mentioned, maternal weight and height are partially genetically determined. As a result, mothers with different racial, or ethnic origins, cannot be

compared using the same standards. An Asian mother, who is smaller and lighter than a Caucasian one, may give birth to an infant weighing less than 2,500 grams (for example, 2,300 grams). According to the 2,500 gram standard now in use this infant would be classified as LBW. However, such an infant would not be necessarily at higher risk of dying, because as previously described the LBW standard for Asian infants should probably not be 2,500 grams. In a recent study carried out in British Columbia, it was found that mortality amongst infants of Chinese origins was significantly lower than the Provincial norm, despite the fact that the average birth weight for these infants was about 150 grams less than the Provincial norm (Kierans & Hu, 1995). This phenomenon could have numerous causes, but regardless of why it occurs, it throws into question about the use of 2,500 grams as a universal LBW standard (Kierans & Hu, 1995).

2.3.4 Exposure Experiences

The physiological interconnections between pregnant women and their feta are so close that the stability of the internal maternal environment has a major impact on normal fetal development and growth. The connection between a mother and her feta is via an umbilical cord. The physiological connection between a pregnant woman and her feta is so direct that changes occurring in her body must affect her fetus. To illustrate, maternal rubella infections during pregnancy can cause congenital cataracts and other malformations in their infants (Gregg, 1941). Thalidomide taken by mothers during pregnancy has caused severe limb reductions and other defects in their offspring (Lentz, 1961).

Mothers' exposures appear to influence fetal development and growth in two ways. Firstly, many substances are absorbed by the mother's body and then subsequently passed on to their fetus via the umbilical cord. This route, however, is very limited by the physiological barrier formed by the placenta, which only allows substances of a certain molecular size to pass. As a result, this size-limiting property of the placenta to certain degree protects the developing fetus from some hazardous substances in its mother's blood. The rubella virus, for example, is small enough to pass the placental barrier and so reaches the fetus via its umbilical cord, causing malformations. Secondly, hazardous agents may not actually reach the fetus but may make the placenta or the umbilical cord function inefficiently, if at all. As a result, the fetus may not be adequately nourished and its development or growth may be retarded. To illustrate, it has been hypothesised that the association between LBW births and maternal smoking during pregnancy occurs because the carbon monoxide (CO) absorbed into the mother's blood from tobacco causes the absolute quantity of oxygen carried by mother's red blood cells to be reduced. This is because of the competitive combination to hemoglobins that occurs between carbon monoxide and oxygen. Such smoking related oxygen-unsaturated-hemoglobins produce a relatively oxygen-deficient intrauterine environment, and therefore, to some degree, retards fetal development and growth. This process may ultimately lead to a LBW infant, or even to an intrauterine-growth-retarded (IUGR) birth.

Certain hazardous substances can be secreted via mothers' milk. Breast-fed babies, therefore, can be exposed to these substances after birth. In addition,

infants may be passively exposed. For instance, those born to smoking parents can become secondary smokers, inhaling second-hand smoke. Finally, infants raised in a polluted living environment may also be directly affected by toxic substances.

Maternal smoking, together with the use of alcohol or non-medical drugs, results in the most common chemical exposure of the human fetus (Källén, 1988). The association between maternal smoking and infant mortality probably occurs via the path of smoking-LBW/IUGR-infant mortality. So that while maternal smoking may not directly cause infant death, those born to smoking mothers experience a significantly higher prevalence of LBW/URGR than those born to non-smoking mothers, and mortality among LBW/IUGR infants is significantly higher than amongst those with normal birthweight. This may be why smoking and infant mortality are related. The relationship between smoking during pregnancy and low birth weight was firstly demonstrated 40 years ago (Baltzar, Ericson, & Källén, 1979). The effect of smoking on birthweight persistently remains, even after standardization for all identifiable confounders (Källén, 1988). Indeed, the relationship between smoking during pregnancy and reduced infant birthweight has been shown clearly and consistently by in a large number of studies (Bakketeig, Hoffman, Oakley, 1984).

Maternal smoking and infant birth weight also shows some dose-effect relationship which is an important criteria to establish cause-effect relationship in epidemiology (Lilienfeld & Stolley, 1994). The mean weight shift downwards is in the region of 150 to 250 grams and is not due to a mean shift in gestational age. This weight reduction is proportional to the number of cigarettes smoked and is

independent of many other factors (Meyer, 1977). The physiological mechanisms for such an effect, however, are multiple. The most likely mediators are carbon monoxide and nicotine. Carbon monoxide can interfere with oxygen delivery to the fetus, either by replacing oxygen from haemoglobin, or by shifting the oxyhemoglobin dissociation equilibrium to the left, so that less oxygen is released to the fetal tissues for a given partial oxygen pressure (Longo, 1977). Nicotine is an appetite suppressant and is believed to result in rapid increases in maternal catecholamine and consequent uterine vasoconstriction, resulting in a relative deficiency in the blood supply to the uterus and umbilical cord (Quigley, et. al, 1979). In addition, tobacco smoke also contains cyanide compounds which may cause cyanide-mediated interference with fetal oxidative metabolism, again leading to a reduced quantity of oxygen available for fetal tissues (Andrews, 1973). The combined effects of all these mechanism is to cause retarded fetal growth and development leading to a small (lighter) or even retarded growth, infant. It was estimated that, depending on prevalence rate of smoking amongst mothers of the newborn population, 8 to 14 percent of prematurity is due to maternal smoking. As a result the average birthweight of infants born to smoking mothers is 150 grams less than those born to non-smoking mothers. Some 22 to 36 percent of IUGR is also due to maternal smoking (Kramer, 1987). While survival probability for LBW/IUGR is reduced in comparison with normally developed infants, not every LBW/IUGR will eventually die because advanced medical care will save some LBW/IUGR infants. As a result maternal smoking, an etiological factor involved in infant mortality, could result in a number of infants having a higher probability of

dying but yet might not necessarily cause deaths. Maternal smoking, however, should be considered as an etiological factor when comparisons of mortality are made amongst populations.

Maternal alcohol consumption during pregnancy, often associated with smoking, is another important hazard which can have profound impacts on fetal growth and development, and, therefore, on infant mortality. Fetal alcohol syndrome, which consist of growth retardation, cognitive defects, short palpebral fissures, and maxillary hypoplasia, occurs in infants who have been severely affected by maternal alcohol consumption during pregnancy (Jones & Smith, 1974). In experimental animals, the fetal growth-inhibiting effect of high doses of alcohol has been amply demonstrated. Such alcohol may result in fetal hypoxia or the decreased incorporation of amino acids into protein (Mukherjee & Hodgen, 1982; Abel, 1982). It has been estimated that the average birthweight of infants born to mothers who consume two drinks or more per day is about 150 grams less than those born to non-alcohol drinkers. About 2 percent of IUGR is attributed to such maternal drinkers (Kramer, 1987). Although the association with increased infant mortality has been demonstrated only for animal populations (Streissguth, 1978), the increased prevalence rate of LBW/IUGR amongst infants born to maternal drinkers appears to parallel an increased infant mortality rate within the same population.

In addition to smoking and alcohol consumption, other types of maternal exposures which could potentially exert impacts on fetal growth and development, leading to an infant death include the use of illicit drugs during pregnancy and

exposure to pollutants in the work environment. Widespread use of psychoactive drugs exist in many societies, not for therapeutic use, but for their effects on the central nervous system. When such drugs are used during pregnancy addiction of the fetus may occur, together with direct harmful effects causing birth defects (Källén, 1988). Infant addiction is a significant problem amongst users of heroin or methadone (Stotzer & Wardell, 1972). Tremors and other signs of central nervous system effects due to the maternal use of marijuana during pregnancy have been described in newborns (Fried, 1982). However, it is difficult to separate direct individual drug effects from confounding factors. The women involved are often polydrug users, abuse alcohol, smoke heavily, and have a poor social environment (Källén, 1988). As a result, Kramer's comprehensive meta-analysis failed to establish direct harmful effects of drugs abuse on infant birthweight and mortality (Kramer, 1987).

2.2.5 Morbidity during Pregnancy

Since there is a close physiological connection between mothers and their infants it is not surprising that maternal health status, in both general and specific terms, has significant impacts on fetal and infant health that influence survival probability. Generally, healthy mothers have healthier babies, with reduced mortality rates. Specifically, mothers experiencing particular diseases during pregnancy will have a higher risk of delivering unhealthy babies, leading to increased infant mortality. For instance, data from a large scale prospective survey of newborns carried out in Sweden, showed that infants born to mothers with diseases such as

diabetes mellitus, urinary infections, renal disease, pre-eclampsia and eclampsia had significantly higher perinatal mortality than those born to healthy mothers. Increases in mortality associated with these diseases, varied from 150 to 400 percent (Bakketeig, Hoffman, Oakley, 1984). Maternal general morbidity and episodic illness also can affect intrauterine growth or gestational duration, leading LBW or IUGR infants who have an elevated mortality rate. The mechanism for such an association is multidimensional. Morbidity, or episodic illness, often results in a decreased caloric intake by pregnant woman which, if prolonged, may lead to an inadequate nutritional reserves impairing fetal growth. In addition, the metabolic cost of maintaining febrile temperature and of mounting the appropriate host defence may reduce the energy available to the fetus, even with a constant dietary caloric intake. Finally, infection may lead to diminished uterine blood flow, or even spread to the placenta or amniotic fluid and hence interfere with intrauterine growth, or precipitate premature delivery (Kramer, 1987). It is clear, therefore, that there is a sound physiological basis for considering maternal morbidity during pregnancy as an important etiological factor in infant mortality.

The major issue in the study of the contribution made by maternal morbidity to total infant mortality as a whole is that there are many confounding factors which cannot be easily isolated from the pure effects of maternal morbidity. Episodic infections are more likely to occur among the poor, especially those living in crowded accommodation. They may also be more likely to occur among women with low pre-pregnancy nutritional status, regardless of their socioeconomic status. For instance, respiratory symptoms occur more frequently amongst women who

smoke, and those with heavy alcohol consumption also may be prone to a variety of symptoms such as headaches and gastrointestinal upsets. As a result, pre-pregnancy weight, smoking and alcohol consumption habits, as well as poor socioeconomic status often co-exist with maternal morbidity, which makes it difficult to isolate the effects caused by such morbidity alone. In addition, the effect of maternal illness may be confounded by the treatment given. If a medication is itself capable of affecting pregnancy outcome the treatment, rather than the infection, may be responsible for the observed outcome (Kramer, 1987; Källén, 1988).

2.3 Physical Environment

The potential impacts of the physical environment on infant mortality, if any, usually result from maternal and infant exposure to sub-quality water and food. To illustrate, in the Developing World, many infants die of bacterial infections caused by contaminated drinking water or food. The development of antibiotics and mass vaccination programs and more widely available safe drinking water, air, and food have reduced infant deaths from bacterial infections in most Developed countries, including Canada (Thouez, 1992).

Current research into the relationship between the physical environment and infant mortality in Developed countries tends to focus on the roles played by non-biological factors, such as deficiencies in essential minerals (inorganic elements) and/or exposure to hazardous materials. It has been suggested, for example, that Sudden Infant Death Syndrome (SIDS) occurs most often when maternal body reserve of iodine and selenium are insufficient. Such reduced iodine and selenium

reserves are generally the result of an insufficient daily intake from food and water. When most of the diet consists of locally grown foods, such reduced maternal reserves of iodine and selenium are probably the result of depressed levels in local soil and water supply. The physical environment, therefore, causes deficiencies of iodine and selenium in local food and water which then result in an inadequate daily intake by pregnant women, leading to deprived body reserves. Subsequently, the supply of iodine and selenium for infants will also be reduced and mortality increased (Foster, 1992). The same hypothesis has also been proposed by Foster (1995) to explain infant Respiratory Distress Syndrome (RDS).

It has also been suggested that infant mortality rates are influenced by seasonal variations in temperature, cold weather being a potential causal determinant. Cold weather may promote respiratory infection in infants but may also hinder access to physicians, or cause people to be reluctant to use medical facilities (Guntheroth, Lohmann, Spiers, 1992).

This chapter has provided an overview of the etiology of infant mortality. It is clear this is so complicated that it is often described as multifactorial, or multi-dimensional. In addition, the unique relationship between infants and their mothers makes the etiology of infant mortality even more complicated because many factors influencing maternal health status can also exert significant impacts on infants. Etiology is also population specific and different populations may have their own dominant causes of infant mortality. As a result, in order to explore spatial variations in infant mortality in British Columbia factors specific to local areas must be considered and controlled for.

CHAPTER THREE

THE ECOLOGY OF INFANT MORTALITY

Ecology is "the scientific study of the pattern of relations of plants, animals, and people to each other and to their surroundings" (Procter, 1978), or "a branch of science concerned with the interrelationship of organisms and their environments; the totality or pattern of relations between organisms and their environment" (Mish et al. 1983). Therefore, an ecological health perspective seeks to discover associations or relationships between people and the environment, or the settings in which they are engaged in their daily activities and in which environmental, organizational and personal factors interact to affect human health (Chu & Simpson, 1994). From the point of view of this principle, the ecological study of infant mortality emphasizes not the immediate causal factors, but related conditions that determine the existence of such factors. The ecological perspective on disease can be demonstrated by research into the plague. Much of this has focused on the environmental conditions which enable the plague to occur, and on the transmission paths of the causal bacillus from its primary host, the black rat, to humans (Last, 1987). An understanding of the plague's ecology has permitted its virtual eradication. As this example illustrates, health ecology uses population, or geographical area based data, rather than individual-specific information to explore, or explain the relationships that exist between human health and the physical and social environments (Morgenstern, 1982; Silcocks & Murphy, 1987; Hampson 1992). The significance of health ecology and its many dimensions has been illustrated by the use of the human ecosystem model (Hancock & Perkins, 1985)

and the health field concept (Lalonde, 1973), which identify human biology, the environment, life-style, and medical care as the principle determinants of health.

The ecology of infant mortality, therefore, involves research into social and physical environments and the interactions amongst their components which determine why etiological factors related to infant mortality occur. Variations in per capita income and adult illiteracy, for instance, have been used to explain international ecological differences in infant mortality rates (Tresserras et.al 1992). However, it is significant that ecological associations or relationships are different, in principle, from etiological or causal associations. In an etiological study, an attempt is made to identify primary factors which cause the disease. A typical example of this problem would be the etiology of tuberculosis, known to be caused by a bacillus. Studies of the ecology of this disease, however, would place emphasis on identifying those conditions or elements which create the environments where this bacillus can flourish, and where conditions occur which favour a higher prevalence of tuberculosis than elsewhere. The ecology of the plague, for example, involves study of those environmental conditions which encourage black rats, the primary host of the plague bacillus. As such systematic conditions, or interactive relationships, are hard to measure, ecologists still divide or classify ecological elements into groups according to some common characteristics in order to quantitatively measure the extent to which the occurrence of a disease can be attributed to them. The ecology of infant mortality, therefore, involves factors such as the socio-economic living conditions of families, the medical or health care system of the community, the physical environment of the

residential area, and the human biology of the population. These factors, however, are not independent, but usually interact and constitute the *milieu* which determines the health status of infants in different geographical areas, or populations. They are structural determinants that are responsible for a substantial portion of the variance in state-level infant mortality, because of the difference in ecological structure that occurs at this scale (Bird & Bauman, 1995). An overview of the roles played by different aspects of the human ecology in infant mortality is now provided.

3.1 Socio-economic Status of Family

The specific meaning of the term socio-economic status is unclear. There is no comprehensive index available to quantitatively describe or measure this variable. The complexity of the situation can be illustrated by a series of different indicators which have been used by numerous researchers to tentatively define socio-economic status. These indicators are generally grouped into the following categories:

- Income, including per capita income and total family income. Obviously this may be associated with poverty, which itself is unfavourable to infant and maternal health. It is now recognized that the most important variable is the equity of income distribution (Tresserras et al, 1992; Millar, 1994).

- Parental occupation, especially the father's occupation (the type of economic activity in which he is engaged), conveys some information about socio-economic status. Studies of infant mortality across different socio-economic classes based on types of occupation have showed a distinct inequity (United Nations, 1985; Millar, 1994; Bakketeig, Hoffman,

Oakley, 1984).

- Parental education has been treated as a predictor of economic status. Based on the premise that the time and effort devoted to child care is greater for women than for men as well as on suggestive empirical findings concerning its importance, the mother's education is thought to be more significant in predicting infant health status than the father's (United Nations, 1985). The *WHO Report on Social and Biological Effects on Perinatal Mortality* (World Health Organization, 1978) described a consistent inverse relationship between educational level and perinatal mortality in several countries.

- Marital status and household structure. Single parent families have been reported to experience a higher infant mortality than complete parent families (United Nations, 1985).

- Housing status (such as ownership, renting) also reflects the socio-economic status of a family.

- Ethnicity and religion. In both Canada and United States, as well as in western Europe, it has been reported that variations in infant mortality rates reflect ethnicity and race (Klerans & Hu, 1995; Polednak, 1991).

- Other indicators, such as geographic regions (rural and urban) (Geronimus, 1986; West, 1988; Siegel et.al 1985; Hu, 1995).

3.1.1 Income

The potential role of income in infant mortality is complex, mainly due to the multi-faceted nature of income itself. According to the United Nations (1985), various aspects of income have been used to explore infant mortality. These

include:

- Household Monetary Income:
  This is defined as the total value of financial instruments (mainly cash), received by members of a household during a specified period of time.

- Household Real Income:
  Household real income is the total value of financial instruments received by members of a household, during a specified period of time, together with the value of goods and service received directly ("in-kind" income).

- Household Full Income:
  This is defined as the total value of financial instruments during a time period if all of its resources, especially the time endowment of its members, were devoted to income-generating activities. Measurement of such full income requires information on wage rates for each household member.

From an ecological point of view, infants experiencing good health can be expected to live in an environment that ensures them protection from potential risk factors, provides sufficient nourishment for them to develop internal resistances to external invasions, and gives them easy access to high-quality medical and health care. The provision of such an environment is influenced in many ways, by household income level. Infants from rich families have a greater probability of experiencing such conditions and, therefore, experience diminished mortality when compared to those from poor families. International comparisons, for example, show that infant mortality in Developing countries is far greater than that in Developed countries (Cronin & Danderfer, 1996; Danderfer, (eds). 1997; Danderfer (eds),

1998; Millar, 1996; Liu et al., 1991; World Health Organization, 1978; Lardellli, 1993). Such an inverse association between infant mortality and income level, however, exists, not only in less developed countries (Koch-Weser & Yankauer, 1991; Shin, 1975; Carvalho & Wood, 1978), but also in nations with advanced economies (Oechsli, 1995; Singh & Yu, 1995; Nordström, Cnattingius, Haglund, 1993). It has been estimated that 50% of the inequity of health status, amongst varying populations or regions within a country, can be attributed to differences in socio-economic status, while only 10%, 15%, and 25% is attributable respectively to the physical environment, genetics, and health care (Millar, 1994).

Theoretically, this income-infant mortality relationship infants occurs because expenditures are needed to provide a variety of goods and services that affect infant health. High income level families are assumed to be able to provide such support for their infants. However, this is not always true. One of the arguments made against this relationship is that family income does not always correlate linearly with an infant's consumption of health-enhancing goods and services. The economic resources directly available to, or consumed by, infants does not necessarily reflect total family income (United Nations, 1985; Millar, 1996). In addition, sources of household income may vary. If higher family income is achieved by extra hours of outside work, that in turn reduces time spent on infant care and the beneficial effect of income on reduced mortality may be reversed (Cochrane, O'Hara, Leslie, 1980).

Despite this problem, income is a reasonable measure with which to categorize the quality of an infant's ecological setting. In high income environments threats to both infants' and mothers' health are reduced, and so too is the infant

mortality rate. It has been recognized that income only affects infant mortality "through" other variables (United Nations, 1985) and, as such, is an ecological determinant rather than a causal factor in infant mortality.

3.1.2 Parental Occupation

Parental occupation may be associated with infant mortality in three ways. Firstly, parental occupation is related to occupational hazards exposure. This in turn may impose direct negative impacts on fetal development and growth, and therefore on infant health status. This is especially true of maternal exposure to hazards (Källén, 1988). Secondly, some types of paternal occupation may have an adverse effect on perinatal survival. Women who must continue to work throughout their pregnancies, therefore, may have disadvantageous social circumstances leading to the birth of unhealthy infants (Bakketeig, Hoffman, Oakley, 1984). This issue has previously been reviewed. Thirdly, parental occupation, from an ecological point of view, is seen as an indicator or proxy variable which is highly associated with a household's socio-economic status. A father's occupation, for example, is the best proxy variable for socio-economic status, providing an indicator of an infant's micro-environment and determining the basic characteristics of infant health status. It must be recognized, however, that members of the same occupational group can have different socio-economic status, depending on their position in the work hierarchy. Similarly, a particular occupation can denote varying socio-economic status in different countries. Nevertheless, occupational group conveys some information about one's socio-economic status. As a result, occupation traditionally

has been used as the basis for establishing different socio-economic classes (United Nations, 1985; Garn, Shaw, McCabe, 1977; Jones & Moon, 1987). Factors related to infant health status and mortality probability have shown remarkable differences between occupation-based socio-economic classes. To illustrate, the prevalence of premature infants, defined by either birth weights or gestational ages, has shown a significant disparity amongst occupation groups. This trend is consistent amongst different ethnic or racial groups, including Whites, Blacks, and Puerto Ricans (Garns, Shaw, McCabe, 1977; Carr-Hill & Pritchard, 1985; Källén, 1988), confirming the significance of family socio-economic status in the prevalence of premature infants.

While there is a consistent relationship between parental occupations and infant mortality rates, there are many confounding factors involved in this association. As a result, it is questionable whether occupation is an independent ecological determinant of infant mortality, or just a duplicated index of other factors such as income and education. In a broad sense paternal occupation is associated with household's income level, and represents the social position of the family. It is probable that parental occupations are only a partial indicator of socio-economic status as a whole. According to a large scale prospective study carried out in the United States, for example, the prevalence of prematurity births, either measured by gestational age or birth weight, showed different links to family socio-economic status depending on whether this was modelled by income, occupation, or educational level. This may have indicated that either the power, or the aspect of socio-economic status represented by these three variables, was different. That is

occupation, income, and education reflect different aspects of socio-economic status (Garn, Shaw, McCabe, 1977; Oechsli, 1995), probably explaining why all three variables have always been used together to measure it (Kramer, 1987; Oechsli, 1995).

3.1.3 Parental Education

Among those socio-economic indicators which have been used in infant health studies, parental education, especially maternal education level, is regarded as the most appropriate measure of family socio-economic status and to some extent is considered better than other indicators such as income and occupation (De Meer, Bergman & Kusner, 1993; Nordström, Cnattingius & Haglund, 1993; Dollfus et al, 1990; Rooth, 1980; Shoham-Yakobovich & Barell, 1988; United Nations, 1985). Family income provides a household's economic base but does not necessarily ensure that infants are well cared for. The quality of care is determined largely by parental (especially maternal) knowledge about the appropriate utilization of available health services and other resources. Educated parents, although they may not possess a high income, generally provide excellent care for their children. Distribution of premature infants across varying socio-economic groups as defined by maternal income, education, and occupation, show that the most striking disparity appears related to maternal education level (Garn, Shaw & McCabe, 1977). Premature infant birth patterns indicate that maternal education is the most sensitive measure of the socio-economic status of family, and that differences in infant mortality are much more related to maternal education level than they are to

income and/or occupational class (Garn, Shaw & McCabe, 1977). This link between education and infant health status has been identified in both Developing and Developed countries (Behm, 1980; Caldwell, 1979; Cochrane, O'Hara, Leslie, 1980; Thernlund, Samuelsson, 1993). The most impressive aspect of this relationship is the association's strength and persistence, even when other socio-economic, as well as more proximate variables, are controlled for (United Nations, 1985).

Like most socio-economic factors, maternal education does not influence infant mortality independently, rather it interrelates with other factors to provide an ecological setting which influences infant health status. For example, much of the importance of the mother's education may result from the ability of a better educated woman to attract a husband who earns more, and vice versa. Well-educated and rich men tend to bond with educated women to form families. Educated women, therefore, tend to be associated with high family income and social positions, which together favour infant health. This phenomenon has been observed in several countries (Lindenbaum et al, 1983). Also, formal education may provide dietary and hygiene knowledge and encourage better use of available health care facilities. In this respect, education and health facilities are complementary and such educational effects might be expected to be greatest in low-mortality areas. However, an important question remains to be answered "Do educated women seek out modern medical treatment because they can afford it, or because schooling has undermined their belief in traditional remedies?" Regardless of the answers to this question, there appears to be an inverse

relationship between maternal education and infant mortality (United Nations, 1985).

3.2 Availability and Utilization of Medical/Health Care

The term, medical/health care refers to the availability and accessibility of medical and health care resources, including facilities, staff and medical technologies (Joseph & Phillips, 1984). It is not unreasonable to expect child survival to be linked to the availability of such health care services. Access to modern medical facilities throughout a mother's pregnancy, at delivery, and during infancy and childhood, is thought to be particularly important in reducing infant mortality (United Nations, 1985). Advances in medical sciences and associated technologies such as intensive care, induced births, and operational deliveries have contributed to a worldwide decline in infant mortality, especially since the 1960s. Perinatal and infant mortality is presumably a good indicator of the medical care offered to pregnant women, their deliveries, and their newborn (Bakketeig, Hoffman, Oakley, 1984). Antenatal care, for example, may have a generally beneficial impact on intrauterine growth or gestation. This is a consequence of timely diagnosis and treatment of pregnancy complications (such as toxaemia, gestational hypertension or diabetes, antepartum hemorrhage, or cervical incompetence), or of the elimination or reduction of modifiable risk factors (Kramer, 1987). In a study of infant mortality in 17 Developed countries, it was found that preventable infant mortality was more highly correlated with the availability and accessibility of health service variables than with per capita income (Buck & Bull, 1986). However, availability and accessibility to medical and health care resources itself depends on many other

political, social, and economic factors. There are also no standard criteria for the ratio of medical staff to population, or for medical facilities to population. Geographical location is a further significant factor which influences the availability and accessibility of medical and health care resources.

The type or structure of a medical/health care system for a population or community is a gross measurement of availability, accessibility and utilization of medical services. Countries and regions with universal health care systems, like those of Sweden and Canada, for example, can be expected to display a favourable population health status which will include greater life expectancy and a lower infant mortality rate than those countries without it, or than those countries that rely on private health insurance systems, such as the United States (Millar, 1996). In other words, the nature of a nation's or regional health care system should be viewed as an important parameter which determines whether individuals are able to use its facilities. Depending on the design of the health care system, access to it may be constrained spatially and/or economically. Clearly, universal or selective coverage systems differ in who is permitted access (Joseph & Phillips, 1984). Accessibility to a private health insurance system largely depends on income. Poorer people will not be able to afford use, or less are likely to gain access to such a system, even though it is theoretically available. Differences in health service utilization, therefore, appears to contribute to the international variations seen in infant mortality (United Nations, 1985). They also influence spatial patterns at the state scale (Thorburn & Bauman, 1995), and survival at the individual level. As a result, the frequency of use of perinatal care and when such utilization begins have been recognized as

determinants of infant mortality (United Nations, 1985; Lardellli et al. 1991; Singh & Yu, 1995).

In countries and areas where a universal health care system is available, such as Canada and British Columbia, every citizen theoretically has equal accessibility to it from the point of view of economic affordability. This implies that health care is equally available to every individual. The impact of health care on the ecology of infant mortality, therefore, will rest on how equally people can access and/or utilize available resources. As mentioned previously, availability does not necessarily parallel accessibility and utilization. In fact it has been generally recognized that accessibility can be of two main types, physical and socio-economic. The former implies that a service and the means of reaching it are available (Moseley, 1979), whilst socio-economic accessibility involves an individual's ability to pay for a service, whether they feel it is appropriate, and whether they are permitted to use it. That is, access is also influenced by organizational and institutional restrictions on accessibility (Joseph & Phillips, 1984). Practically, it is sensible to consider access in terms of whether or not those who need care obtain it. The proof of such access is the use of services, not simply the presence of a facility. The measurement of the level of use in relation to need, is termed utilization and is regarded as revealed accessibility (Donabedian, 1973; Aday & Andersen, 1974).

In the late 1950s, the provinces of Canada began to develop universal health care systems. Such systems have been demonstrated to substantially increase the use of health care by lower-income households and lower socio-economic groups

(Enterline et al. 1973; Beck, 1973). This universal health care system has eliminated financial barriers to equitable access across social groups. The main concern regarding accessibility/utilization as an ecological determinant of infant mortality in British Columbia, therefore, is restrictions related to physical accessibility. Such restrictions may vary significantly in different geographical areas within the province, and may constitute an essential ecological aspect of infant mortality. Physical accessibility depends upon the geographic separation of facilities and potential consumers, and the consumers' mobility (Joseph & Phillips, 1984). The population distribution within British Columbia is very uneven, with the majority of people residing in the Lower Mainland. Individuals living in more northern regions are faced with greater physical separation from health care facilities, while simultaneously often having less mobility due to bad weather which may block or make transportation inconvenient. This combination may effectively constrain their use of medical and health care services. As a result, the infant population of these areas may receive fewer services, so increasing mortality risks.

Like other ecological indicators of infant mortality, accessibility and utilization of health care resources are not independent determinants, but often co-exist with other significant factors. To illustrate, areas with a deficiency of, or poor accessibility to health care resources are often less developed economically. People residing in such areas tend to be under-educated. This mixture of health determinants make it difficult to isolate the "pure" contribution made by health services to reducing infant mortality. It is this combination of factors that establish the particular ecological setting determining a newborn's environment. Infants born in remote,

undeveloped areas can be expected to experience higher mortality rates than others born elsewhere. In addition, infants born elsewhere but taken into such an area will, sooner or later, show the survival probability patterns of infants born locally. This phenomenon is often called the "environment determined" theory and is widely accepted in Immigration Epidemiology (Lilienfeld & Stolley, 1994).

3.3 Physical Environment

There have been many published reports which have demonstrated that aspects of the physical environment including air, drinking water, and food quality, are associated with the health status of populations residing in particular geographic areas. In Developing countries, the impacts of the physical environment on infant health status and mortality are probably more significant than in Developed countries. Even within Developed countries, however, there are still differences in infant mortality between particular geographical areas which occur even after data has been adjusted for other possible confounding factors. Weather, for example, may be an important physical environmental determinant of health since it has been reported that perinatal mortality tends to be higher during the winter season (Bakketeig, Hoffman, Oakley, 1984). Foster (1992; 1993) has argued repeatedly that sudden infant death syndrome (SIDS), responsible for almost 50% of all postneonatal infant deaths in British Columbia (Hu, 1995), is more common in iodine and selenium deficient environments (Foster, 1992; 1993).

Infant health status and mortality could be associated with aspects of the physical environment by means of three general relationships. Firstly, the natural

chemical composition in a particular geographic area may be conducive to the development of particular diseases and health problems leading to elevated incidence of morbidity and/or increased mortality rates amongst long-term inhabitants of the area. Such localized health problems are often referred to as endemic diseases. Examples of diseases displaying such an environmental relationships include goitre, an iodine-deficiency disorder, and Keshan disease, which is associated with a deficiency of selenium intake from food and drinking water (Foster, 1992). The second type of relationship is due to manmade environmental pollution. Such pollution can impact on human health via the air, the water supply, and/or soils through the food chain. Thirdly, some regions have naturally occurring hazards, such as high levels of background radiation, or elevated fluorine in water and soil. Long-term residents of these areas are often adversely affected by such background exposure. Developed and Developing countries both have all these types of environmentally-related health problems. It has been estimated, for example, that 15 to 20 percent of illnesses in Developed countries can be attributed to such environmental causes (Millar, 1994; Somosi, 1996).

The adverse impacts of air pollutants, such as nitrogen dioxide (NO_2), sulphur dioxide (SO_2), and particles with a mass median aerodynamic diameter of equal to or less than 10 microns (PM_{10}) have been recognized for decades (Hu & Lu, 1996). Such pollution continues to exist even in those countries with advanced economies. Due to the gradual implementation of environmental protection policies, the major sources of such pollutants have changed from industrial emissions to motor vehicles exhausts, and concern has slowly shifted from outdoor to indoor

pollution. A recent study carried out in Japan, using serum hyaluronate levels as a biological marker of lung tissue damage, found that serum hyaluronate levels were highest in schoolchildren who lived closer than 50 meters to major traffic roads. The author of this study suggested that exposure to automobile exhaust fumes (main pollutants are nitrogen oxides and suspended particulate matter) may cause injuries to lung tissue, which may, in turn, lead to increased sensitivity to respiratory infections and illness (Shima & Adachi, 1996). A very recent study carried out in the Czech Republic compared the pulmonary functions of high school students living in an area that was highly polluted with SO_2 and PM_{10}, with those living in a clean air area. This established that height-adjusted forced expiratory volume in 1 second, and forced expiratory flow between 25 and 75 percent forced vital capacity, were significantly lower in students from the polluted area. Such a difference could not be explained by other confounding factors, such as smoking and exposure to heating/cooking fuels. It was suggested that chronically depressed lung function may be caused by long-term exposure to highly polluted air (Horstman, et al., 1997).

Flow of polluted outdoor air definitely raises the pollutant level in indoor air (Cote, Wade, Yocum, 1994). In addition, many indoor pollutants are created by indoor activities. Gas cookers, gas water heaters, space heaters, and smoking increases the concentration of NO_x, which has been reported to be associated with several infant diseases. For instance, the use of a gas cooker may be a proxy measure for indoor exposure to NO_2. The rates of lower respiratory tract infection, otitis media, and diarrhea in preterm infants less than 32 weeks of age were found

to be positively associated with gas cooking (Dekker, Dales, Bartlett et al, 1991). A recently published British cross-section study reported that indoor nitrogen dioxide level is significantly correlated with the prevalence of infant diarrhea (Farrow, Greenwood, Preece, et al 1997). A similar link has been reported previously (Dekker, Dales, Bartlett et al. 1991). Farrow et. al (1997) proposed three possible mechanisms for such an association. The first involves absorption of exogenous NO from NO_x, via the infant buccal mucosa. Excess NO might reverse the production of endogenous NO formed within the gut and which is reportedly protective against gut pathogens (Benjamin, O'Driscoll, Dougall, et al, 1995; Duncan, Dougall, Johnston et al, 1995). The reduced internally-formed NO, due to excessive externally-exposed NO might increase the activity of gut pathogens, leading to diarrhea. Secondly, excess NO absorbed from the atmosphere might have a direct effect on the immature gut. NO is apparently identical to the endothelial cell-derived relaxing factor (Palmer, Ferridge, et al, 1987). The enzyme, neuronal nitric acid synthase (NOS), is localized to the nerves of the myenteric plexus of the gastrointestinal tract. Release of NO from these nerves is responsible for adaptive gastric dilation and peristalsis (Calignano, Moncada, Di-Rose, 1991), and as a purely physiological action, might result in diarrhea without the presence of a gut pathogen. Finally, NO is related to excessive formation of methaemoglobin (MetHb). The main sign of methaemoglobinaemia is a bluish discoloration of the skin followed by cyanosis, and diarrhea (Yano, Danish, Hsia, 1982; Bricker, Jefferson, Mintz, 1983).

British Columbia has a land area of approximately 950,000 km^2 and includes

a variety of physiographic regions resulting in a diversity of physical environments (Foster & Edgell, 1992). Air pollution, for instance, has been consistently worse in major urban areas, and drinking water quality varies locally (Hu & Lu, 1996). Clearly, studies of spatial variation in infant mortality must take variations in the physical environment into considerations as a significant ecological determinant.

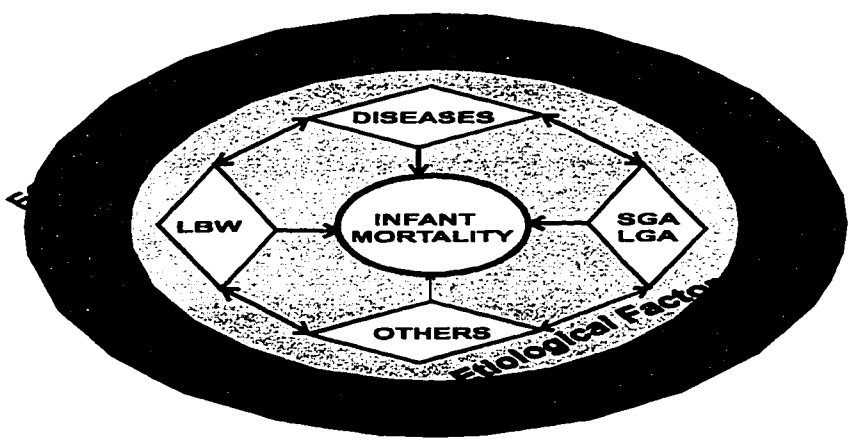
In summary, an ecological perspective sees infant mortality as the result of the fundamental nature of society. To some extent, therefore, infant mortality can be used as a comprehensive indicator reflecting societal characteristics. From this literature review, it can be seen that there are no independent determinants in the ecology of infant mortality. All are mutually related, and have distinct contributing weights to the infant mortality that occurs. A poor family, for instance, often consists of uneducated parents with a low income, living in a depressed neighbourhood, and having risky lifestyle habits. Their children are more likely to die than those of a wealthy family. An ecological study would, therefore, address the reasons behind such socio-economic inequity and attempt to identify the determinants with potential detrimental maternal and infant health outcomes.

3.4 Interrelationships of Etiology and Ecology in Infant Mortality

The etiology and ecology of infant mortality addresses from different perspectives the causes or determinants of poor infant health and the inequity of infant health status between populations, or specific geographical areas (see model in the next page). A combination of both approaches gives a more complete picture of the infant health status of particular regions. In British Columbia in 1994, for

example, infant mortality was less than 6 per 1,000. This was one of the lowest rates in the world. However, an ecological study of infant mortality in B.C. has demonstrated that significant inequity still exists between geographical regions and different communities (Hu, 1995; Kierans & Hu, 1994). This inequality illustrates that there is still the potential to further reduce infant mortality in the province. A first stage in this process involves research into the reasons for such regional inequity in infant mortality. An attempt will be made in the remainder of this thesis to identify specific causal factors and health determinants related to such infant mortality patterns.

In British Columbia the power to make decisions regarding health services, public health, and disease prevention is being shifted to local administrators. It will be useful, therefore, to provide local profiles of factors related specifically to infant health status for specific areas. This study provides data that should assist local health officers to implement policies which will ultimately help reduce geographical inequalities in infant mortality in British Columbia.



Interrelationship between Ecology and Etiology of Infant Mortality

CHAPTER FOUR

INFANT MORTALITY IN BRITISH COLUMBIA

4.1 Introduction

British Columbia is the most westerly province of Canada, bordering on the shores of the Pacific Ocean. It has a land area of approximately 950,000 km^2 and includes a variety of physiographic regions. These regions can generally be divided, from southwest to northeast, into the Outer Mountain Area, Coast Trough, Coast Mountain Area, Northern, Southern, and Central Plateau and Mountain Areas, Rocky Mountain Trench, Rocky and Mackenzie Mountain Area, and Alberta Plateau (Foster & Edgell, 1992). Variations in latitude, elevation, land and sea distribution, and relief combine to create a complex climatic mosaic. In general, the interior and the border with Alberta are cold in winter with annual temperature as low as –30°C. Their river basins receive most of their annual precipitation as snow during the winter months. The southwest coastal areas are warmer, having an average annual temperature ranging 10 to 15 °C. Precipitation in this area falls mainly as rain during winter months (Foster, 1987).

The population of British Columbia is distributed very unevenly. The majority of the province's inhabitants reside in the extreme southwest of the mainland and in southern and eastern Vancouver Island, where it is concentrated in several cities (Foster & Edgell, 1992). As a result, most of the major settlements are located in southwestern British Columbia, where the metropolitan areas of Vancouver and Victoria, together account for almost 57 percent of the total population, while the northern part of the province is the least populated area with as few as 0.36

people/km^2 living in the North West Health Region (British Columbia Ministry of Health and Ministry Responsible for Seniors, 1996). The population profile presents a wide diversity of ethnic groups. According to 1991 Census of Canada, 60 percent of British Columbia belongs to a single ethnic origin, of which the majority are of British origin, and constitute about 25 percent of the total provincial population. Other significant ethnic groups include those of Chinese (6 percent), Germany (5 percent), East Indian (3 percent), French, and Dutch (2 percent respectively), and Aboriginal origin (2 percent). The rest (about 39 percent) is multiple origin. These ethnic groups are unevenly distributed, creating an ethnic multicultural population mosaic. To illustrate, many aboriginal people reside both in urban Vancouver and in more remote coastal, interior, and northern communities. Another demographic characteristic of British Columbia is its seniors-dominated population in comparison with other provinces and territories of Canada. The age structure of the provincial population presents the typical pyramid characteristic of most western societies, with the characteristic 20 to 39 year age baby-boom bulge. However, more than 12 percent of the province's population are seniors, the majority of whom are female. This group is the fastest growing section of the population, not only because of aging of the province's own population, but also because British Columbia is a preferred retirement destination for Canadian seniors (British Columbia Ministry of Health and Ministry Responsible for Seniors, 1996; Millar, 1998).

The provincial economy has five major components of which the forest-based industries are the most important. Many communities rely to some degree on forestry, which directly employs about seven percent of the labour force. As a result

of its natural beauty, tourism is the second-largest sector of the British Columbia economy. Also significant are the mineral-based industries, agriculture, and the harvesting of marine animals (particularly salmon) and plants. This economic profile determines the provincial employment pattern. As a result, in 1991, over 70 percent of the workforce was employed in management, services, and sales, with only a very small proportion of the labour force being engaged in manufacturing (Edgell & Foster, 1992).

According to the 1991 Census of Statistics Canada, about 20 percent of the British Columbian population has attended university or has had an equivalent level of education, 27 percent have post-high school degrees, 42 percent have high-school or an equivalent education, and 11 percent have had less than a grade 9 education. The median value of household annual income in 1986 was $33,497, but for about half of all households it was only $28,770. About 25 percent of British Columbian households had an annual income less than $20,000, 40 percent had incomes of between $20,000 and $50,000, and another 20 percent earned as much as $50,000.

The province has been acknowledged as having one of the best publicly funded health systems in the world (British Columbia Royal Commission on Health Care and Costs, 1991; United States General Accounting Office, 1991). It was estimated, for example, that in 1990 British Columbia had the fewest patients per physician of any Canadian province (British Columbia Royal Commission on Health Care and Costs, 1991). In 1989, there were 38.9 acute care hospital beds (excluding bassinets), 23.3 physicians, 77.9 registered nurses, and 15.1 licensed

practical nurses per 10,000 population. The health expenditure in the 1989/90 fiscal year was about 5.7 percent of the GDP, some $4.27 billion, equivalent to $1,418 per capita. The major financial components of the British Columbian provincial health care costs are its hospital programs (48 percent), and its Medical Services Plan (27 percent).

The preceding brief review provides a profile of the ecological setting of province, that is its physical, socio-economical, demographical, and health care related dimensions. However, this overview does not adequately represent the province's regional ecology because there are enormous spatial variations in each of these geographical characteristics. This regional variation seems to be the most significant in studying geographic inequity of health status in this province. For instance, the overall mortality rate by Health Region during the period 1990-1994 after adjustment for age and gender, varied from less than 3 per 10,000 population in North Shore to almost 5 per 10,000 in South Central Health Regions. The provincial average mortality rate was 3.6 per 10,000. Variations clearly occur in the use of health facilities.To illustrate, services utilization for prenatal ultrasound, by Health Region, in fiscal year 1994/95, varied from 1.6 to over 2.5 per delivery, with a provincial average rate of 2.1 per delivery. Socio-economic variables, such as income, educational level, and employment, also present significant regional differences (British Columbia Provincial Health Officer, 1996). These can be expected to have a significant impact on both mothers' and infants' health status, and play an important role in creating spatial differences in infant mortality. The core of this study is to seek an explanation of the potential interactive relationship

between these ecological factors and the etiological causes of geographical variations in infant mortality in this province.

4.2 Infant Mortality in British Columbia

Considerable social and economic flux has occurred in Canada and British Columbia in the last 40 years and this has resulted in significant changes in demography and population health status. Since 1950, the population of the province has more than tripled, yet the birth rate (live and still births) has decreased by more than 45 percent (Cronin & Danderfer, 1996; Danderfer, 1997; Danderfer, 1998). Births, therefore, have not been the major contributor to total provincial population growth.

The provincial live birth rate, defined as the total number of live births per 1,000 population, dropped from 23.8 in 1950 to 12.4 in 1995, representing nearly a 50 percent decrease in live birth rate. It is important to be aware of this change when a temporal comparison of infant mortality is made, because the infant mortality rate is calculated by dividing the total number of infant deaths by the total number of live birth in any calendar year. This significant decline in the percentage of live births over time, therefore, may create a statistically biased comparison, since infant death is a low-probability event, which needs a relative large population (number of live births) to stablize its probability (infant mortality rate). Fortunately in the 15 years time period covered by this study, both live birth rate and the infant mortality rate, in terms of their annual changes, for the province as a whole have been relative consistent without significant yearly fluctuations, displaying stable

trends. In addition the absolute value of the denominator, the total number of live births, has increased significantly. As a result, secular comparison of infant mortality within this 15 year period is unlikely to have been unduly influenced by fluctuations in the number of live births.

Since 1965, the infant mortality rate for British Columbia as a whole generally has showed a consistent downward trend, reaching a low of 5.9 infant deaths per 1,000 live births in 1995, compared to a high of 20.3 infant deaths per 1,000 live births in 1965 (Cronin & Danderfer, 1996). The average annual reduction in this rate has been about 3.6 percent, assuming a linear relationship between infant mortality rate and time at yearly intervals. The British Columbia infant mortality rate has been slightly lower than the Canadian rate except for the period between the late 1980s and early 1990s, when the provincial rate was slight higher than the national rate. There are two major components of infant mortality, neonatal and postneonatal infant mortality rates. The former is defined as the number of infants dying during the first 27 days of life, while the latter includes all infant deaths that occur between 28 days and 1 year of age. In British Columbia, since 1965, both have experienced similar declines but of different magnitudes. The annual reduction in neonatal mortality rate has been faster than the fall in the postneonatal mortality rate, influencing the different contributions made by neonatal and postneonatal infant mortality rates to total annual infant mortality. As a result, since 1965, the neonatal-postneonatal ratio decreased from 2 to 1.5. Such changes in infant mortality trends might be attributed to a wide range of factors including advances in medical and health care technologies, improvements in education (especially parental perinatal

education), as well as increasing income linked to economic progress. Certainly, an inverse relationship between infant mortality rate and educational level and family income have been reported elsewhere (Singh & Yu, 1995).

According to the Provincial Health Officer's Report (1996), British Columbia's infant mortality rate in 1994, about 6 per 1,000 live births, was the lowest in Canada while the national rate ranked the third lowest in the world, following only Japan and Sweden. In comparison, the World's highest rate was 164 infant deaths per 1,000 live births in Sierra Leone (Provincial Health Officer of British Columbia, 1996). However, infants born in the North, Island/Coast, and Vancouver regions experienced up to twice the death rate of those born in the metropolitan areas surrounding Vancouver, a remarkable regional inequality. According to the Vital Statistics Agency of British Columbia, the aggregated infant mortality rate for the period between 1990 and 1994 varied from a significantly high 21 deaths per 1,000 live births in Prince George (Local Health Area #57) to a significantly low 4 deaths per 1,000 live births in Abbotsford (Local Health Area #34) (Cronin & Danderfer, 1996).

Such a regional disparity seems very unlikely to be due solely to the instability of the statistical methods used to deal with rare events, and appears to indicate that the causal factors behind this regional difference require further investigation. However, within British Columbia, most attention has been paid to reducing the provincial infant mortality rate and such regional disparities in mortality have not yet received comprehensive study. However, geographical differences in infant mortality rates recently have become the focus of research into the social and

geographical aspects of diseases and population health (Vázquez-Vizoso et al., 1993; Knöbel, Yang, Ho, 1994). This is because spatial differences in infant mortality at certain geographical levels may identify inappropriate policies and plans for infant health care, revealing effects that will not be removed by advances in medical sciences and associated technologies, nor by improvement in general educational levels and health (Bird & Bauman, 1993).

The major causes of infant deaths in British Columbia are congenital anomalies, perinatal respiratory conditions, and sudden infant death syndrome (SIDS). These causes have retained their relative positions during the last decade, but the contributions to total infant deaths made by each of these causes have altered. To illustrate, deaths due to perinatal respiratory conditions have declined during the period from 1981 to 1990, while deaths caused by SIDS have tended to increase, especially in male infants. In contrast, deaths due to congenital anomalies have shown non-significant changes (Cronin & Danderfer, 1994). Most notably, between 1981 and 1990, the contribution made to infant deaths by immaturity/prematurity has declined 7.5 percent, while the contribution made by deaths due to perinatal respiratory conditions rose 5.3 percent. However, regional variations specific to these leading causes have not yet been investigated.

Sudden Infant Death Syndrome (SIDS) has been of particular concern to British Columbia's Children's Commissioner. It has been recently found that there is a tremendous decrease in SIDS in British Columbia. Fisk and colleagues (Fisk, Macdonald & Kuyl, 1997) reviewed a 10-year trend of British Columbia sudden infant death syndrome, demonstrating that there has been a 56 percent reduction

of SIDS deaths during this period of time. This decline was experienced by both Aboriginal infants and the rest of BC. This trend was not associated with decrease of those infant deaths due to unknown causes. It was concluded that the SIDS mortality decline in BC is a real phenomenon, although there hasn't been found any specific factors which may be contributing to such a reduction (Fisk, Macdonald & Kuyl, 1997).

4.3 Current Studies of Infant Mortality in British Columbia

Each year the Annual Report published by the Vital Statistics Agency (formerly the Division of Vital Statistics) of the Ministry of Health and Ministry Responsible for Seniors contains some provincial summary statistics illustrating infant health status. Most of these statistical reports are descriptive and contain mortality rate for neonates, postneonates, and infants. Incidence rates of low birth weight and preterm (premature) births are also presented. In addition, conditions related to mothers' and infants' (perinatal conditions) health status, which in turn are associated with infant survival probability, are tabulated and displayed graphically. Differences in neonatal and infant mortality and the provincial average rates for specific geographical areas (such as Health Units and Local Health Areas) are also mapped and statistically analyzed. As a result, a general profile of infant health status is provided annually, at both the local and provincial scale. In addition to this regular overview, several papers have also been published by this Agency, in order to address particular topics of concern to provincial population health. Infant mortality has been always used as an indicator of health status comparison

between or within communities (British Columbia Provincial Health Officer Report, 1996).

One of the most recent of such studies of inequality in infant mortality in British Columbia was the current author's report, which compared infant mortality rates of rural and urban regions in British Columbia (Hu, 1995). In this study, all Provincial Local Health Areas (LHAs) were grouped into urban and rural regions based on certain working criteria such as population size. All infant health-related indicators including neonatal, postneonatal, and infant mortality rates were then compared by regions. Perinatal conditions, incidence of low birth weight and premature births, incidence of congenital anomalies, maternal age at birth, and availability of health care resources were also taken into account as confounding factors. The objective of this study was to attempt to answer questions such as "Is there any systematic difference in health between urban and rural regions in British Columbia?" "Are there real differences in the levels of health between urban and rural regions in British Columbia?" "What are the implications of any such regional disparity to health and health delivery in the province?"

The indicators employed in this study were subdivided into two groups: the first included those which directly affect the pregnancy process, including its end result, birth. The second group of indicators were measurements of negative end results of pregnancy, especially birth-related mortality. It was discovered that the first group of birthing status indicators were similar for both types of regions. However, there is a consistent, systematic, and statistically significant diversity with regards to birth-related mortality, between urban and rural regions. But the most

important difference occurs in post-neonatal mortality, which accounts for most of the regional disparity in infant mortality. Further investigation of this issue has showed that sudden infant death syndrome (SIDS), diseases of the respiratory system, injury and poisoning are the three leading underlying causes of post-neonatal mortality for both regions. However, variations in the frequency of death from each of these causes is the main reason for rural-urban differences in post-neonatal mortality. It has also been found that over 85 percent of post-neonatal mortality can be prevented and avoided. The evidence suggests that poor accessibility to health care is the underlying cause behind this regional diversity in post-neonatal mortality (Hu, 1995). Some factors, not considered in the study, but which vary between rural and urban regions, may also partially determine regional diversity in infant mortality in British Columbia.

The health status of immigrant groups have been compared with those of the province as a whole (Kierans & Hu, 1995). In this study, health status was measured using a set of indicators which included infant mortality, low birth weight and premature births, as measures of infant health. Two major immigration communities in B.C., Chinese and Indo-Canadians were studied. Members of these ethnic groups were identified by matching mothers' surnames with those typically used by people with Chinese and Indo-Canadian origins. The primary purpose of this project was to describe and produce a set of indicators for each community that could be used to measure health status. These could then be used to help identify the principal health needs of those two ethnic communities, and to design, implement, and evaluate health promotion activities. Based on these objectives, a

model was developed that could be used by other jurisdictions as a standard for health status assessment.

This study found significant differences in infant health status indicators between such immigrants and the provincial norm. To illustrate, the average birth weights of infants born to both Chinese and Indo-Canadian mothers were lower than the provincial average birth weight; while infant mortality and other health related indices, such as perinatal conditions and congenital anomalies were lower than the provincial average. The overall results were comparable to those in other published papers focusing on Asian immigrant infants (Singh & Yu, 1995; Weeks & Rumbaut, 1991). Together this literature suggested that further research may be needed into determinants that are specific to ethnic infants and into possible ways of reducing differences in mortality. This research also has implication for regional inequality in infant mortality, since uneven ethnic population distribution may be one of the causes of spatial differences in infant mortality.

One of the present key issues in British Columbia's population health is the health of Status Indians. Since 1989, the Vital Statistics Agency of British Columbia has been involved in an analysis of British Columbia's Status Indian population. Two recent publications specifically address this issue. To illustrate, the *Analysis of Status Indians in British Columbia, A Vital Statistics Overview,* published in 1994, provided valuable information on the health status of B.C. Status Indians at the provincial level (British Columbia Vital Statistics Agency, 1994). However, the Status Indian population is unevenly distributed within the province and, as a result, an analysis of the health status for the province as a whole can not meet the demand

for regional level information. In order to address regional variations in Status Indian health status another report that focussed mainly on the spatial characteristics of Status Indian health profile, was also published by the Agency in the same year. This provided more detailed analysis of regional Status Indian health status (Burd, 1994).

It was estimated that in 1992 there were some 97,000 Status Indians in British Columbia, comprising less than 3 percent of the total population. The distribution of this ethnic group varies significantly by health regions. For example, the percentage of Status Indians compared to total Health Region population ranges from a high of 22 percent in Skeena to less than 2 percent in Richmond. Such a distribution pattern naturally is also paralleled by the births, resulting in 18 percent of all provincial Status Indian births in Skeena and only 5 percent in Upper Vancouver Island.

The health status of infants born to Status Indian mothers differs from both regional and provincial norms. Generally, such infants are less healthy than either regional or provincial averages. To illustrate, the low birth weight rate is higher amongst infants born to Status Indian mothers in fourteen out of a total of twenty Health Regions; sixteen Health Regions also have a higher rate of premature births for Status Indian infants than the regional norms. In addition, infant mortality rate is consistently higher for the Status Indian infants in every Health Region, and births to teenage mothers (under 20 years of age) are also consistently higher among Status Indian births across all Health Regions. One of the exceptional characteristics of Status Indian infants is that Status Indian mothers experience a

significantly lower rate of C-caesarean sections. This phenomenon also seems to be consistent across every Health Region and, therefore, for the province as a whole (Burd, 1994).

Foster and colleagues performed, from a vital statistics perspective, a more detailed analyses on Native health in British Columbia (Foster et al., 1995). Although it encountered the same problems in defining different types of Aboriginal people, this analysis is probably one of the most comprehensive studies of the health of Aboriginal people in this province. It included cause-specific mortality comparisons for Status Indians and the remainder of the population of British Columbia and examined maternal conditions associated with infant mortality for both groups. The infant mortality rate (13.9 per 1,000 live births) for Status Indians, for the time period from 1987 to 1992, was more than twice that of the rest of BC (6.9 per 1,000 live births). Interestingly, infancy mortality was subdivided into early-neonatal, neonatal, post-neonatal, both the early neonatal and neonatal mortality rates for Status Indians were lower than those of the rest of BC, but the post-neonatal mortality was nearly 4 times of that of the rest of British Columbia. Elevated SIDS appears to be responsible for this high post-neonatal mortality rate. The post-neonatal deaths due to SIDS for the Status Indians was almost 4 times that of the rest of British Columbia (Foster et al, 1995). This was confirmed two years later by Fisk and colleagues (Fisk, Macdonald, Kuyl, 1997). Although no causal factors were identified by Fisk and colleagues, their results demonstrated that there is a significant difference in infant mortality between Status Indians and the rest of the BC population.

As a consequence, the spatial distribution of Status Indian infants must account for some of the geographical inequality in infant mortality in British Columbia. However, it is not the only determinant. Differences in infant mortality between Status Indian births and regional norms, for example, do not always consistently follow the pattern of difference of live births. To illustrate, in Skeena, Status Indian births account for over 30 percent of total infants, while the percentage for the Upper Fraser Valley is slightly over 5 percent. However, difference in infant mortality between Status Indian births and the regional norm is significantly different, less than 4 per 1,000 live births in Skeena, but about 15 per 1,000 live births in the Upper Fraser Valley. Low birth weight (LBW) births present a similar pattern. The Status Indian LBW rate in Skeena is slightly lower than the regional average, while Status Indian LBW rate in the Upper Fraser Valley is one-third more than the regional norm. These figures demonstrate that Status Indian infants are less healthy than other British Columbian infants, a fact that can explain part of the regional disparity in infant mortality across all Health Regions. As a result, aboriginal infant status is an important factor which must be taken into account when investigating geographical inequality in provincial infant mortality. However, such geographical inequality is not solely determined by the distribution of Status Indian infants, since they experience different survival probabilities depending on the Health Regions involved.

Out-of-province services utilization for infants residing along the border between British Columbia and Alberta may confound the study in regional difference in infant mortality. For instance, there is relatively a large number of vital events

involving British Columbia residents that occurred in Alberta, which is particular true for people residing in the Kootenay region in the southeast corner of BC (Burr, et al. 1995). Due to variety of the reasons, these out-of-BC occurred infant deaths have not been captured in BC Vital Statistics mortality database. As a result, the number of recorded infant deaths for this region are actually lower that what has actually occurred in that region, which leads to a lower-than-BC-average infant mortality rate in this region. To illustrate, during the time period from 1985 to 1992, there was a total of 53 infant deaths that were not in the BC mortality database, but which were captured in Alberta registered death database. This number accounts for about 2 percent of total infant deaths registered in BC for the same time period. However, when these out-of-BC occurred infant deaths were split into Health Regions, the percentage of the deaths, in comparison with total infant deaths, can reach up to 5 percent in the Kootenay Area (Burr, et al. 1995). When these out-of-BC occurred infant mortality were combined to the deaths registered to BC, the infant mortality rates for certain regions may be changed from significant lower to higher than the provincial norm.

4.4 Infant Mortality in British Columbia: Areas Requiring Additional Study

The reduction of infant mortality achieved by British Columbia has been very impressive. This success is reflected by the significant reduction in the provincial infant mortality rate that has occurred in the past 30 years. As previously discussed, the provincial infant mortality rate has declined by some 71 percent between 1965 and 1995. In addition, this rate is the lowest provincial rate in Canada which in turn

is one of the lowest in the world (Millar, 1996). However, significant regional difference in health status, including infant mortality, strongly indicate that there is still potential to further improve infant health status through the reduction of regional inequalities. Causal factors, that is potential determinants, and their interactions which are responsible for this regional disparity must be identified. Such a recognition will definitely assist policy design, as decision makers strive to achieve the WHO's goal of "Health for All by Year 2,000" (World Health Organization, 1985). In fact, 1997 Provincial Health Officer's Annual Reports of British Columbia, which was dedicated to "the Health and Well-being of British Columbia's Children", one of the actions related to health status of British Columbia's children was to develop strategies to address the factors underlying the inequities in children's health status (Millar, 1998).

An investigation of regional inequalities in infant mortality along with an analysis of time trends in infant deaths would permit a more complete modelling of infant health status in the province. This in turn would permit insights into the efficiency of existing health policies related to population health management. There are as yet no effective models of geographical inequalities in infant mortality, nor how these alter over time because of methodological difficulties. Assessments of infant mortality have always focused on changes in a single mortality rate over time (Singh & Yu, 1995; Centers for Disease Control, 1991; British Columbia Provincial Health Officer, 1996). An index which can appropriately describe dynamic change in geographical inequality in infant mortality over time needs to be developed. Some recent publications have addressed this issue (Wing et al, 1990; Lardelli et al, 1991,

Wagstaff, Paci, van Doorfslaer, 1991, Vázquez-Vizoso et.al, 1993).

The majority of current studies addressing geographical inequalities in infant mortality have produced general descriptive analyses, illustrating such factors as infant mortality in different social classes, income groups, and/or regions. Such results are relatively ineffective in stimulating policies to reduce disparity. To reduce inequalities, descriptions of it are not enough. What is needed is a clear understanding of their causes. The ecological and etiological factors which determine regional inequality in infant mortality, therefore, must be studied and identified. Region-specific interrelationships between etiological causes and ecological correlates must be clarified so that decision-makers and policy planners can most effectively utilize limited economic resources to reduce infant mortality. This is another area of British Columbian infant mortality requiring further study, the relationships or pathways linking etiological causes and ecological determinants that result in geographical inequality in infant mortality.

CHAPTER FIVE

DATA AND METHODOLOGY

The methodology used in this thesis involves several steps, the first of which requires analysis and mapping of the infant mortality rate at the Health Unit scale. Step two focuses on a more detailed investigation of regional variation in such mortality rates over time. The third step is a study of the etiological profiles of infant deaths using multiple correlation and regression analyses to determine the risk that can be attributed to etiological factors at the Health Unit scale. The fourth step involves the development of an ecological profile of infant mortality at the Health Unit level. Finally, emphasis is placed on exploring possible interactions between the etiological causes and ecological determinants. As a result, recommendations are made that, if implemented, may reduce geographical inequity in British Columbia's infant mortality.

5.1 Research Design

Scale is a key factor in the study of regional inequalities of health. The selection of geographical scale should be governed by a series of factors including the objectives of the study, the availability of data and the nature of the events under study. Theoretically, a research scale should be chosen that ensures that all etiological and ecological characteristics of the infant population are evenly distributed or comparable within specific regions, yet are significantly different between them. The goal is to subdivide the data so that they are homogeneous

within regions and heterogeneous between them. Unfortunately such geographical areas do not exist in British Columbia. Even if they did, they would not be practical for use in this study. Therefore, the actual selection of geographical scale must be largely determined by those factors previously mentioned, including the objectives of the study, and the availability and limitations of the data. As a consequence, Health Units rather than Local Health Areas were chosen for study for the following reasons. Firstly, the Health Unit has been the main local health administrative unit of this province. Currently, there are a total of 21 Health Units. Each of them has a Medical Health Officer (MHO) and a Chief Environmental Health Officer (CEHO) who are the key administrators of population health within each Health Unit. Most regional publications and reports compiled which discuss local population health status have been based on Health Units. Secondly, the relative large physical size of each Health Unit generally ensures that they possess varied physical, socio-economic, and demographic characteristics. These differ markedly from region to region, so enhancing the power of statistical analyses used to examine relationships between etiological and ecological factors and spatial infant mortality variations. Thirdly, the population size, especially of the infant population, at the Health Unit scale is likely to be suited to the requirement of a series of statistical analyses performed against these data. Fourthly, certain data including information specific to each birth and data for ecological profiles are only available at the Health Unit level. Fifthly, there are currently no other appropriate geographical units more suitable for use in this analysis. This is because the Local Health Area (LHA) scale

is too small to permit the correct use of certain statistical analyses. For instance, total annual infant deaths in some LHAs are zero. This prevents the use of LHA data in stable statistical analyses. For these five reasons, therefore, the Health Unit is used as the basic geographical unit throughout this study.

5.1.1 Descriptive Statistics of Infant Mortality Rate by Health Unit for British Columbia, 1987 to 1996

Infant mortality rate (including early neonatal, neonatal, post-neonatal, and total infant mortality rates) were analysed for each year, for the period 1987 to 1996, for British Columbia. The major objective here was to illustrate both time trends and the geographical distribution patterns of infant mortality rates at the Health Unit level. Differences in the four infant mortality indicators were then investigated, and regional variations in them, over time, presented.

5.1.2 Time Trends of Regional Variations of Infant Mortality Rate for British Columbia, 1981 to 1996

The Vital Statistics Agency of British Columbia's annual report of 1995 presented a time trend for the provincial infant mortality rate for the past 30 years (Cronin & Danderfer, 1996). A very clear decline is obvious for this time period, especially for the rates of early neonatal and neonatal mortality. Post-neonatal mortality rate declined far more slowly. However, such trends in provincial rates tend to mask regional disparities in infant mortality at the Health Unit scale, since the temporal change in infant mortality rate in each Health Unit may not have followed

the provincial trend. As a result, time trends of individual infant mortality rates, calculated at the Health Region scale, provide a clearer view of infant health status in the province.

The statistic best suited for the exploration of annual regional inequality in infant mortality rate is the weighted coefficient of variation (WCV). According to Vazquez-Vizoso and colleagues (1993), the annual regional variation of infant mortality, where it exists, can be derived from the sum of the square root of the difference in infant mortality rates between each Health Unit and the provincial norm for each year. This coefficient is the ratio of the variation over its standard deviation, allowing comparison amongst all kinds of variables with different units. Taking sample size (actual number of live births in each region) into account, the annual number of live births in each region is used to weigh the standard deviation of infant mortality. As a result, the coefficient developed summarizes annual regional differences in infant mortality rate, yet takes regional sample size into account. The required formula is as follows:

$$\text{W.S.D.} = \sqrt{\frac{\Sigma (W_i \times (R_i - R)^2)}{\Sigma W_i}}$$

$$W.C.V. = \frac{W.S.D.}{R}$$

Where:
- W_i: Number of live births at Health Region i in a year,
- R_i: Infant mortality rate at Health Region i in a year,
- R: Provincial infant mortality in a year,
- W.S.D.: Weighted standard deviation of regional variation of infant mortality rate in a year,
- W.C.V.: Weighted coefficient of variation.

The weighted coefficients of variation for early neonatal, neonatal, postneonatal, and infant mortality rates, for the period 1987 to 1996, were calculated at the Health Unit level. An examination of time trends of provincial infant mortality and associated regional differences then indicated whether the declining trends in provincial infant mortality rate during the last 30 years were accompanied by a reduction in regional inequalities, or whether the trends of both phenomena were completely different. While the declining trend seen in the infant mortality rate in the province as a whole indicates an overall improvement in infant health status, it does not necessarily imply that such a decline has been shared equally by all Health Units. Indeed, several recently published papers have found that regional inequality in infant mortality has continued and even increased, in spite of the significant provincial reduction (British Columbia Provincial Health Officer Report, 1996).

5.1.3 Association Analysis between Infant Mortality and Etiological Factors

In this step, etiological factors of infant mortality were firstly identified. The

etiological factors were chosen based on two conditions. Firstly, a significant number of publications have identified such variables as etiological or causal factors in infant mortality. Secondly, the data is available for this study. As a result five factors have been chosen, low birth weight, pre-term birth, teenage birth, births with perinatal conditions, and births with maternal complications.

After these factors were chosen, the prevalence rates of these variables for each of the Health Units were developed. These rates were then compared with the three infant mortality rates by multiple correlation and regression analyses, in order to identify their associations with infant mortality at the Health Unit scale.

5.1.4 Ecological Analysis of Infant Mortality Rate by Health Unit

As previously discussed, the ecological study of health differs from that of etiology in that it emphasizes patterns of population health and their relationships to the environment. Such relationships are not necessarily cause-effect. However, they summarize and provide an integrated overview of how people interact with their environments. Therefore, during this step research was focussed on Health Regions. The ecological factors investigated in this area based analysis included:

1) Socio-economic factors, such as:

- Education;
- Ethnic population;
- Native indian population;
- Employment status;
- Income (average individual income);
- Family structure and status (1 or 2 parents, number of children).

2) Health services factors, including:

- Acute and extended health care beds per 1,000 population;
- Number of general practitioners per 1,000 population;
- Child care capacity;
- Number of registered nurses;
- Average transported distance to health care facilities.

3) Population health profile factors, including:

- Male life expectancy;
- Female life expectancy;
- Crude birth rate;
- Crude death rate.

Obviously the factors to be studied were selected on the assumption (based on a literature review) that they impacted on infant health status in a variety of ways. For instance, low level education may be related to lower maternal awareness of infant care; low income may result in less regular visits to a physician for necessary perinatal and infant care; or bad weather conditions (heavy snowfall) may cause parents to be reluctant to make essential visits to physicians. Such factors may overlap in their contributions to the health of an infant population. This step of the research project aimed to classify these factors into clusters, based upon the magnitude of their contribution to regional differences in infant mortality.

Multiple correlation and factor analyses were then performed on these ecological factors in order to examine possible correlations between them and infant mortality. Cluster analysis was also performed based on the ranking of these ecological factors amongst the 21 Health Units, to detect any clustering of Health

Units in terms of the similarity of their ecological environment. Based on these new clusters, mortality analysis was then performed, in order to examine associations between patterns of deaths and new areal clusters. Factor analysis was used to extract the significant components from the original ecological factors since they are inter-correlated with each other. The extracted common factors were then analysed in relation to mortality indices to determine whether or not there is a significant association between these factors and infant mortality rates at the Health Unit scale.

5.1.5 The Effects on Infant Mortality of Interactions between Ecological and Etiological Factors

Based on the analytical steps already described, important ecological and etiological factors contributing to infant deaths were identified. A canonical correlation analysis was then performed on ecological and etiological variable groups to examine association between them. Canonical correlation coefficients indicate association between two groups correlated by a different variable, according to their ability to explain variance in infant mortality. It was, therefore, possible to identify pathways connecting ecological factors to infant death through etiological factors.

5.2 Data

The data required to conduct this study were of two types. Infant deaths records were essential, as was ecological information at the Local Health Area scale. Infant death data was derived from the birth registration database of the Vital

Statistics Agency of British Columbia. This database is one of British Columbia's most complete, systematic, comprehensive, high-quality health information sources. It is complete because it covers every birth and infant death that occurs in the province. It is systematic because it is a routinely-collected and population-based birth registration system. It is comprehensive because it contains information specific to each birth and infant death varying from genetic and biomedical to demographic characteristics. It is of high-quality because important clinical information such as diagnosis for cause of death, birth weight, and gestational weeks, are collected and coded as accurately as possible.

The second type of data needed for this study was Local Health Area and Health Unit based ecological information. The main source of this type of data is the Health Planning Database (HPDB database), and other Ministries' publications, created and maintained by the Ministry of Health of British Columbia. Relevant information in this data bank includes:

- Demographic Information
  - . percent under 15 population;
  - . percent 65 and over population;
  - . percent 80 and over population;
  - . percent aboriginal population;
  - . percent ethnic population;

- Health Status Information
  - . crude birth rate;
  - . crude death rate;
  - . median age at death;
  - . life expectancy;

- Family and Children

. percent of families with husband and wife;
. percent of families with lone parent;
. percent of children in two parent families;
. percent of children in lone families;
. average number of children;

- Occupation Classification based on households;

- Income Information

. average individual income;
. percentage of the population receiving unemployment insurance;

- Education

. grade 12 enrollment rate;
. grade 12 graduation rate;

- Health Care Resources

. hospital resources - acute and extended care beds;
. continuing care beds and facilities;
. capacity adult and child care;
. full-time-equivalent general practitioners and specialists;
. registered nurses;
. health care workers.

5.3 Statistical Methodology

Frequency and association analyses were the main statistical methods used in this study. As mentioned previously multiple correlation and regression, factor analysis, and canonical correlation were applied to the data.

Group correlation analyses, between etiological and ecological factors, also were used to explore how ecological factors impacted on infant mortality through etiological factors.

CHAPTER SIX

RESULTS

6.1 History of Infant Mortality in British Columbia

The British Columbia Vital Statistics Agency has published official Annual Reports since the late nineteenth century. The most recent volume, the 127th Annual Report, was that published for the year 1997 (Danderfer, 1998). These annual reports provided most of the information that was used in the following tables and figures which illustrate historical changes and trends in infant mortality, in British Columbia. These data indicate that British Columbian infant mortality rates, including neonatal and post-neonatal mortality rates, have been gradually and consistently declining at least since 1965 (Danderfer, 1997; Danderfer, 1998).

Table 1 summarizes data on annual live births, neonatal, post-neonatal, and infant deaths in British Columbia, from 1965 to 1996. In order to illustrate graphically historical trends amongst these rates, Figures 1, 2, and 3 were plotted to display the annual mortality rates of these three infant death indicators for the last 30 years. The information provided in these table and figures shows that the total number of live births during last three decades has increased by more than 12,000, from 33,669 in 1965 to 45,881 in 1996. In addition, these increases have occurred consistently every year. Infant mortality, however, has shown a consistent declining trend during the same period. For example, the number of infant deaths in 1996 was less than one third that for 1965. Such opposing trends in changes in live births and infant deaths have resulted in remarkable reductions in the rates of infant, neonatal, and post-neonatal mortality. These declines reflect significant progress

TABLE 1
History of Infant Related Mortality in British Columbia
Neonatal, Postneonatal, and Infant Mortality Rate (1/1,000 Live Births) from 1965 to 1996

Calendar Year	Total Live Births	Infant Deaths	Neonatal Deaths	Post-Neonatal Deaths	Infant Death Rate	Neonatal Death Rate	Post-Neonatal Death Rate	Neonatal/ Postneonatal Death Ratio
1965	33,669	683	453	227	20.29	13.45	6.74	2.00
1966	32,502	761	494	263	23.41	15.20	8.09	1.88
1967	32,899	689	470	218	20.94	14.29	6.63	2.16
1968	33,687	656	438	214	19.47	13.00	6.35	2.05
1969	35,383	573	374	199	16.19	10.57	5.62	1.88
1970	36,861	611	416	193	16.58	11.29	5.24	2.16
1971	34,852	635	450	185	18.22	12.91	5.31	2.43
1972	34,563	569	373	195	16.46	10.79	5.64	1.91
1973	34,352	551	363	185	16.04	10.57	5.39	1.96
1974	35,450	546	348	196	15.40	9.82	5.53	1.78
1975	36,281	491	321	169	13.53	8.85	4.66	1.90
1976	35,848	478	324	152	13.33	9.04	4.24	2.13
1977	36,691	476	276	200	12.97	7.52	5.45	1.38
1978	37,231	464	286	178	12.46	7.68	4.78	1.61
1979	38,432	406	239	167	10.56	6.22	4.35	1.43
1980	40,104	421	235	186	10.50	5.86	4.64	1.26
1981	41,679	402	259	140	9.65	6.21	3.36	1.85
1982	42,942	401	251	150	9.34	5.85	3.49	1.67
1983	43,047	359	212	145	8.34	4.92	3.37	1.46
1984	44,040	356	205	150	8.08	4.65	3.41	1.37
1985	42,989	331	198	133	7.70	4.61	3.09	1.49
1986	41,845	343	196	147	8.20	4.68	3.51	1.33
1987	41,655	353	197	156	8.47	4.73	3.75	1.26
1988	42,913	356	219	137	8.30	5.10	3.19	1.60
1989	43,585	353	215	138	8.10	4.93	3.17	1.56
1990	45,333	331	225	106	7.30	4.96	2.34	2.12
1991	45,312	287	161	126	6.33	3.55	2.78	1.28
1992	45,712	273	171	102	5.97	3.74	2.23	1.68
1993	45,922	251	140	111	5.47	3.05	2.42	1.26
1994	46,798	291	199	92	6.22	4.25	1.97	2.16
1995	46,762	276	179	97	5.90	3.83	2.07	1.85
1996	45,881	227	160	67	4.95	3.49	1.46	2.39

Footnotes:

1. The primary source of the information is the BC Vital Statistics Agency Annual Reports.
2. Infant deaths are defined as the number of live births who die within 1 year after delivered.
3. Neonatal deaths are defined as the number of live births who die within 28 days after delivered.
4. Postneonatal deaths are defined as the number of live births who die between 28 days to 1 year after delivered.
5. Infant, neonatal, and postneonatal mortality rates are all based on number of deaths per 1,000 live births.

Figure 1: Historical Trend of infant Mortality Rate (per 1,000 Live Births)
From 1965 to 1996, British Columbia

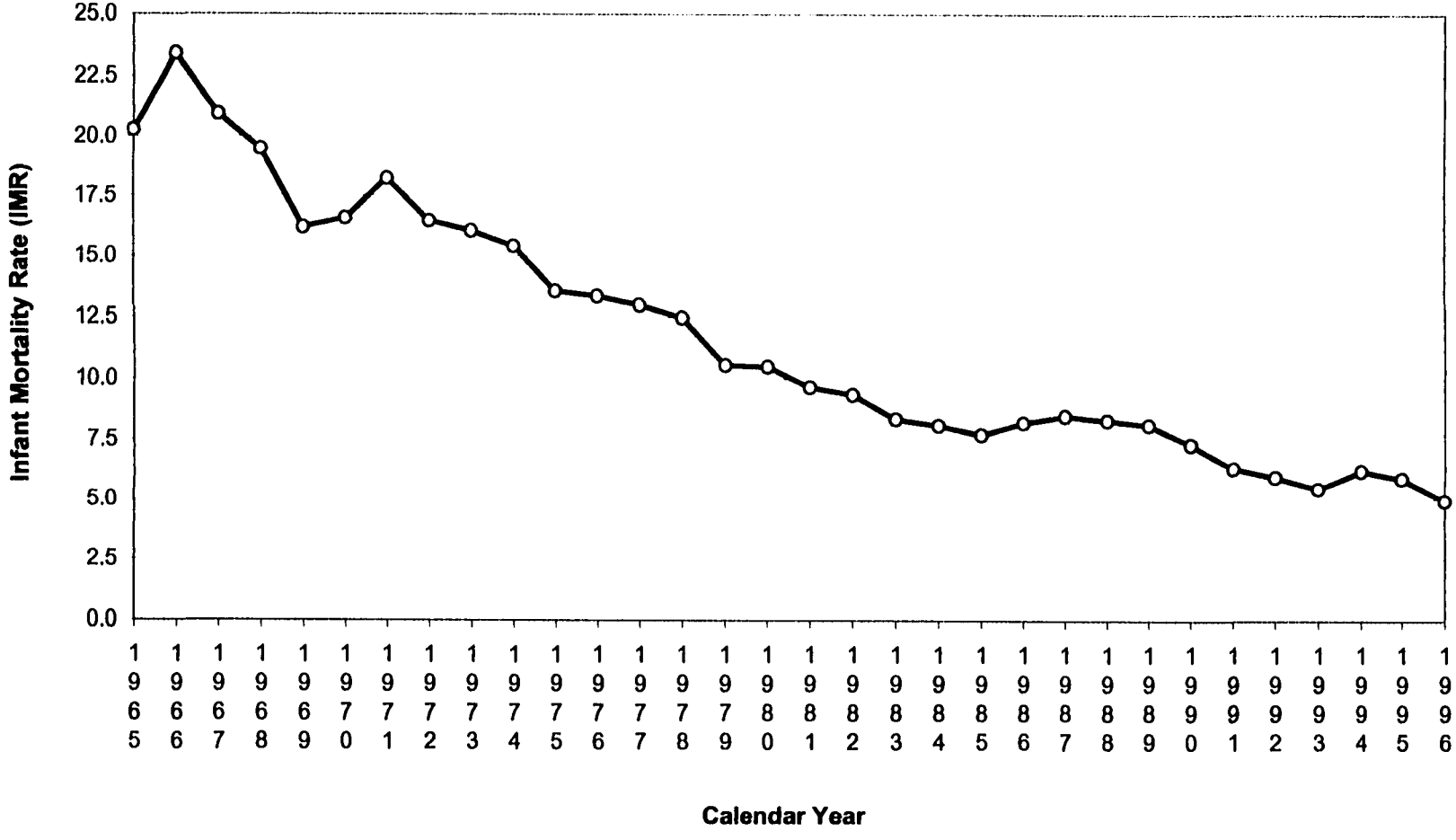


**Figure 2: Historical Trend of Neonatal Mortality Rate (per 1,000 Live Births)
From 1965 to 1996, British Columbia**

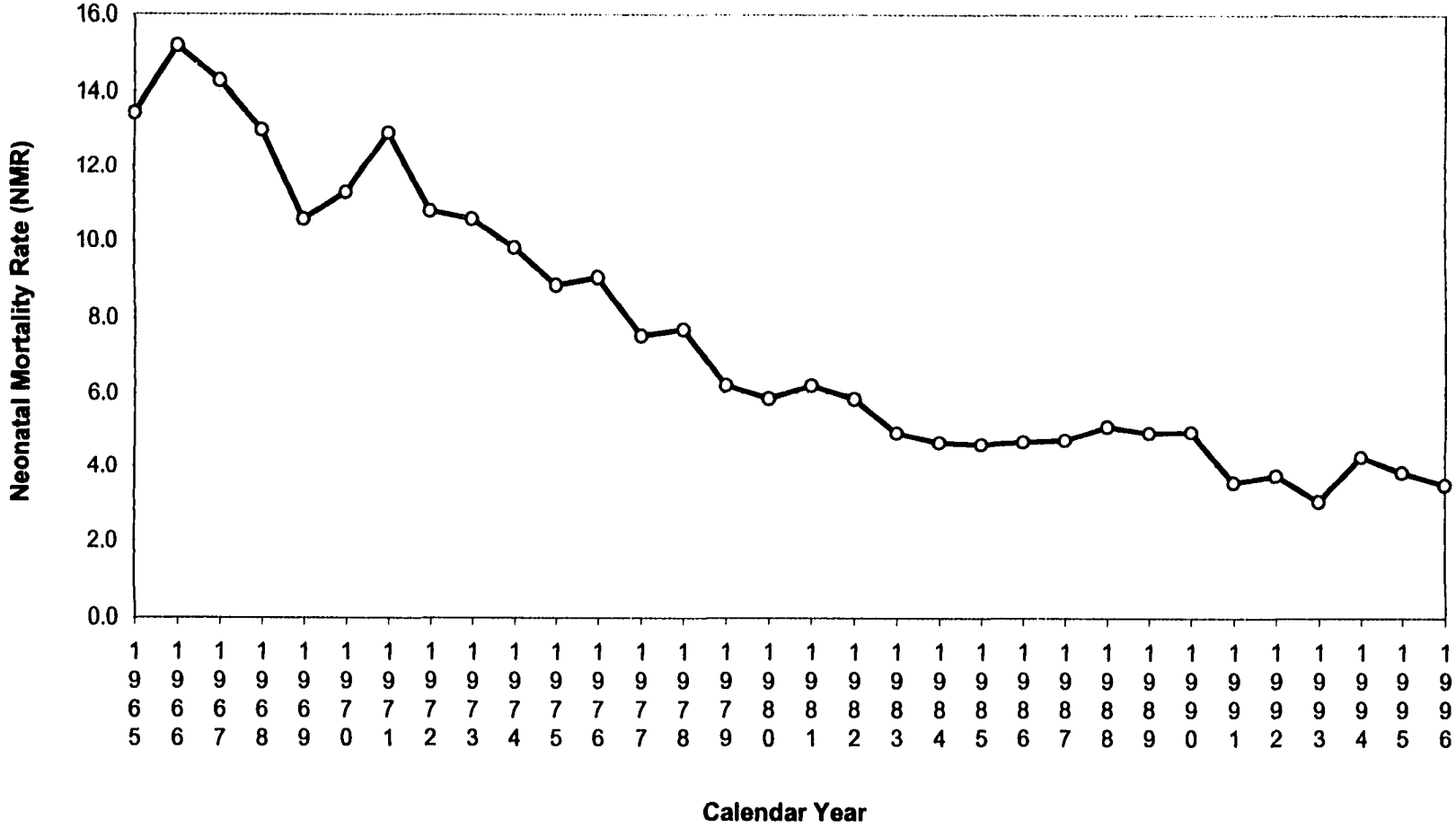
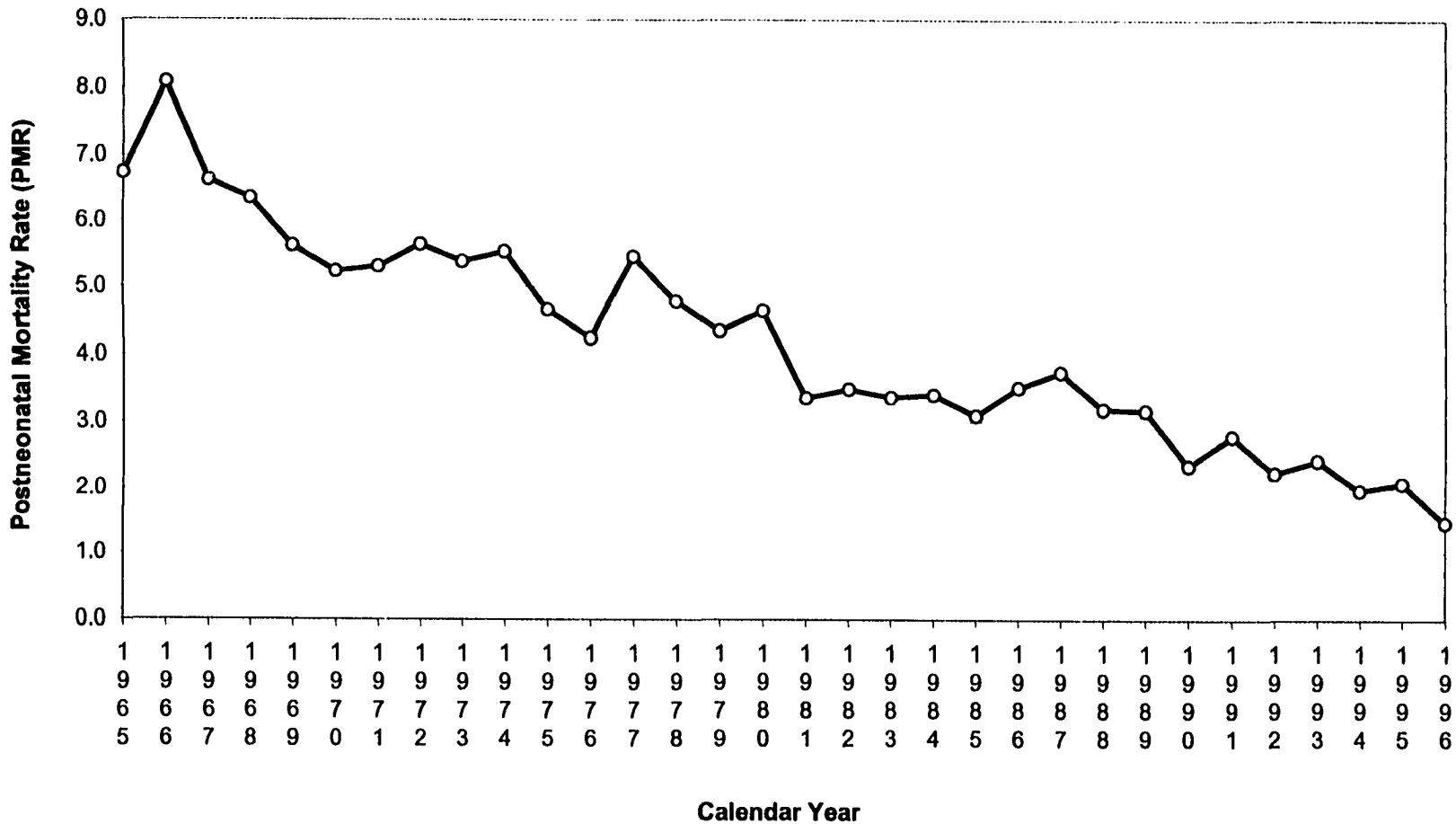


Figure 3: Historical Trend of Postneonatal Mortality Rate (per 1,000 Live Births) From 1965 to 1996, British Columbia



in improving infant health status, in British Columbia, during the last 30 years. To illustrate, the infant mortality rate, as shown in Table 1, decreased from more than 20 infant deaths per 1,000 live births in 1965 to 5 deaths in 1996. The neonatal mortality rate dropped from more than 13 neonatal deaths per 1,000 live births in 1965 to less than 4 deaths in 1996 (Table 1). A similar decline is obvious in the post-neonatal mortality rate, which has been reduced from 7 deaths per 1,000 live births in 1965 to less than 2 deaths in 1996 (Table 1). British Columbia's current infant, neonate, and post-neonate mortality rates appear to be the lowest in Canada, while the national rate is the world's third lowest, only Japan and Sweden have more depressed rates (Millar, 1995).

Infant mortality is a combination of both neonatal and post-neonatal mortality. Amongst the total 227 infant deaths that took place in the province in 1996, for example, 160 deaths (neonatal deaths) occurred within 28 days of delivery while only 67 deaths (post-neonatal deaths) took place between 29 to 365 days. In that year, therefore, neonatal mortality rate was responsible for 70 percent of infant mortality. This relationship occurred consistently throughout the past 30 years. As a result the reduction in infant mortality as a whole, during this period, was caused largely by a significant decrease in neonatal mortality. Nevertheless, post-neonatal mortality also declined, so that its percentage contribution to the total infant death rate remained relatively constant.

Although fluctuations have occurred in annual neonatal, postneonatal, and infant mortality rates during the past 30 years (Figures 1, 2 and 3), historical trends have been significantly downwards. As shown in Table 2 and Figures 4, 5, and 6,

TABLE 2
Time Trends of Infant Related Mortality in British Columbia
Neonatal, Postneonatal, and Infant Mortality Rate (1/1,000 Live Births) from 1965 to 1996
(Based on the Vital Statistics Annual Report 1996)

Year	Total Live Births	Infant Death Rate	Infant Reg. IMR*	95% Lower Limit**	95% Upper Limit**	Neonatal Death Rate	Neonatal Reg. NMR*	95% Lower Limit**	95% Upper Limit**	Postneonatal Death Rate	Postneonatal Reg. PMR*	95% Lower Limit**	95% Upper Limit**
1965	33,669	20.29	19.96	18.84	21.08	13.45	13.16	12.22	14.11	6.74	6.74	6.37	7.11
1966	32,502	23.41	19.43	18.26	20.61	15.20	12.80	11.80	13.79	8.09	6.57	6.19	6.96
1967	32,899	20.94	18.90	17.67	20.13	14.29	12.43	11.39	13.47	6.63	6.41	6.00	6.82
1968	33,687	19.47	18.37	17.08	19.66	13.00	12.06	10.98	13.15	6.35	6.25	5.82	6.67
1969	35,383	16.19	17.83	16.49	19.18	10.57	11.70	10.56	12.83	5.62	6.08	5.64	6.52
1970	36,861	16.58	17.30	15.90	18.70	11.29	11.33	10.15	12.52	5.24	5.92	5.46	6.38
1971	34,852	18.22	16.77	15.31	18.23	12.91	10.97	9.73	12.20	5.31	5.75	5.27	6.23
1972	34,563	16.46	16.24	14.72	17.75	10.79	10.60	9.32	11.88	5.64	5.59	5.09	6.09
1973	34,352	16.04	15.70	14.13	17.27	10.57	10.23	8.91	11.56	5.39	5.43	4.91	5.94
1974	35,450	15.40	15.17	13.54	16.80	9.82	9.87	8.49	11.24	5.53	5.26	4.73	5.80
1975	36,281	13.53	14.64	12.96	16.32	8.85	9.50	8.08	10.92	4.66	5.10	4.54	5.65
1976	35,848	13.33	14.11	12.37	15.84	9.04	9.14	7.67	10.60	4.24	4.93	4.36	5.51
1977	36,691	12.97	13.57	11.78	15.37	7.52	8.77	7.25	10.29	5.45	4.77	4.18	5.36
1978	37,231	12.46	13.04	11.19	14.89	7.68	8.40	6.84	9.97	4.78	4.61	4.00	5.22
1979	38,432	10.56	12.51	10.60	14.41	6.22	8.04	6.43	9.65	4.35	4.44	3.81	5.07
1980	40,104	10.50	11.98	10.01	13.94	5.86	7.67	6.01	9.33	4.64	4.28	3.63	4.92
1981	41,679	9.65	11.44	9.42	13.46	6.21	7.31	5.60	9.01	3.36	4.11	3.45	4.78
1982	42,942	9.34	10.91	8.83	12.99	5.85	6.94	5.19	8.69	3.49	3.95	3.27	4.63
1983	43,047	8.34	10.38	8.24	12.51	4.92	6.57	4.77	8.37	3.37	3.79	3.08	4.49
1984	44,040	8.08	9.85	7.66	12.03	4.65	6.21	4.36	8.06	3.41	3.62	2.90	4.34
1985	42,989	7.70	9.31	7.07	11.56	4.61	5.84	3.95	7.74	3.09	3.46	2.72	4.20
1986	41,845	8.20	8.78	6.48	11.08	4.68	5.48	3.53	7.42	3.51	3.29	2.54	4.05
1987	41,655	8.47	8.25	5.89	10.60	4.73	5.11	3.12	7.10	3.75	3.13	2.35	3.91
1988	42,913	8.30	7.71	5.30	10.13	5.10	4.74	2.71	6.78	3.19	2.97	2.17	3.76
1989	43,585	8.10	7.18	4.71	9.65	4.93	4.38	2.29	6.46	3.17	2.80	1.99	3.61
1990	45,333	7.30	6.65	4.12	9.17	4.96	4.01	1.88	6.15	2.34	2.64	1.81	3.47
1991	45,312	6.33	6.12	3.53	8.70	3.55	3.65	1.46	5.83	2.78	2.47	1.62	3.32
1992	45,712	5.97	5.58	2.94	8.22	3.74	3.28	1.05	5.51	2.23	2.31	1.44	3.18
1993	45,922	5.47	5.05	2.36	7.75	3.05	2.91	0.64	5.19	2.42	2.15	1.26	3.03
1994	46,798	6.22	4.52	1.77	7.27	4.25	2.55	0.22	4.87	1.97	1.98	1.08	2.89
1995	46,762	5.90	3.99	1.18	6.79	3.83	2.18	-0.19	4.55	2.07	1.82	0.89	2.74
1996	45,881	4.95	3.45	0.59	6.32	3.49	1.82	-0.60	4.23	1.46	1.65	0.71	2.60

Footnotes:

1. The primary source of the inform ation is the BC Vital Statistics A gency Annual Reports.
2. Infant deaths are defined as the num ber of live births who die within 1 y ear after delivered.
3. Neonatal deaths are defined as the num ber of live births who die within 28 day s after delivered.
4. Postneonatal deaths are defined as the num ber of live births who die between 28 day s to 1 year after delivered.
5. Infant, neonatal, and postneonatal m ortality rates are all based on num ber of deaths per 1,000 live births.

* *IMR, NMR, and PMR stand for infant, neonatal, and post-neonatal mortality rate, respectively.*

** *The regressed IMR, NMR, and PMR were developed based on the regression analyses between IMR, NMR, and PMR against year respectively.*

Figure 4: Annual Reduction of Infant Mortality Rate (IMR, per 1,000 Live Births) From 1965 to 1996, British Columbia

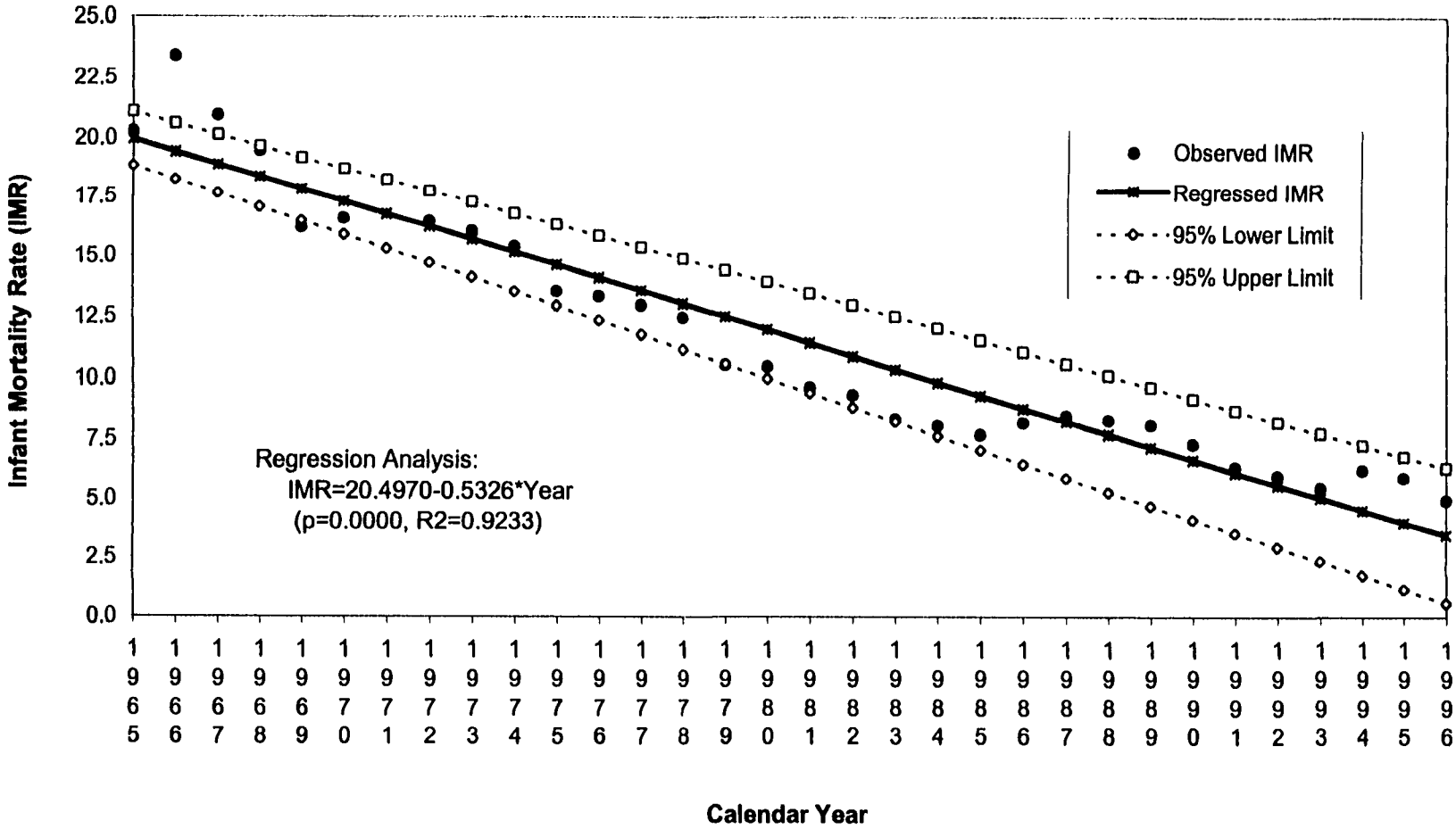


Figure 5: Annual Reduction of Neonatal Mortality Rate (per 1,000 Live Births) From 1965 to 1996, British Columbia

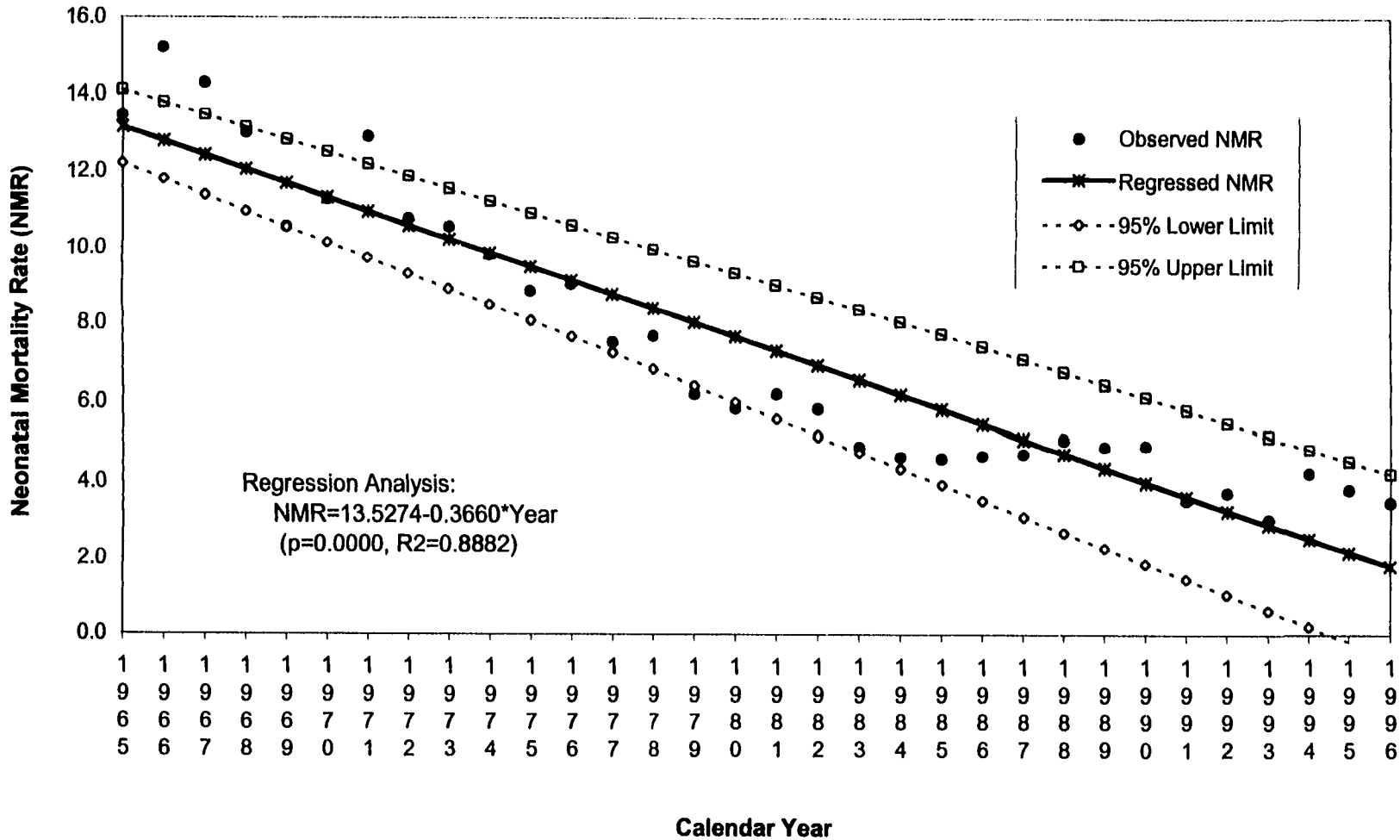
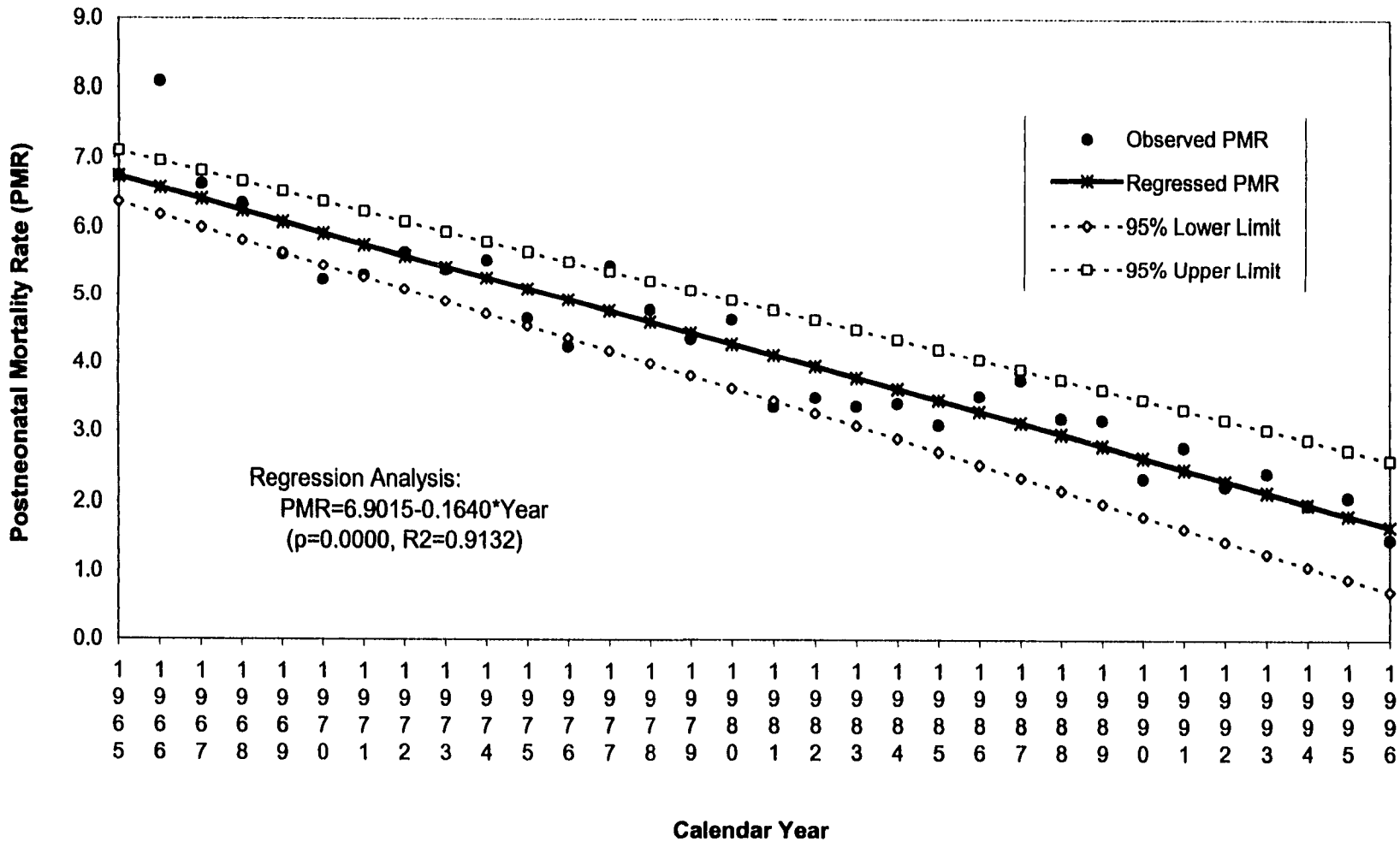


Figure 6: Annual Reduction of Postneonatal Mortality Rate (per 1,000 Live Births) From 1965 to 1996, British Columbia



these three mortality rates show close linear relationships with a time variable, representing the period 1965 to 1996. To illustrate further, the average reduction in infant mortality rate, as measured by the number of deaths per 10,000 live births, was estimated to be five deaths per 10,000 live births. That is, on average, there were about five infants, every year, in each 10,000 births, whose lives were saved during the past 30 years, resulting in a total decrease in mortality of some 81 percent (Table 3). This linear relationship is statistically significant, and the R^2 value, which measures the degree of determination, was about 92 percent, indicating a well-fitted linear trend in the change of infant mortality rate over the past 30 years.

For neonatal deaths, a similar relationship also was found. The annual decrease in the neonatal morality rate was estimated to have been nearly four neonates for every 10,000 newborns each year, representing an 84 percent total reduction in neonatal mortality during the last 30 years (Table 3). This relationship is again statistically significant, with an R^2 value of nearly 90 percent.

The annual reduction in post-neonatal death followed a similar pattern, although the decrease was lower. The average annual reduction in this mortality rate was estimated to have been approximately two infants in every 10,000 newborns, who would have died, but who survived every year from 1965 to 1996, accounting for a 74 percent total reduction in this mortality rate during this time period (Table 3). This linear relationship also was statistically significant, with an R^2 value of about 91 percent. Table 3 summarizes statistics derived from a linear regression analysis between these three infant mortality rates and the year of death.

Infant health status in British Columbia as a whole has dramatically improved

TABLE 3:
Regression Analysis:
Temporal Trends of Infant Mortality Rates (per 1,000 Live Births)
From 1965 to 1996, British Columbia

Regression Statistics	Infant Mortality Rate	Neonatal Mortality Rate	Post-neonatal Mortality Rate
Total Observations	32	32	32
R^2 Value	0.9233	0.8882	0.9132
Regression Significance	p<0.0001	p<0.0001	p<0.0001
α Value (Intercept)	20.4970	13.5274	6.9015
p Value for α	<0.0001	<0.0001	<0.0001
α Value: 95% Lower Limit	19.4335	12.6288	6.5513
α Value: 95% Upper Limit	21.5606	14.4260	7.2516
β Value (Regression Coefficient)	-0.5326	-0.3660	-0.1640
p Value for β	<0.0001	<0.0001	<0.0001
β Value: 95% Lower Limit	-0.5889	-0.4135	-0.1825
β Value: 95% Upper Limit	-0.4764	-0.3185	-0.1455
Average Decreases in Mortality Rate (per Year)	About 5 deaths per 10,000 live births	About 4 deaths per 10,000 live births	About 2 deaths per 10,000 live births
Percent of Total Reduction in Mortality Rate for Last 30 Years	80.55%	83.87%	73.67%

Notes:

1. The regression analyses were performed between infant mortality rates and number of year, starting from 1965 (as value 1). Therefore, the dependent variable was infant mortality rates while the independent variable is the number of year.
2. The annual average decreases in mortality rates were estimated by the value of β, the slope of the regression equation.
3. Percent of total reduction in mortality rate for last 30 years was estimated by $31*\beta/\alpha$. Here the β value is annual average decrease, while the α value is the base morality at the beginning of this 32 years.

during the past 30 years. This can be seen clearly from the steady, consistently declining trends of the three mortality measures previously described. BC infant mortality rates have generally been lower than the Canadian national average, indicating a healthier infant population in this province than elsewhere in the country (Danderfer, 1996; Millar, 1996). In fact, British Columbia the lowest infant mortality rates in Canada which has one of the lowest in the world (Millar, 1996). This is largely because infant mortality rates in the province have fallen to approximately one fourth of those experienced thirty years ago. Although the underlying causes or contributing factors, responsible for this decline are multi-factoral, it has been suggested that decreases in early neonatal mortality (newborns who die within 7 days of delivery), which is a significant part of the neonatal mortality rate, may be a major contributor to the overall decline in infant mortality rate experience in the past 30 years (Danderfer, 1996). From an ecological point of view, economic development, improvement in living conditions, availability of universal health care, and advances in medical technology used in intensive care services, as well as better education opportunities may together have contributed to this decline (Yankauer, 1990).

6.2 Geographical Variations of Infant Mortality Rates in British Columbia

Health inequality is a relatively new public health topic which has been widely addressed only since mid-1980s. In 1984, the World Health Organization (WHO) included 38 targets for Europe in its policy document "Health for All by the Year 2000" (HFA-2000). Target 1 of HFA-2000 was that "by the year 2000, the actual

differences in health status between countries, and between groups within countries, should be reduced by at least 20 per cent, by improving the level of health of disadvantaged nations and groups" (World Health Organization, 1985). As a consequence, researchers have been focussing their attention on health status inequality between countries and on internal inequalities between regions and population groups at the national level. Health inequality is essentially the existing health differences within and between nations, communities, and populations. Patterns of infant death, for example, derived from comparisons between and/or amongst populations may reflect complex socio-economic and technological factors. They are fundamentally outcomes resulting from combinations of poverty/wealth, lifestyle choices and medical technologies which are fluid, and may be moving in different directions. As a result of current trends, some of these factors may even result in wider inter-country and intra-country differentials in infant mortality (Illsley, 1990).

In British Columbia, the Annual Reports of Vital Statistics Agency, and the Provincial Health Officer's Annual Report, have both discussed regional differentials in infant mortality rates (Danderfer, 1996; Millar, 1996). However, none of these, or other associated studies, have addressed time trends in such regional variations in infant mortality in British Columbia. Whether or not existing regional inequalities in infant health status have followed a similar pattern of change as that of the mortality rate as a whole. Such a study might be expected to reflect the efficiency and effectiveness of health policies and strategies, as well as practical interventions designed to correct regional differentials in infant health status. In summary,

although patterns of geographical differentials in infant mortality, within British Columbia have already been studied, relatively little progress may have been made in correcting them. This thesis investigates whether geographical variations in infant mortality rates have been reduced, mirroring the remarkable declining trend experienced by infant mortality rates as a whole.

Table 4 and Maps 1, 2, 3 presented geographical distribution patterns of three infant death indicators, at the Health Unit level, for the time period from 1987 to 1996. During this ten year period, there were a total of 449,817 live births. Unfortunately, 2,962 of these infants died within one year of birth, and 1,824 of these within 28 days (neonatal deaths). The remaining 1,138 deaths occurred between 29 and 365 days of birth (post-neonatal deaths). Together, these deaths represent an infant mortality rate of 7 per 1,000 live births, composed of a neonatal mortality rate of 4 per 1,000 live births, and a post-neonatal mortality rate of nearly 3 per 1,000 live births. Since these rates were derived from 10-year aggregated data, they can reasonably be used to represent the average infant, neonatal, and post-neonatal mortality rates for British Columbia as a whole, over the last 10 years.

From these data, it can be seen that all three morality rates differed greatly at the Health Unit level. Five of these 21 Units, including Central Vancouver Island (IMR=8.74 per 1,000 live births), Cariboo (IMR=9.20 per 1,000 live births), Skeena (IMR=8.18 per 1,000 live births), Northern Interior (IMR=9.47 per 1,000 live births), and Vancouver (IMR=7.43 per 1,000 live births), experienced significantly higher infant mortality rates than the provincial mean, while four, including Upper Fraser Valley (IMR=5.27 per 1,000 live births), Boundary (IMR=5.36 per 1,000 live births),

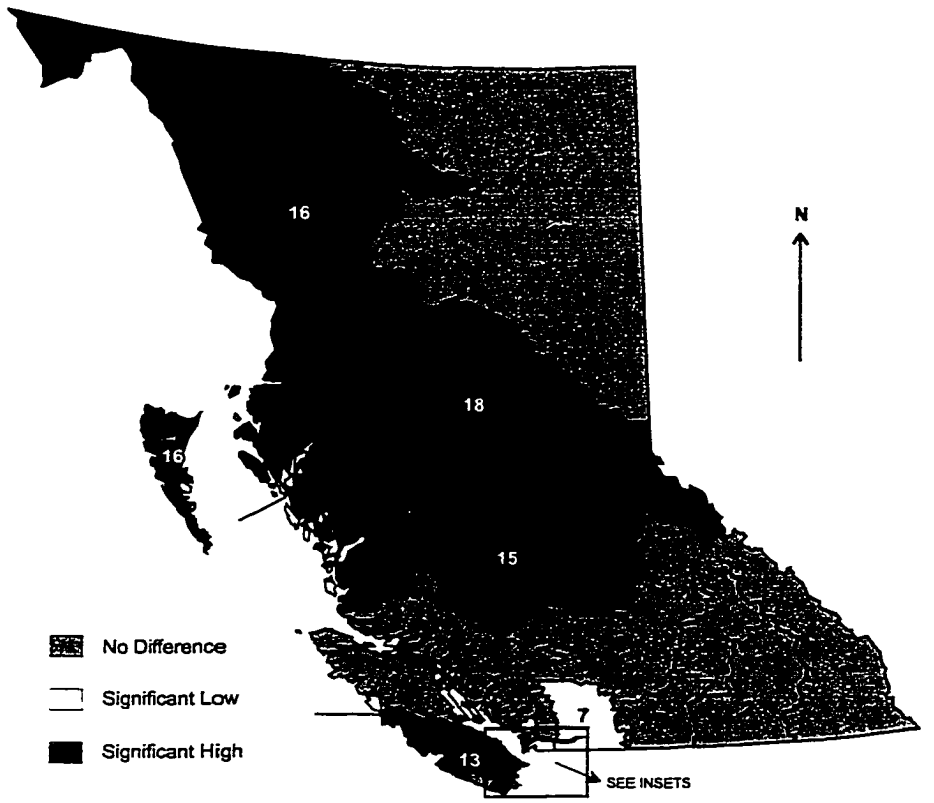
TABLE 4:
Geographical Distribution of Infant Related Mortality Rate by Health Unit
British Columbia, 1987-1996

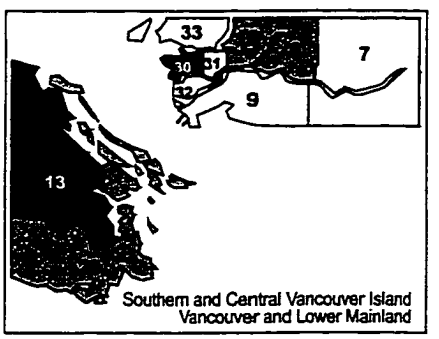
Health Unit Name & Code	Total Live Births	Infant Mortality				Neonatal Mortality				Postneonatal Mortality			
		Observed Deaths	Ratio (Obs/Exp)	p Value	Death Rate	Observed Deaths	Ratio (Obs/Exp)	p Value	Death Rate	Observed Deaths	Ratio (Obs/Exp)	p Value	Death Rate
01 - East Kootenay	8,699	45	0.786		5.17	21	0.596	*	2.41	24	1.090		2.76
02 - Central Kootenay	8,117	55	1.030		6.78	36	1.095		4.44	19	0.925		2.34
04 - North Okanagan	11,410	74	0.986		6.49	50	1.082		4.38	24	0.831		2.10
05 - South Okanagan	20,492	139	1.031		6.78	90	1.084		4.39	49	0.945		2.39
06 - South Central	14,713	102	1.054		6.93	57	0.957		3.87	45	1.209		3.06
07 - Upper Fraser Valley	24,658	130	0.801	*	5.27	62	0.621	**	2.51	68	1.090		2.76
08 - Central Fraser Valley	27,370	173	0.961		6.32	105	0.947		3.84	68	0.982		2.48
09 - Boundary	56,383	302	0.814	**	5.36	189	0.828	**	3.35	113	0.792	*	2.00
10 - Simon Fraser	28,521	165	0.879		5.79	128	1.108		4.49	37	0.513	**	1.30
11 - Coast Garibaldi	8,374	54	0.980		6.45	35	1.032		4.18	19	0.897		2.27
13 - Central Vancouver Island	24,377	213	1.328	**	8.74	104	1.053		4.27	109	1.767	**	4.47
14 - Upper Vancouver Island	13,578	106	1.186		7.81	64	1.164		4.71	42	1.223		3.09
15 - Cariboo	9,785	90	1.398	**	9.20	52	1.312	*	5.31	38	1.535	**	3.88
16 - Skeena	14,789	121	1.243	*	8.18	68	1.135		4.60	53	1.416	*	3.58
17 - Peace River	10,461	65	0.944		6.21	37	0.873		3.54	28	1.058		2.68
18 - Northern Interior	18,698	177	1.439	**	9.47	104	1.373	**	5.56	73	1.543	**	3.90
20 - Capital Regional District	24,522	142	0.880		5.79	90	0.906		3.67	52	0.838		2.12
30 - Vancouver	43,737	325	1.129	*	7.43	195	1.101		4.46	130	1.175		2.97
31 - Burnaby	44,420	302	1.033		6.80	209	1.162	*	4.71	93	0.828		2.09
32 - Richmond	16,850	83	0.749	**	4.93	55	0.806		3.26	28	0.657	*	1.66
33 - North Shore	19,863	99	0.757	**	4.98	73	0.907		3.68	26	0.517	**	1.31
99 - Province Total	449,817	2,962	-	-	6.58	1,824	-	-	4.05	1,138	-	-	2.53

Footnotes:

1. The infant related mortality rate is 10-year aggregrated rate, total number of deaths over total number of live births from 1987 to 1996.
2. The expected number of deaths are obtained by multiplying BC 10-year mortality rate with total number of 10-year live births at each HU.
3. The p-value for the ratio is estimated by Chi-square test, derived by the square value of observed deaths minus expected deaths over the expected deaths at each Health Unit.
4. * indicates that the Ratio is significant at level of 5% (two tails), and ** indicates that the Ratio is significant at 1% level.

Map 1: Geographical Distribution Pattern of Infant Mortality Rate (per 1,000 Live Births)
By Health Unit, British Columbia, 1987 to 1996

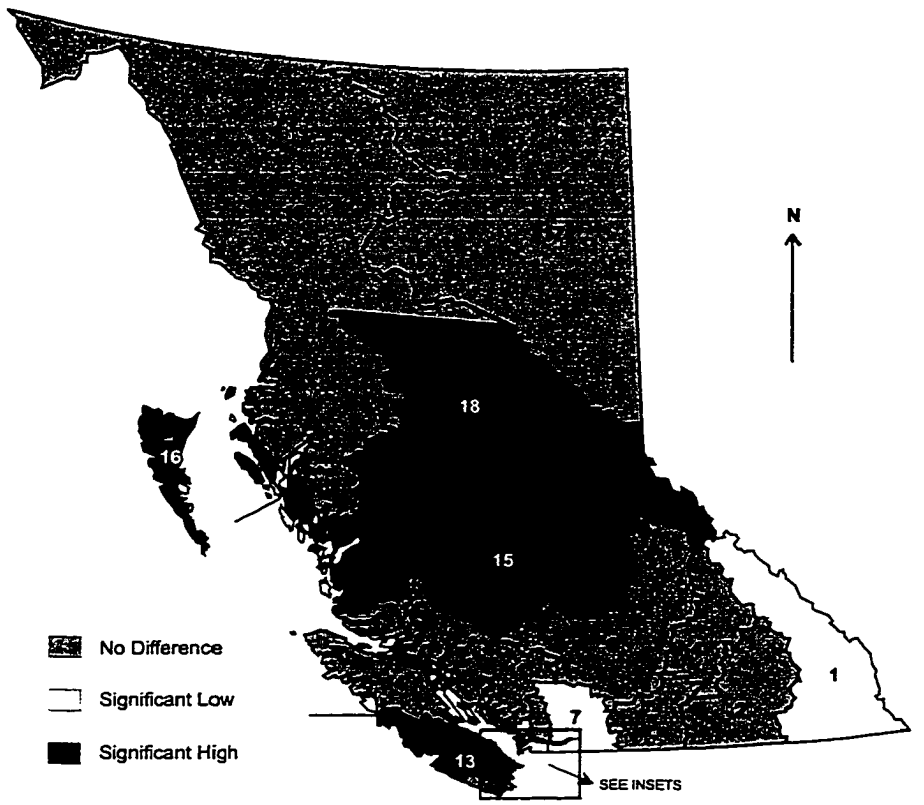


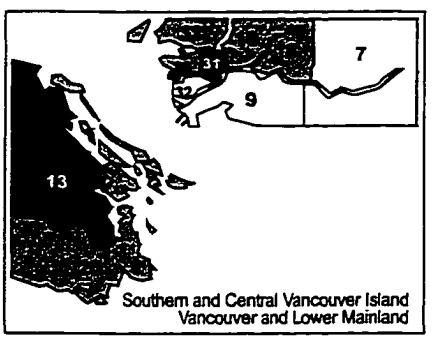


HEALTH UNITS

1. East Kootenay
2. Central Kootenay
4. North Okanagan
5. South Okanagan
6. South Central
7. Upper Fraser Valley
8. Central Fraser Valley
9. Boundary
10. Simon Fraser
11. Coast Garibaldi
13. Central Vancouver Island
14. Upper Vancouver Island
15. Cariboo
16. Skeena
17. Peace River
18. North Interior
20. Capital Regional District
30. Vancouver
31. Burnaby
32. Richmond
33. North Shore

Map 2: Geographical Distribution Pattern of Neonatal Mortality Rate (per 1,000 Live Births)
By Health Unit, British Columbia, 1987 to 1996





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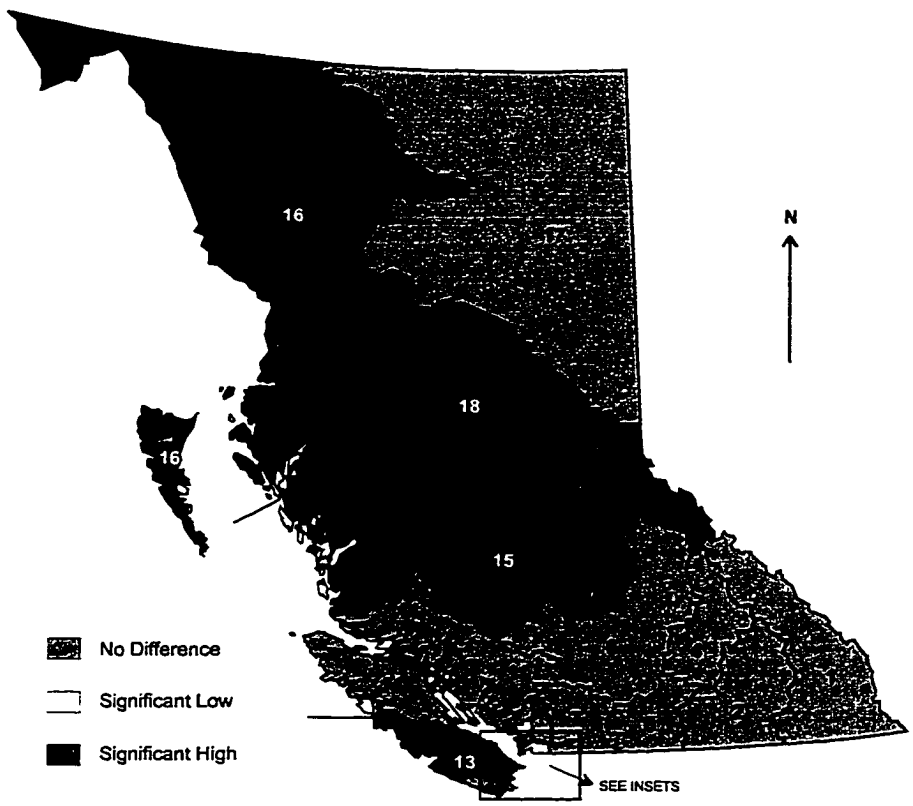
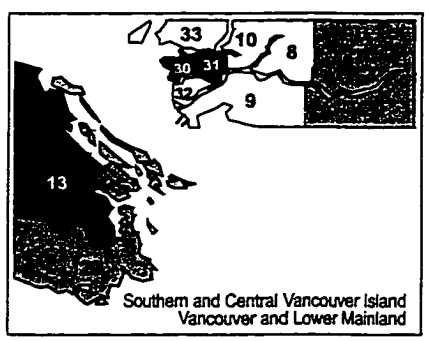
11. Coast Garibaldi
13. Central Vancouver Island
14. Upper Vancouver Island
15. Cariboo
16. Skeena
17. Peace River
18. North Interior
20. Capital Regional District
30. Vancouver
31. Burnaby
32. Richmond
33. North Shore

Map 3: Geographical Distribution Pattern of Postneonatal Mortality Rate (per 1,000 Live Births)
By Health Unit, British Columbia, 1987 to 1996





HEALTH UNITS

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5. South Okanagan
6. South Central
7. Upper Fraser Valley
8. Central Fraser Valley
9. Boundary
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17. Peace River
18. North Interior
20. Capital Regional District
30. Vancouver
31. Burnaby
32. Richmond
33. North Shore

North Shore (IMR=4.98 per 1,000 live births) and Richmond (IMR=4.93 per 1,000 live births), had significantly lower rates. Neonatal mortality rates were also significantly elevated in three Health Units including Cariboo (NMR=5.31 per 1,000 live births), Northern Interior (NMR=5.56 per 1,000 live births), and Burnaby (4.71 per 1,000 live births), while three others, East Kootenay (NMR=2.41 per 1,000 live births), Upper Fraser Valley (NMR=2.51 per 1,000 live births) , and Boundary (NMR=3.35 per 1,000 live births), had depressed rates of neonatal mortality. In addition, four regions, including Cariboo (PMR=3.88 per 1,000 live births), Skeena (PMR=3.58 per 1,000 live births) and Northern Interior (PMR=3.90 per 1,000 live births), and Central Vancouver Island (PMR=4.47 per 1,000 live births), showed significantly higher post-neonatal mortality than the provincial mean, while four others, including Boundary (PMR=2.76 per 1,000 live births), Simon Fraser (PMR=1.30 per 1,000 live births), North Shore (PMR=1.31 per 1,000 live births) and Richmond (PMR=1.66 per 1,000 live births), experienced lower rates than the average post-neonatal mortality rate. The scale of these differences in mortality rates were remarkable. To illustrate, the highest rate of infant mortality, 9.47 per 1,000 live births in the Northern Interior Health Unit, was almost double the lowest rate, which was 5 per 1,000 live births experienced in the Richmond and North Shore Health Units. The highest neonatal mortality rate (some 6 per 1,000 live births) experienced in the Northern Interior Health Unit, was almost triple that of the East Kootenay Health Unit which had the lowest recorded rate (about 2 per 1,000 live births). The highest post-neonatal mortality rate experienced in Central Vancouver Island, was nearly 5 per 1,000 live births. This rate was nearly 3 times

as high as that experienced by the North Shore Health Unit (1.3 per 1,000 live births).

Table 4 and Maps 1, 2, 3 illustrate recent geographic inequalities in three infant mortality indicators, at the Health Unit scale, in British Columbia based on ten years aggregated data. This approach to studying regional differentials in population health has been widely used elsewhere (Danderfer, 1997; Foster & Edgell, 1992). Unfortunately, these summary data can only display a static pattern of regional differences, and do not illustrate trends. As previously discussed, one of the major aims of this study is to investigate whether regional inequalities in British Columbia's infant health status have been reducing over time, paralleling the decline in infant death rates as a whole. Analysis of thirty years of data has confirmed a steadily declining infant mortality rate. For comparison, an indicator which reflects dynamic change in geographical inequality in infant mortality statistics, needs to be developed. The weighted coefficient of variation, as discussed in the Chapter 5, appears capable of meeting this need.

Table 5 summarizes temporal trends in geographical inequalities in British Columbia of the three infant death indicators and the corresponding mortality rates, from 1981 to 1996, at Health Unit scale. The values of these three mortality rates show clear declining trends over this time period (Figures 1 to 6). The weighted coefficients of variation, which represent the geographical inequalities of these rates, however, show remarkable fluctuations during the same period. Linear regression analyses, performed on both death rates and weighted coefficients of variation against the year, beginning in 1987, was used to explore further how

Table 5

Weighted Coefficient of Variation (WCV) of Infant Mortality Rate (IMR), Neonatal Mortality Rate (NMR), and Postneonatal Mortality Rate (PMR) By Health Unit for Calendar Years 1981 to 1996, British Columbia

Calendar Year	Live Births	IMR (1/1,000)	WCV for IMR	NMR (1/1,000)	WCV for NMR	PMR (1/1,000)	WCV for PMR
1981	40,868	10.03	31.757	6.51	39.303	3.52	39.796
1982	42,258	9.65	22.730	6.06	32.450	3.60	46.046
1983	42,296	8.61	32.672	5.01	34.771	3.59	41.099
1984	43,347	8.33	31.125	4.71	39.224	3.62	42.425
1985	42,803	7.64	27.711	4.51	38.513	3.13	45.531
1986	41,795	8.11	27.339	4.62	37.513	3.49	43.291
1987	41,626	8.38	32.003	4.64	45.243	3.75	36.778
1988	42,823	8.03	32.418	4.81	33.974	3.22	41.946
1989	43,583	7.85	32.990	4.63	43.835	3.21	43.354
1990	45,357	7.30	37.350	4.87	39.716	2.43	63.068
1991	45,295	6.29	28.060	3.51	28.058	2.78	47.210
1992	45,704	5.91	40.450	3.70	43.170	2.21	53.501
1993	45,986	5.46	37.374	3.04	37.138	2.41	57.836
1994	46,923	6.12	30.044	4.16	44.859	1.96	47.982
1995	46,639	5.92	26.809	3.84	27.309	2.08	57.649
1996	45,881	4.95	31.883	3.49	35.991	1.46	70.363

Footnotes:

1. **The primary source of this table is the BC Vital Statistics Agency Annual Reports.**
2. **Infant mortality is death of children under one year of age.**
3. **Neonatal mortality is death of a child under 28 days of age.**
4. **Postneonatal mortality is death of a child between the ages of 28 days and less than one year.**

weighted coefficients of variation for the three death rates have been changing during this time period. As shown in Figure 7, the weighted coefficient of variation of infant mortality rate displays an obvious ascending trend, in contrast to the trend of the infant mortality rate. The β value of the weighted coefficient of variation for infant mortality rate during the last 16 years was 0.28, indicating that there appears to have been nearly a one-third increase in the geographical inequality of infant mortality rate at the Health Unit level per year (Table 6). This annual increase resulted in a total 14.5 percent rise in geographical inequality in the infant mortality rate during the last 16 years. In summary, therefore, while the infant mortality rate in British Columbia fell by 45 percent from 1981 to 1996, the geographical difference in infant mortality amongst its 21 Health Units increased by some 15 percent during the same period (Table 6). Although the latter increase is not statistically significant, it clearly demonstrated that regional differences in infant mortality rate did not parallel the declining trend of the infant mortality rate, for the province as a whole.

Post-neonatal mortality rates, at the Health Unit level, also showed a similar time trend for geographical inequality, (Tables 5 and 6, Figure 9), from 1981 to 1996. However, the change in geographical inequality of the post-neonatal mortality rate was statistically significant, rising almost 60 percent during this period (Table 6). In contrast, during this time, the mortality rate decreased by 48 percent. Regional differences in post-neonatal mortality rate, therefore, appear to be far greater than differences in infant mortality rates.

Regional differences in neonatal mortality rate during the last 16 years,

however, show a slightly different trend, when compared with those for infant and post-neonatal mortality rates (Tables 5, 6, Figure 8). The neonatal mortality trend in geographical inequality declined slightly less than 3 percent at the Health Unit level, while the neonatal mortality rate, in contrast, experienced a significant decline of more than 40 percent during the same period. Although regional differences in neonatal mortality rate were not significant, the analysis illustrated that a reduction in neonatal mortality rate has not been experienced by all Health Units.

The use of the weighted coefficient of variation to measure the geographical inequality in mortality rates raises certain problems. The weighted coefficient of variation itself is very closely related to the size and number of geographical areas used in the evaluation. For instance, the weighted coefficient of variation could be diminished to zero by the aggregation of all the 21 Health Units into one area, that is into the province as a whole. It is possible, therefore, that the preceding analyses of the weighted coefficients of variation for infant, neonatal, and post-neonatal mortality rates over the last 16 years may merely reflect the artificial division of British Columbia into 21 regions, that is into Health units. In order to investigate the impacts on the values of weighted coefficients of variations in the size of the geographical regions and the time period, all the province's 79 Local Health Areas (LHAs) which are aggregated to form Health Units, were randomly grouped into different sized geographical regions. A two-way analysis of variance was then performed to examine the impacts on the values of weighted coefficients of variation of both the size of the region analysed and the time period selected. Table 7 summarizes the results obtained.

Table 6:
Trends in the Average Rate and Geographic Inequalities
For Neonatal, Postneonatal, and Infant Mortality Rates (per 1,000 Live Births)
Aggregated by Health Unit, British Columbia, 1981-1996

Infant Death Indicator	Dependent Variable (Y)	Linear Regression Equation	β Significance (p Value)	Coefficient Of Determination	Absolute Value of Change	Percentage Of Change
Infant Mortality	IMR	Y= 9.95-0.30X	p<0.0001	0.89	-4.50	-45.23%
	WCV	Y=29.02+0.28X	p=0.2575	0.85	+4.2	+14.45%
Neonatal Mortality	NMR	Y= 5.87 -0.16X	p<0.0001	0.69	-2.40	-40.89%
	WCV	Y=38.14-0.07X	p=0.8272	0.45	-1.05	- 2.75%
Post-neonatal Mortality	PMR	Y= 4.07 - 0.13X	p<0.0001	0.81	- 1.95	-47.91%
	WCV	Y=36.37+1.44X	p=0.0001	0.51	+21.60	+59.39%

Notes:

1. IMR, NMR, and PMR represent Infant Mortality Rate, Neonatal Mortality Rate, and Postneonatal Mortality Rate, respectively.
2. WCV stands for Weighted Coefficient of Variation.
3. The regression coefficients for IMR, NMR, and PMR were all statistically significant, but those for WCVs were not significant.
4. Coefficient of Determination is R^2 value, which describes the proportion of the variance of dependent variable explained by independent variables. In this particular table, the coefficient of determination gave the percent of the variances of IMR, NMR, PMR, and their corresponding WCVs explained by the number of year starting from 1981.

Figure 7: Weighted Coefficient of Variation (WCV) for Infant Mortality Rate (IMR) By Health Unit, 1981 to 1996, British Columbia

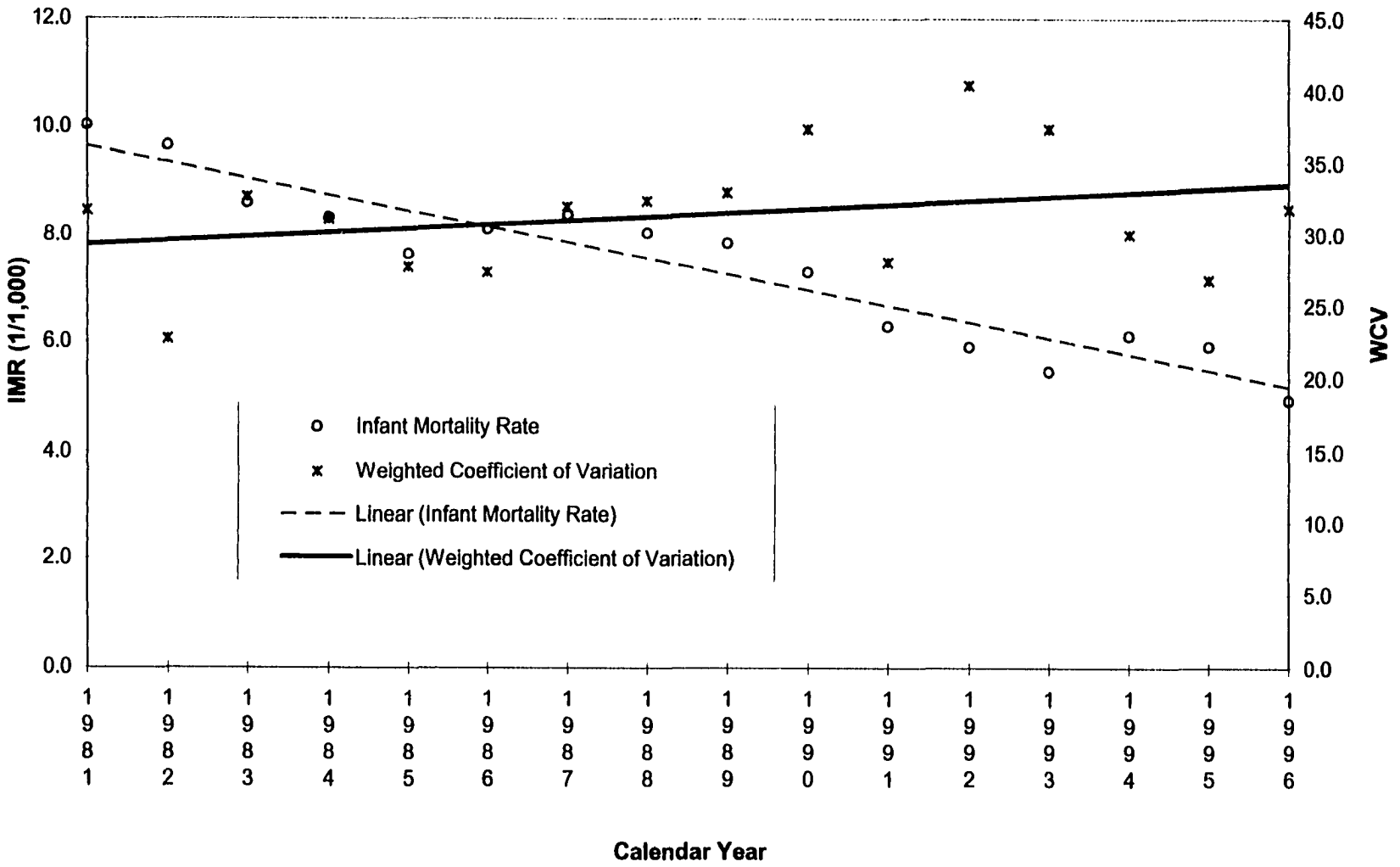


Figure 8: Weighted Coefficient of Variation (WCV) from 1981 to 1996 For Neonatal Mortality Rate by Health Unit, British Columbia

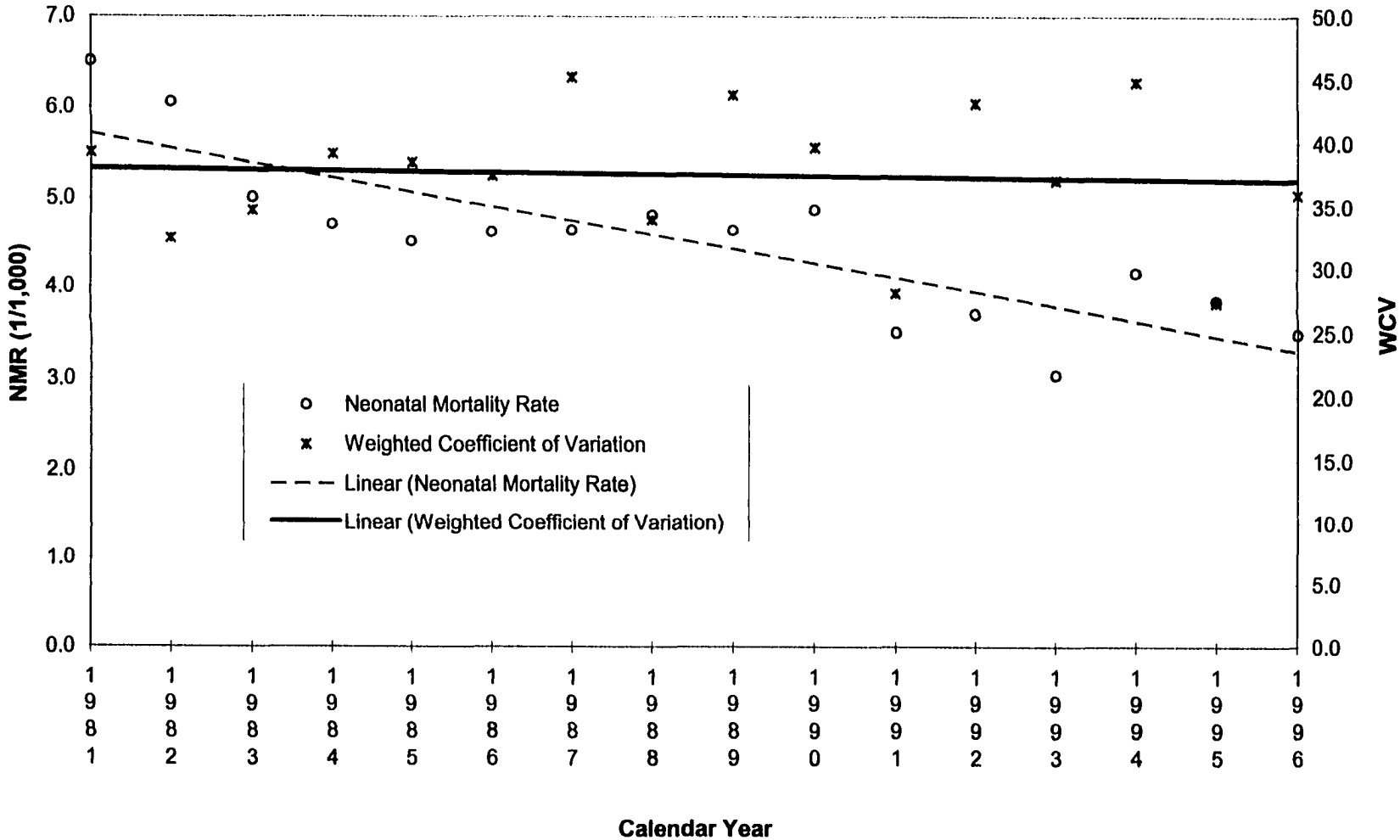
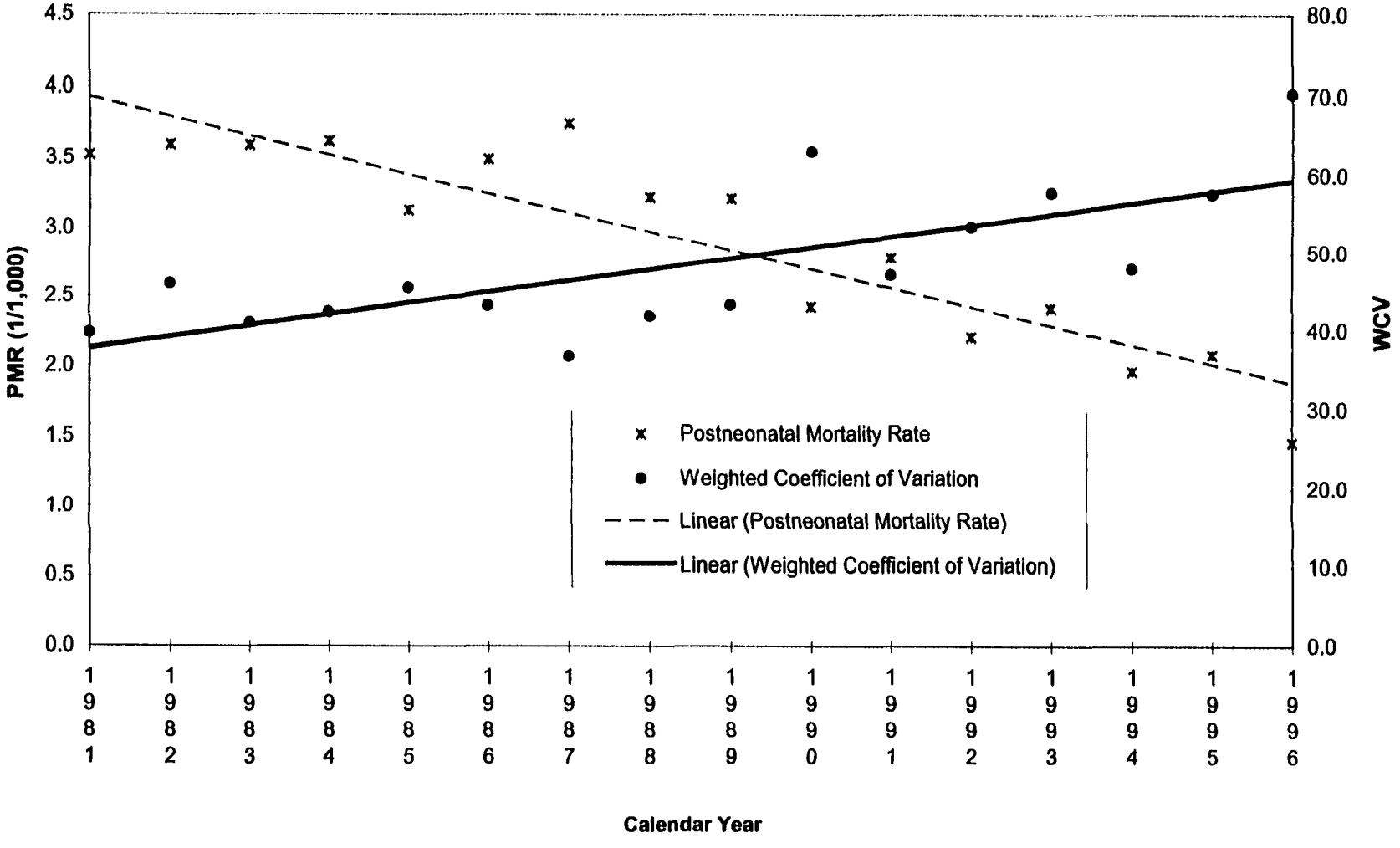


Figure 9: Weighted Coefficient of Variation (WCV) from 1981 to 1996
For Postneonatal Mortality Rate (PMR) By Health Unit, British Columbia



In Table 7, the first column is the number of randomly aggregated LHAs. For instance, the number, 3, indicates that all 79 LHAs have been aggregated into 3 areas. Since this aggregation is random, the actual number of LHAs in each of these 3 synthetic areas is comparable, about 15 to 16 LHAs. Similarly, the second number, 10, indicates that the original 79 LHAs have been randomly grouped into 10 areas with an equivalent number of LHAs in each. The number of 21 is the actual number of Health Units in this province, while the number 79 represents actual LHAs. The figures listed in the first column, named the LHA Group, represent the number of areas represented by the original 79 LHAs, the real units for which actual infant mortality statistics were developed. This arrangement means that as the number increases (from the top to the bottom of column one), the size of the aggregated areas is reduced. The first row contains the calendar year from 1981 to 1996. As a result, the number contained in each cell in the table is the weighted coefficient of variation of infant mortality developed by aggregated areal infant mortality, by year. To illustrate, 8.63, which is located in the cell represented by LHA Group 3 and Year 1981, is the weighted coefficient of variation of infant mortality in year 1981 when the original 79 LHAs are randomly aggregated into only 3 large areas. The values contained in each row indicates how the weighted coefficient of variations has changed when mortality data are summarized at different geographical scales.

Comparisons of the values of weighted coefficient of variation, at various geographical scales and for different years within each scale highlight two important facts. Firstly, the weighted coefficient of variation increases dramatically as the

aggregated geographical scales decrease. To illustrate, comparison by row from top to bottom, indicates that the smaller the geographical areas analysed, the larger the areal variations of infant mortality. Secondly, the weighted coefficient of variation also tends to increase over time, regardless of geographical scales. It is clear, therefore, that the size of the geographical region indicated by the number of aggregated LHAs significantly influences the value of the weighted coefficients of variation of infant mortality rates. As the areal size increases, that is as the number of geographical regions based on the 79 LHAs is reduced, the weighted coefficients of variation falls. This is understandable because the difference in the rates amongst smaller areas is diminished as they are merged into larger areas. Real geographical variations in infant death rates, therefore, would not have occurred had the values of the weighted coefficients of variation been completely determined by the sizes of geographical region, on which such rates were calculated.

From a statistical point of view, the variances of the weighted coefficient of variation over the study period would be attributed completely to the sizes of aggregated regions. However, it was also found that the size of the region itself did not fully explain all the variance of the weighted coefficient of variation, and a portion of the weighted coefficient of variation seems to be related to the time period under study. To illustrate, the total variance of the weighted coefficient of variation of the infant mortality rate was around 39,900, of which about 80 percent could by explained by the size of geographical region, and 20 percent could be attributed to the time (Table 7). Linear regression analysis showed both size and time have a significant association with the weighted coefficient of variation of infant mortality

TABLE 7:
Analysis of Variance: Weighted Coefficient of Variation of Infant Mortality Rate By Randomly LHA Groups, 1981 to 1996, British Columbia

LHA Group*	Year of Death															
	1981	1982	1983	1984	1985	1986	1987	1988	1989	1990	1991	1992	1993	1994	1995	1996
3	8.63	3.19	4.20	3.70	9.88	3.02	5.67	5.15	7.27	10.15	14.27	14.14	12.04	13.48	6.02	15.06
10	15.90	15.81	14.38	8.33	16.84	17.17	9.64	17.79	28.49	26.38	19.77	25.19	28.58	22.95	21.13	23.50
15	20.30	20.67	19.72	16.87	17.97	19.48	19.04	18.14	27.06	27.05	22.44	32.52	31.40	28.44	22.97	22.68
20	23.56	19.82	19.07	21.42	21.94	24.98	23.13	22.49	30.48	31.08	34.46	45.84	39.38	35.25	28.07	40.79
21**	31.64	22.39	32.07	31.26	23.53	26.91	32.55	33.32	33.34	36.32	28.06	40.45	37.37	30.04	26.81	31.88
25	26.51	26.74	31.10	21.56	23.73	31.80	28.87	27.19	28.90	37.93	34.77	48.53	35.60	38.05	29.49	38.54
30	30.46	26.50	33.22	24.11	33.01	31.75	29.87	28.29	33.21	39.63	37.26	49.50	42.26	42.02	30.26	41.94
35	33.96	23.17	31.54	25.50	26.28	33.79	35.26	31.09	36.33	38.09	36.08	50.14	42.71	44.52	41.67	42.34
40	32.26	31.04	34.17	30.88	32.56	32.37	36.79	39.71	38.43	38.29	39.64	50.05	45.62	43.95	39.62	44.64
45	38.04	33.15	35.10	33.91	40.02	34.13	34.96	42.29	44.77	41.43	39.84	52.28	45.42	50.20	39.20	44.82
50	31.00	30.58	39.03	29.66	34.04	34.64	37.01	40.28	42.89	45.65	41.58	51.72	45.74	46.33	36.46	43.10
55	36.81	29.59	38.75	31.18	36.96	36.67	34.95	43.18	41.47	43.03	40.54	52.78	43.98	50.18	43.45	43.33
60	36.71	32.00	38.96	31.58	38.40	36.29	36.62	44.56	41.34	50.35	45.92	53.96	48.44	46.72	42.26	45.17
65	40.73	32.72	39.27	31.86	37.35	35.87	39.40	43.14	46.54	47.04	43.87	56.26	50.02	48.77	45.99	46.29
70	40.08	35.22	39.95	31.26	40.91	37.47	39.55	43.28	46.94	48.28	42.07	55.86	51.32	53.86	51.79	47.28
79***	50.16	42.27	51.95	52.18	50.22	46.48	51.73	54.69	54.90	58.72	51.95	72.36	64.08	62.57	63.93	66.91

Footnotes:

1. Total variance (Total Sum of Squares) = 39,900.5398.
2. The variance explained by independent variable YEAR is 7,971.0533, making up to 20% of the total variance, at p=0.0001 level.
3. The variance explained by independent variable LHAGRP (Random LHA grouping variable) is 29,921.1003, making up to 80% of the total variance, at p=0.0001 level.
4. * LHA group contains a randomly aggregated LHAs; ** these 21 groups are the actual Health Unit grouping; *** these 79 groups are the actual individual LHAs.

rate over the last 16 years (Table 7). Clearly, geographical variation in the infant mortality rate has existed consistently for the last 16 years in British Columbia, and is independent of the size of geographical region for which it is measured.

The weighted coefficient of variation is a summary index describing the trend of regional variation of infant-related mortality rates over the past 16 years in British Columbia. However, certain detailed information could have been lost when data is summarized by use of a single index. In order to examine this issue in more detail, Figures 10 to 12 further display trends in geographical inequalities of infant-related mortality by examining the ranges of infant, neonatal, and post-neonatal mortality rates amongst the 21 Health Units, for the past 16 years. Each of these charts include four curves, the two in the middle are provincial mortality rate and median mortality rate of all 21 Health Units, respectively. The bottom line is the minimum mortality rate of the Health Unit, and the top line represents the maximum mortality rate of the Health Unit. Therefore, the distance or gap between the bottom and top lines is the range of the mortality rate, at the Health Unit scale. The sizes of the gaps illustrate differences in infant mortality rates amongst all Health Units, that is they represent geographical inequalities. Plotting ranges by year displays how geographical differences in mortality rates, amongst Health Units, has changed over the last 16 years.

Figures 10, 11 and 12 all demonstrate that the HU median mortality rates, including those for infants, neonates, and post-neonates, display similar trends to those of the provincial rates, indicating that the provincial mortality rates are representative of the average rates for all Health Units. There illustrations indicate

Figure 10: Geographical Variation in Annual Infant Mortality Rate (IMR) By Health Unit (HU), British Columbia, 1981 to 1996

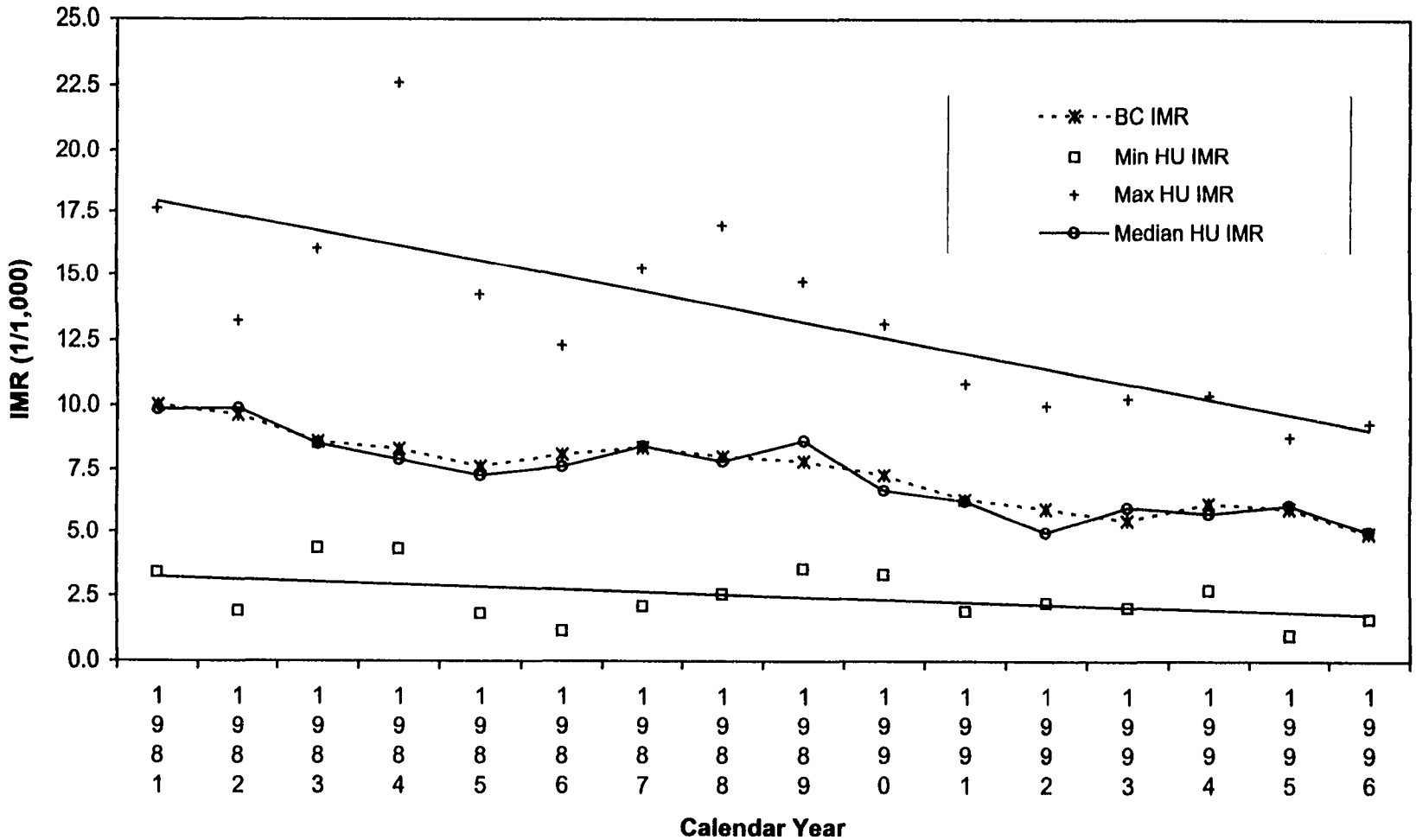


Figure 11: Geographical Variation in Annual Neonatal Mortality Rate (NMR) By Health Unit (HU), British Columbia, 1981 to 1996

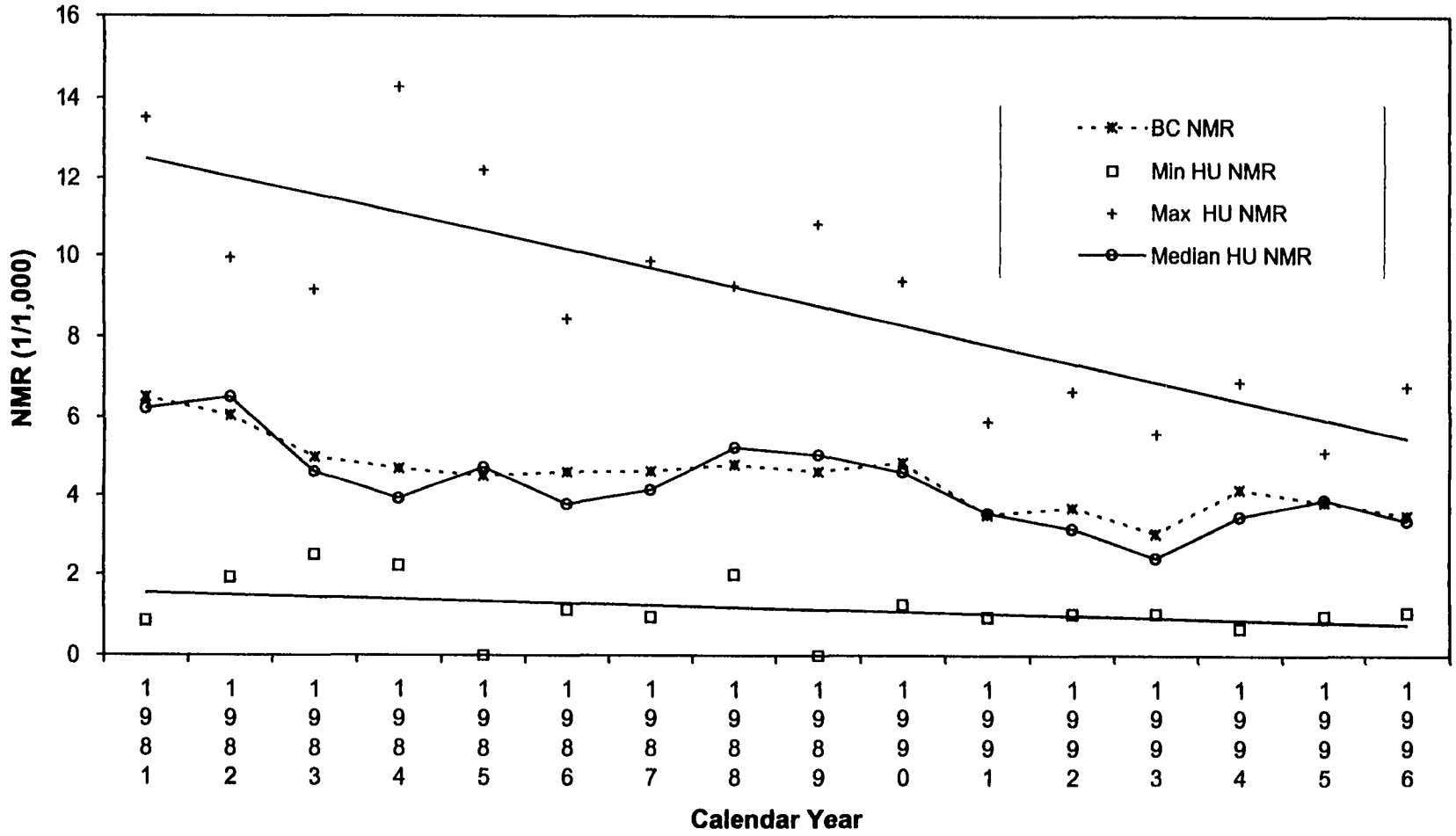
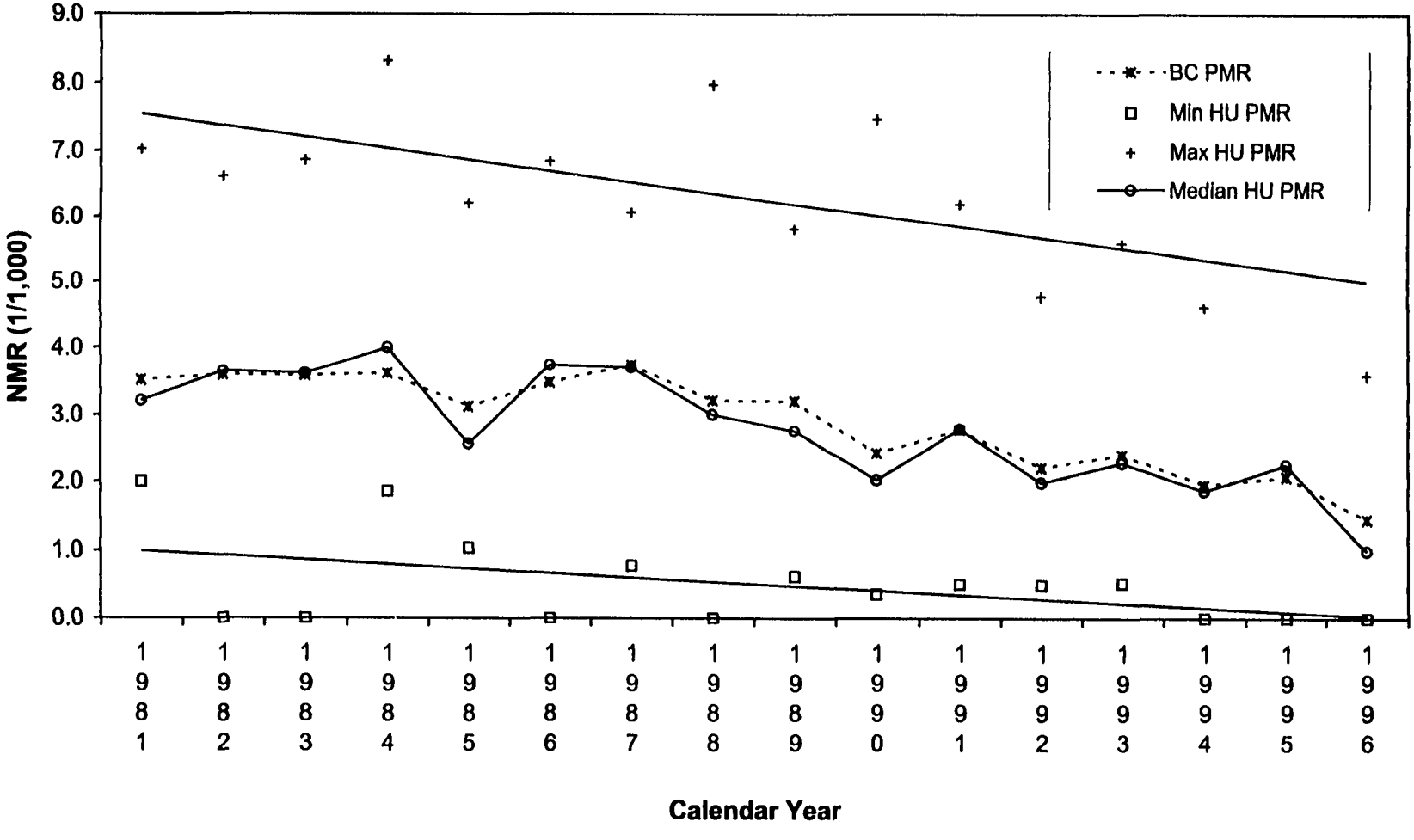


Figure 12: Geographical Variation in Annual Postneonatal Mortality Rate (PMR) By Health Unit (HU), British Columbia, 1981 to 1996



continuous reductions amongst these rates because the average mortality rates of infants, neonates, and post-neonates, at the Health Unit scale, have decreased consistently over the last 16 years, as have the provincial rates. The maximum rates show a deeper declining trend than the minimum rates, indicating that the maximum mortality rates at the Health Unit scale experienced a greater reduction than the minimum rates. As a result, the range also has been reduced. To illustrate, the range of infant mortality rates was reduced from approximately 14.2 in 1981 to 7.7 per 1,000 live births in 1996, a decrease of 46 percent. Similarly, the range of the neonatal mortality rate has been reduced by 55 percent, from 12.7 in 1981 to 5.7 in 1996. Furthermore, the post-neonatal mortality rate has been decreased by 29 percent, from 5.0 in 1981 to 3.6 per 1,000 live births in 1996. This is obvious from figures 10 to 12 where the gap between the top (maximum) and bottom (minimum) lines for all three infant death rates have narrowed over the past 16 years. If the ranges of infant related mortality rates at the Health Unit scale are good indicators of regional differentials of infant deaths, these patterns appear to indicate that regional differences in these infant death rates also have been decreased.

The trends of geographical inequalities described using weighted coefficient of variation (WCV) and illustrated in Figures 7, 8 and 9 seem to contrast with the inferences that have just been drawn from Figures 10, 11, and 12. For instance, as previously pointed out, the weighted coefficient of variations for all the three infant death rates have tended to increase indicating a rising trend in regional differences in these mortality rates, while the ranges discussed above and illustrated in Figures 10 to 12 show that regional differences have been reduced. There is some

ambivalence, therefore, about whether or not geographical variations have increased or decreased, over the last 16 years. However, the explanation of these controversial results may be a statistical artefact, rather than any characteristics of infant health status at the Health Unit scale. A more detailed discussion of different statistical methods that can be used to represent regional mortality variations is presented in Chapter 7. Briefly, it appears that the weighted coefficient of variation takes into accounts several parameters, while the range depends only upon maximum and minimum values which are very unstable, especially when the range is applied to such a low probability event. As a result, the weighted coefficient of variation has been considered by others to be the best index of geographical variation in health status (Lardelli et al, 1991; Wagstaff, Paci, van Doorslaer, 1991; Vazquez-Vizoso, 1993).

6.3 Etiological Factors and Geographical Inequalities in Infant Death Rates

As discussed in Chapter Two, the etiological factors involved in infant mortality include those parameters which are more closely associated with the physical and physiological development of infants, or with the underlying causes of diseases suffered by infants. For instance, the maternal smoking habit would be more likely to be seen as an etiological factor for low birth weight than maternal education level. This is because maternal smoking has been physiologically proved to be related to fetal development and low birth weight (Kramer, 1987; Klaus & Fanaroff, 1986). Although less educated mothers tend to be smokers, the education level of a mother is not directly associated with infant health status. Following this approach,

the following five parameters were selected as etiological factors in infant death rates: low birth weight rate, teenage birth rate, preterm birth rate, perinatal conditions rate, and maternal complications rate.

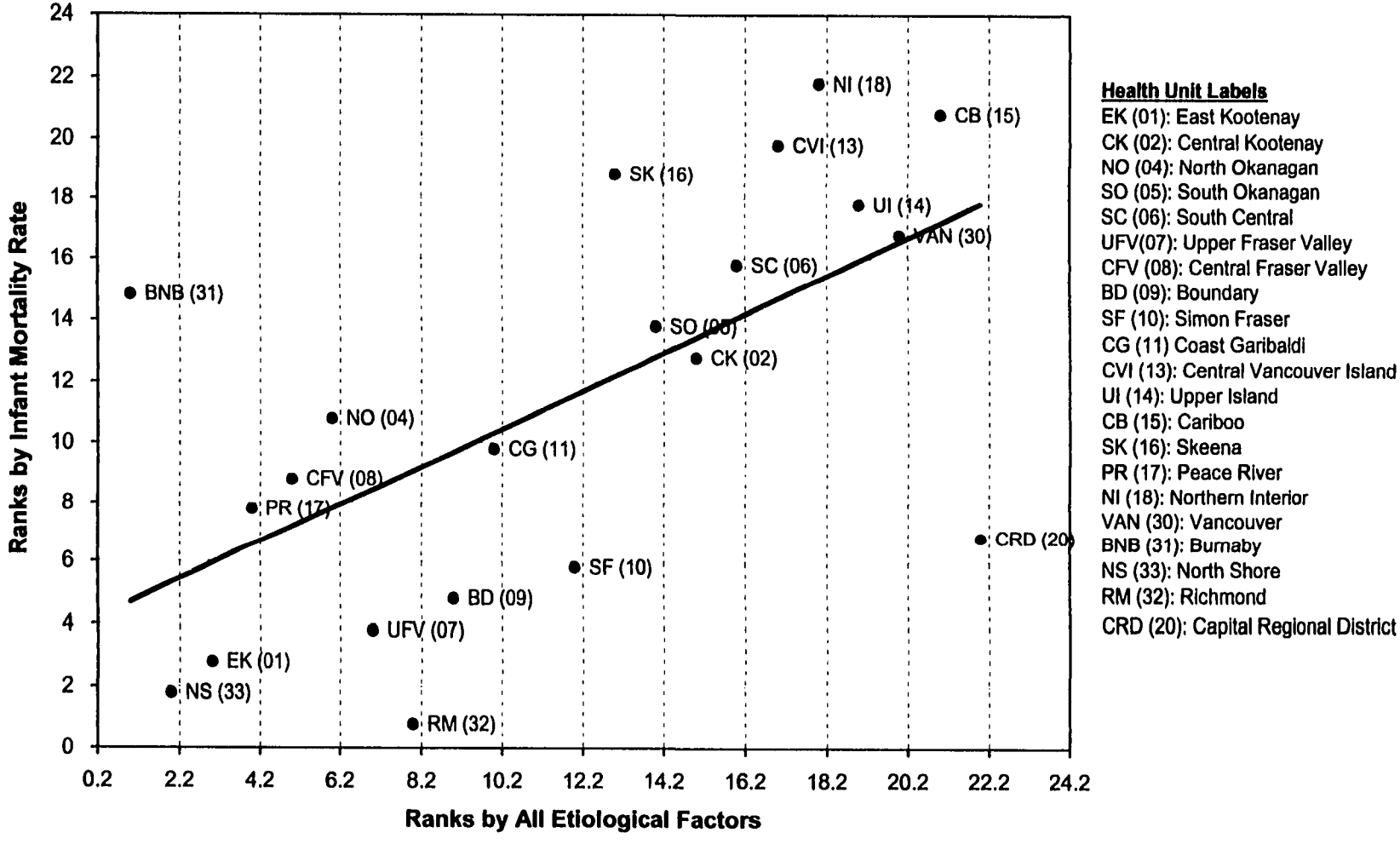
To investigate if there is any association between infant mortality and these etiological factors, correlation and/or regression analyses between these two sets of parameters would normally be performed. In particular, measures of these etiological factors associated with each deceased infant would be analysed to determine if a strong relationship exists between such factors and the probability of death of that infant. In this study, however, information on etiological factors specific to each infant was not conveniently available. Instead, all available data were based on Local Health Areas and/or Health Units, that is on population-based or area-based information. As a result, it is inappropriate to apply these population- or area-based data to any individual infant because of the ecological fallacy issue (Clark & Hosking, 1985; Feinleib & Leaverton, 1984). In order to analyse relationships under such data restrictions, rank analysis rather than classical correlation and regression analyses have been applied.

To do this, each Health Unit was ranked according to the rate for each of these five etiological factors, for example the number of infants with low birth weight per 1,000 live births. As a result, four sets of ranks were created for each Health Unit. Simultaneously, each Health Unit was ranked according to its mortality rates for infants, neonates, and post-neonates. Each Health Unit, therefore, was ranked eight times, three times by mortality rates and five times for etiological parameters. Since this is a rank, quantitative differences between each Health Unit as measured

by mortality rates, or the values of other etiological factors were converted to qualitative indices. As a result, rank differences in the values between Health Units have no quantitative meaning, but simply indicate whether a Health Unit is higher or lower than any other in any one of these eight parameters. It is to be expected that a scattered two-dimension chart, one dimension of which represents mortality rank and the other etiological factor rank, will be able to display a tendency for infant mortality to be higher in those Health Units where there are greater prevalence rates for the five selected etiological variables. Figures 13 to 15 graphically illustrate this concept.

Figure 13 is a scatter plot designed to describe the associations between Health Unit based infant mortality rates and Health Unit based etiological variables. The y-axis indicates the rank of the 10-year aggregated infant mortality rate from 1987 to 1996 for each Health Unit, while the x-axis displayed the average rank of each Health Unit, derived from the ranks of the five etiological variables, low birth weight rate, preterm birth rate, teenage birth rate, maternal complications rate, and perinatal conditions rate. The average rank was developed from the mean values of all the ranks for each Health Unit, based on all five etiological factors. This average rank, therefore, can be used to represent a comprehensive etiological setting as a whole for each Health Unit, because these five variables are all negative indices of infant health status and all positively contribute to the infant mortality rate. In the same way, Figures 14 and 15 display the rank-relationships between these etiological variables and both neonatal and post-neonatal mortality rates. Ranks are in ascending order, so that Health Units with higher values for a

Figure 13: Ranks of Health Units by All Etiological Factors and Infant Mortality Rate From 1987 to 1996, British Columbia



Health Unit Labels

EK (01): East Kootenay
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SO (05): South Okanagan
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UFV(07): Upper Fraser Valley
CFV (08): Central Fraser Valley
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PR (17): Peace River
NI (18): Northern Interior
VAN (30): Vancouver
BNB (31): Burnaby
NS (33): North Shore
RM (32): Richmond
CRD (20): Capital Regional District

Figure 14: Ranks of Health Units by All Etiological Factors and Neonatal Mortality Rate From 1987 to 1996, British Columbia

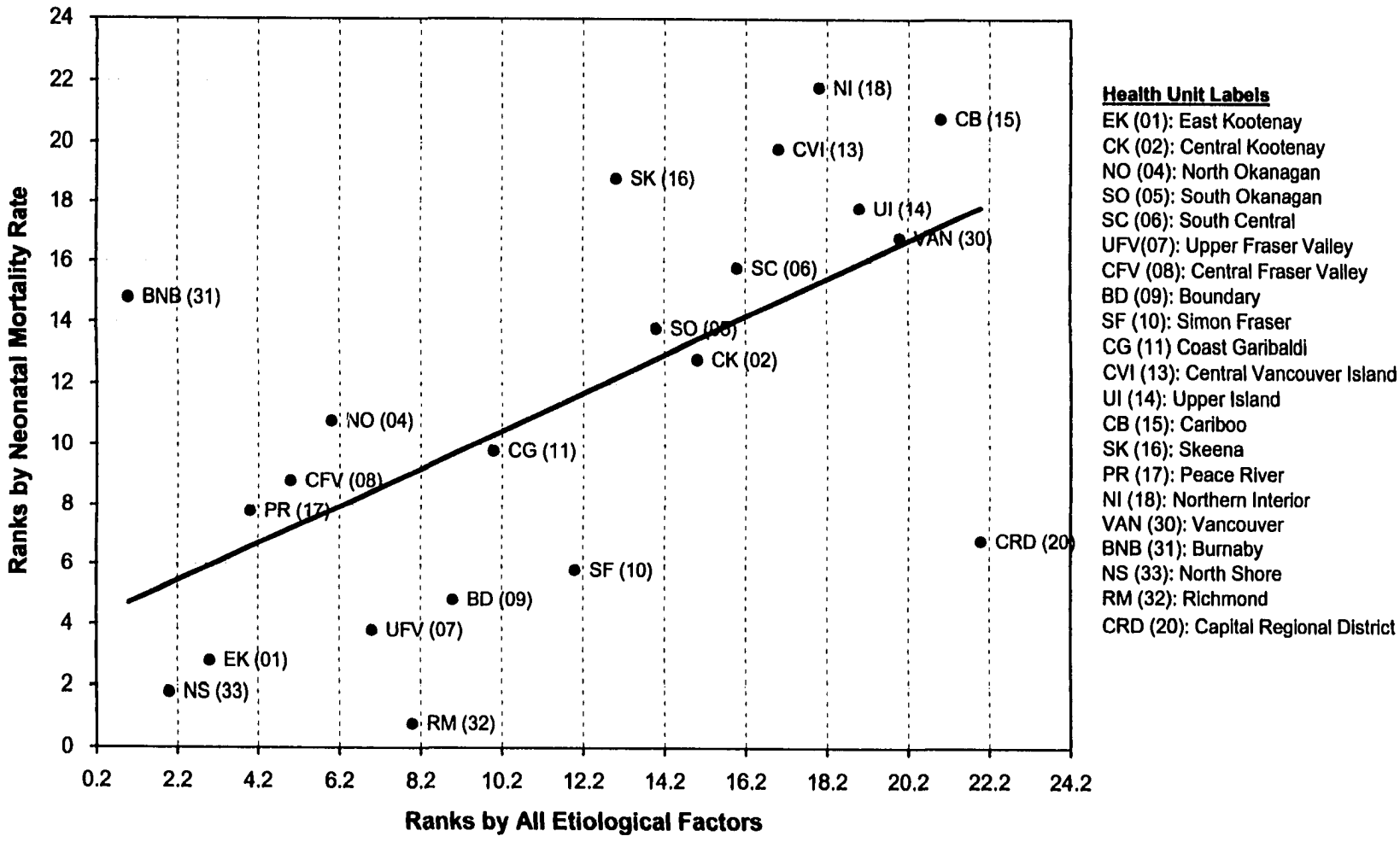
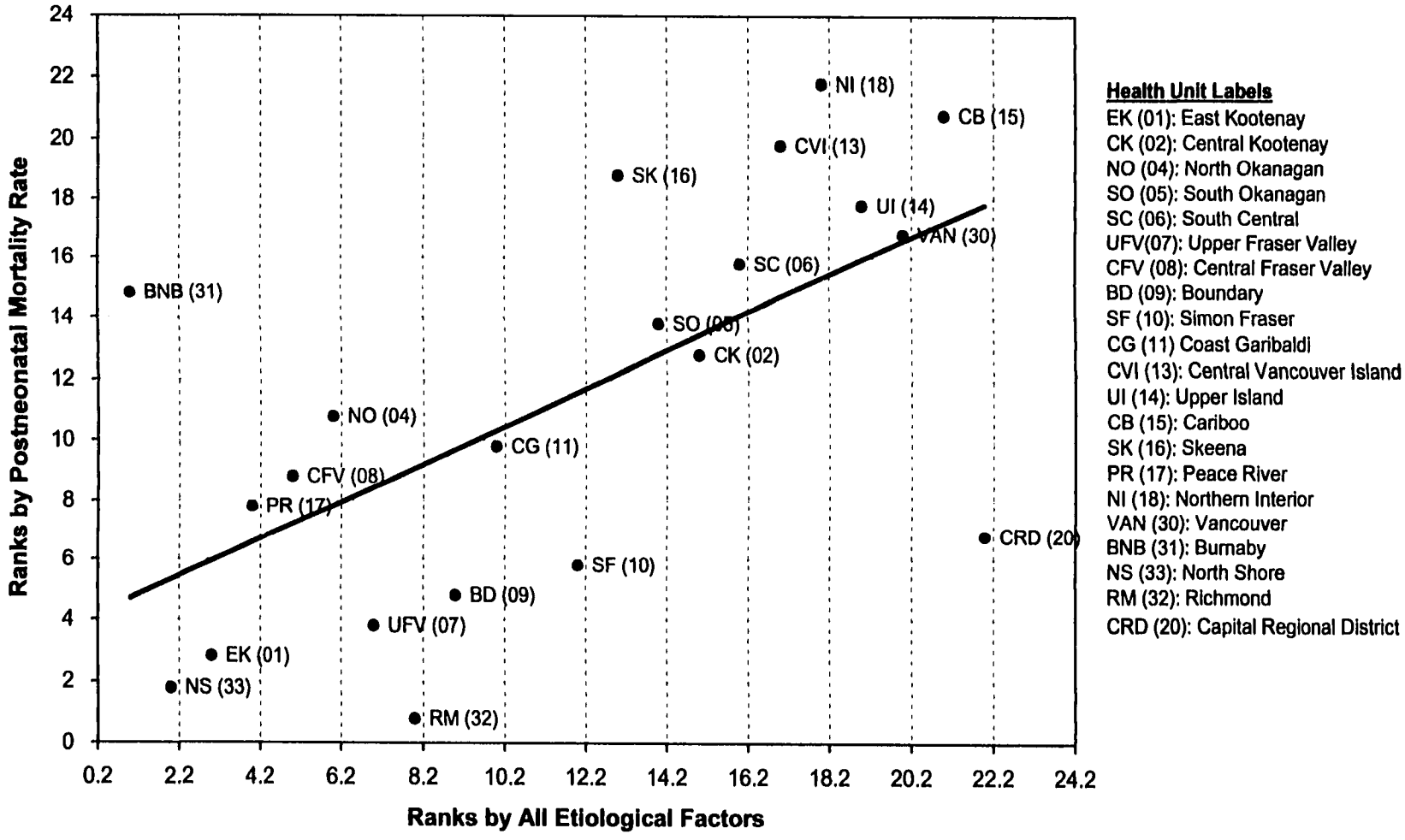


Figure 15: Ranks of Health Units by All Etiological Factors and Postneonatal Mortality Rate, from 1987 to 1996, British Columbia



particular rank, for example infant mortality rate, indicate that this Health Unit has a higher mortality rate than those with lower ranks. These three charts, generally display a clear positive relationship between ranks by mortality rates and by etiological factors. Health Units with higher ranks in etiological factors also have higher infant, neonatal, and post-neonatal mortality rates, indicating that the areas that experience higher rates of low birth weight, preterm birth, teenage birth, perinatal conditions, and maternal complications tend to experience higher infant mortality rates.

In order to study the associations between infant mortality and specific etiological factors further, a Pearson correlation analysis was performed between the three mortality rates and the five etiological factors. Table 8 presents the results of this correlation analysis between infant health indicators and etiological factors, and is based on 10-year aggregated data for each Health Unit. This means that each Health Unit was treated as one observation for this correlation analysis, which utilized infant mortality data for the 10-year period from 1987 to 1996. The *r* value in this table represents the value of the correlation coefficient, and the *p* value indicates the statistical significance of the corresponding *r* value, showing whether or not the correlation is statistically significant. This analysis indicates that Health Unit specific infant mortality rate is significantly correlated with teenage birth rate, with *r* value 0.6003 and *p* value 0.0040. The associations with other etiological factors are not significant. The associations between post-neonatal mortality rate and the etiological factors follow a similar pattern to that of infant mortality. There is a significant correlation between this mortality rate and rate of births to teenage

mothers. The *r* value is 0.7395 with *p* value of 0.0001. The associations with other etiological factors are not statistically significant. Neonatal mortality rate, however, does not show significant associations with any of these five etiological factors. The positive correlation between infant mortality rate and teenage birth rate may indicate that during the last 10 years the Health Units with higher rate of births to teenage mothers have tended to experience a higher infant mortality rate. This might suggest that the existing geographical inequality in infant and post-neonatal mortality at the Health Unit level may be attributed to regional differences in teenage birth rates amongst these Health Units.

In order to confirm this tentative conclusion, derived from the previous correlation analysis, the same method was applied at the Health Unit scale to another 10-year period data, that is from 1981 to 1990. The mortality data and the etiological factors for this time period were once again aggregated for each Health Unit. Correlation analyses were then again performed between Health Unit specific mortality rates and the rates of etiological factors. Table 8a displays the results. It is obvious from this illustration that both Health Unit specific infant and post-neonatal mortality rates for this 10-year time period had significant correlations with the rate of birth to teenage mothers. The correlation coefficients (*r* values) are 0.5991 for infant mortality rate and 0.7825 for post-neonatal mortality rate, with *p* values of 0.0041 and 0.001 respectively. Once again, neonatal mortality rate did not show any significant association with teenage birth rate. The association patterns between mortality rates and other etiological factors for this time period were not significant, except that with pre-term birth rate. Both infant and neonatal mortality

TABLE 8:
Correlation Analysis: Infant Mortality Rates and Etiological Factors
By Health Unit Based on 10-year (1987 through 1996) Aggregated Data, British Columbia

Infant Health Status Indicators	Correlation Statistics indices	Low Birth Weight Rate	Pre-term Birth Rate	Teenage Birth Rate	Perinatal Conditions Rate	Maternal Complications Rate
Infant Mortality Rate	*r Value*	0.2307	0.2930	0.6003	0.1991	0.0316
	p Value	0.3144	0.1974	0.0040	0.3870	0.8917
Neonatal Mortality Rate	*r Value*	0.1468	0.2496	0.2417	0.0671	0.0048
	p Value	0.5256	0.2752	0.2912	0.7726	0.9836
Post-neonatal Mortality Rate	*r Value*	0.2331	0.2361	0.7395	0.2576	0.0465
	p Value	0.3092	0.3028	0.0001	0.2595	0.8413
Low Birth Weight Rate	*r Value*	-	0.9440	0.2220	0.8790	0.9236
	p Value	-	0.0001	0.3334	0.0001	0.0001
Pre-term Birth Rate	*r Value*	-	-	0.1547	0.8527	0.9116
	p Value	-	-	0.5031	0.0001	0.0001
Teenage Birth Rate	*r Value*	-	-	-	0.3296	0.0825
	p Value	-	-	-	0.1445	0.7224
Perinatal Conditions Rate	*r Value*	-	-	-	-	0.9643
	p Value	-	-	-	-	0.0001
Maternal Complications Rate	*r Value*	-	-	-	-	-
	p Value	-	-	-	-	-

Footnotes:

1. The rates are 10-year (1987-1996) aggregated data for each Health Unit.
2. Low birth weight rate is the number of low birth weight live born babies per 1,000 live births.
3. Pre-term birth rate is the number of live born babies age less than 37 weeks of gestation per 1,000 live births.
4. Teenage birth rate was number of live births born to mothers aged 19 years or younger per 1,000 live births.
5. Perinatal conditions rate was number of live births with perinatal conditions at time of birth per 1,000 live births.
6. Maternal complications rate was number of live births born to mothers with complications per 1,000 live births.
7. The correlation analyses were performed on 10-year aggregated HU-based rates for infant health status indicators and etiological factors.

TABLE 8a:
Correlation Analysis: Infant Mortality Rates and Etiological Factors
By Health Unit Based on 10-year (1981 through 1990) Aggregated Data, British Columbia

Infant Health Status Indicators	Correlation Statistics Indices	Low Birth Weight Rate	Pre-term Birth Rate	Teenage Birth Rate	Perinatal Conditions Rate	Maternal Complications Rate
Infant Mortality Rate	*r Value*	0.3561	0.4642	0.5991	0.0121	-0.3669
	p Value	0.1131	0.0340	0.0041	0.9586	0.1019
Neonatal Mortality Rate	*r Value*	0.3291	0.6372	0.2783	0.2197	-0.0295
	p Value	0.1452	0.0019	0.2218	0.3385	0.8990
Post-neonatal Mortality Rate	*r Value*	0.2692	0.1019	0.7825	-0.2404	-0.6478
	p Value	0.2379	0.6603	0.0001	0.2938	0.0015
Low Birth Weight Rate	*r Value*	-	0.4832	0.2746	0.0970	0.0301
	p Value	-	0.0265	0.2283	0.6759	0.8968
Pre-term Birth Rate	*r Value*	-	-	-0.1888	0.5891	0.4746
	p Value	-	-	0.4123	0.0050	0.0297
Teenage Birth Rate	*r Value*	-	-	-	-0.5511	-0.8398
	p Value	-	-	-	0.0096	0.0001
Perinatal Conditions Rate	*r Value*	-	-	-	-	0.6989
	p Value	-	-	-	-	0.0004
Maternal Complications Rate	*r Value*	-	-	-	-	-
	p Value	-	-	-	-	-

Footnotes:

1. The rates were 10-year (1981-1990) aggregated data for each Health Unit.
2. Low birth weight rate was the number of low birth weight live born babies per 1,000 live births.
3. Pre-term birth rate was the number of live born babies age less than 37 weeks of gestation per 1,000 live births.
4. Teenage birth rate was number of live births born to mothers aged 19 years or younger per 1,000 live births.
5. Perinatal conditions rate was number of live births with perinatal conditions at time of birth per 1,000 live births.
6. Maternal complications rate was number of live births born to mothers with complications per 1,000 live births.
7. The correlation analyses were performed on 10-year aggregated HU-based rates for infant health status indicators and etiological factors.

TABLE 9:

Average Differences between Mortality Ranks and the Ranks by Etiological Foctors By Health Unit, British Columbia, 1987 to 1996 Aggregated Data

Mortality Rate	Low Birth Weight	Pre-Term Birth	Teenage Birth	Perinatal Conditions	Maternal Complications
Infant	48.3	36.3	38.8	56.6	71.5
Neonate	57.7	48.8	63.6	56.5	76.0
Post-Neonate	55.6	45.6	20.7	55.9	81.9

Notes:

1. The value in each cell is the mean square of the rank differences between mortality and etiological factors, including the similarity of Health Units ranked by mortality statistics and etiological factors, and the degree of the association between the two statistics.
2. Both mortality and etiological factors data were aggregated data for 10-year period from 1987 to 1996.

rates were also significantly correlated to pre-term birth rate. This relationship was 7not repeated in the 10-year time period (1987-1996).

Tables 8 and 8a show correlations between Health Unit based infant mortality rates and rates of five etiological factors for two 10-year time periods, 1981 to 1990 and 1987 to 1996, respectively. They suggest a continuing relationship between infant and post-neonatal mortality rates and teenage birth rates, which may be responsible for the consistent geographical inequalities seen in infant mortality rates amongst the 21 Health Units. Table 9 summarizes the rank-differences between infant mortality and the etiological parameter indices. The value in each cell is the sum of squared difference between each infant mortality index and the etiological factor at the Health Unit level. The smallest value in the table, 20.7, is the rank difference between post-neonatal mortality rate and teenage birth rate for each Health Unit. This value further confirms the previous correlation analysis which indicated that at the Health Unit scale the teenage birth rate is most closely related to the rate of post-neonatal mortality.

One major weakness of the correlation analyses presented in Tables 8, 8a and 9 is that they are area-based. As a consequence, the correlation coefficient only indicates that there is an association between mortality rates and the incidences of certain etiological factors. It does not show the strength of that association (Comstock, 1988). That is, the correlation coefficient does not explain how much the variance of the mortality rates amongst the 21 Health Units can be attributed to the incidence of specific etiological factors. In order to establish this, a regression analysis must be conducted between mortality rates and the incidence

rates of those etiological factors. The results derived from such regression analysis are displayed in Table 10.

The dependent variables in this regression are Health Unit specific infant, neonatal, and post-neonatal mortality rates respectively. These rates are 10-year accumulated mortality rates developed from the total infant deaths which in each Health Unit in the period 1987 to 1996, over the average number of live births in each Health Unit during this time. Time aggregation is used to increase the size of the numerator of the rate, and therefore the value of the Health Unit rates. This method is used frequently when dealing with small number variables, such as mortality and morbidity, for certain types of diseases (Foster, Uh, Collison, 1992; Foster & Edgell, 1992). The independent variables are the incidence rates of five etiological factors for each Health Unit. The intent of this regression analysis is to investigate which etiological factors are contributors to the Health Unit specific mortality rate, and how much they contribute to the mortality. In order to find the best regression model by discarding those independent variables which did not associate with the independent variable, a multiple regression model with backward selection of independent variables was established to analyse infant, neonatal and post-neonatal mortality rates separately. In Table 10, the first column represents independent variables selection in the regression analysis, while column 2 indicates which independent variable was excluded from the model at each step. The third column lists the independent variables remaining in the model, after selection at each step, while column 4 shows the value of R-square for the model at each step. This represents the percent of the variance of the dependent variable explained by

TABLE 10:
Multiple Regression Analysis - Backward Selection
Infant, Neonatal, Post-neonatal Mortality Rates and Etiological Variables
By Health Unit with 10-Year Aggregated Data (1987-1996)

Steps of Independent Variables Selection	Independent Variables Removed from Regression	Independent Variables Remained After Each Selection	Regression Model R-Square Value	Statistical Significance (p Value)
Model I: Dependent Variable = Health Unit Infant Mortality Rate				
0	None	Low Birth Weight, Preterm Births, Teenage Births, Perinatal Conditions, Maternal Complications	0.6585	0.0036
1	Low Birth Weight	Preterm Births, Teenage Births, Perinatal Conditions, Marternal Complications	0.6585	0.0012
2	Perinatal Conditions	Preterm Births, Teenage Births, Maternal Complications	0.6482	0.0004
Final Regression Equation*:		***IMR=5.5234+0.1669PRETR+0.0257TEENR-0.0178MARTR. R-square=0.6482, p=0.0004***		
Model II: Dependent Variable = Health Unit Neonatal Mortality Rate				
0	None	Low birth Weight, Preterm Births, Teenage Births, Perinatal Conditions, Maternal Complications.	0.3951	0.1437
1	Low Birth Weight	Preterm Births, Teenage Births, Perinatal Conditions, Maternal Complications.	0.3939	0.0756
2	Perinatal Conditions	Preterm Births, Teenage Births, Maternal Complications.	0.3724	0.0432
3	Teenage Births	Preterm Births, Maternal Complications.	0.3559	0.0191
Final Regression Equation*:		***NMR=4.3101+0.1070PRETR-0.0113MARTR. R-square=0.3559, p=0.0191***		
Model III: Dependent Variable = Health Unit Postneonatal Mortality Rate				
0	None	Low Birth Weight, Preterm Births, Teenage Births, Perinatal Conditions, Maternal Complications	0.6609	0.0034
1	Perinatal Conditions	Preterm Births, Teenage Births, Low Birth Weight, Maternal Complications.	0.6600	0.0011
2	Low Birth Weight	Preterm Births, Teenage Births, Maternal Complications.	0.6588	0.0003
Final Regression Equation*:		***PMR=1.4374+0.0644PRETR+0.0217TEENR-0.0069MARTR. R-square=0.3559, p=0.0191***		

Notes:
IMR - Infant Mortality Rate, NMR - Neonatal Mortality Rate, PMR - Postneonatal Mortality Rate.
PRETR - Preterm Birth Rate, TEENR - Teenage Birth Rate, MARTR - Maternal Complications Rate.

the independent variables, which are included in the model, a measure of the strength of association; the last column in Table 10 contains the p value. If this is less than 0.05, the results and the model as a whole are considered to be statistically significant.

When infant mortality rate is used as a dependent variable and all five etiological variables are included in the model as independent variables, the R^2 value is 0.6585 which is statistically significant (p=0.0036). This can be interpreted to mean that about 66 percent of the total regional variation of infant mortality at the Health Unit level was attributed to difference in the incidence rates of low birth weight, preterm births, teenage births, perinatal conditions, and maternal complications. Further analysis of these five independent variables indicated that two of them, low birth weight and perinatal conditions rates, were not significant. When these two variables were removed from the model, the R square value was only reduced by less than 1 percent. As a result, the best regression model for infant mortality rate could be established using only the three independent variables, preterm birth rate, teenage birth rate, and maternal complications rate. Amongst these variables, preterm birth rate made the greatest contribution, its regression coefficient value being 0.17, in comparison with 0.03 for teenage birth rate and -0.02 for maternal complications rate. The explanatory power was some 64 percent (p=0.0004). Using the same method the final model for neonatal mortality rate was established by only two of the original five dependent variables, specifically preterm birth rate (regression coefficient β=0.11) and maternal complications rate (β=-0.01). The R square was some 36 percent (p=0.0191). The best model for post-neonatal

mortality rate included the dependent variables preterm birth rate (β=0.06), teenage birth rate (β=0.02), and maternal complications rate (β=-0.01). The R square value for this model was some 66 per cent (p=0.0003).

In summary, only three of the five etiological factors were significantly associated with infant mortality rate (including neonatal and post-neonatal mortality) at the Health Unit level. Of these three significant independent variables, preterm birth rate appears to make the most significant contribution to regional inequality in infant mortality rates, at the Health Unit scale. It is followed in significance by teenage births rate and the incidence rate of maternal complications. The most interesting conclusion to be drawn from these results appears to be that teenage birth rate is associated with post-neonatal mortality rate but not with neonatal mortality rate. In addition, low birth weight rate does not seem to be related to any of the three mortality indices, although it shows a strong correlation with preterm rate.

6.4 Ecological Factors and Geographical Inequalities in Infant Death Rates

The hypothesis put forward to explain the relationship between ecological factors and regional infant health status is that a local region has been constructed, physically and socio-economically, by a set of structural factors. These factors represent the ecological setting for a particular region, which in turn shapes the health status of its infant population. To illustrate, Bird and Bauman (1995) claimed that a substantial portion of the variance in state-level infant mortality can be accounted for by states' structural characteristics, which are represented by the

proportion of Black and Hispanic individuals, the proportion of the population that has been educated to a specific level, and the proportion of population in professional and managerial occupations. It would appear that although such variables are mediated by health services, they are still responsible for a certain portion of variance in infant mortality. If Bird and Bauman (1995) are correct, relationships between British Columbia specific selected ecological (structural) factors and mortality rates need to be investigated further. In this study, ecological variables were selected for analyses based on the availability of data sources. This resulted in the use of a total of 19 variables representing regional employment, education and income, family structure (single parent), ethnicity, and health care services. Chapter Five provides a detailed list of the ecological variables included in this study.

The first step of the analysis involved explorations at the macro-level of the relationship between infant mortality (including neonatal and post-neonatal) rates and those selected ecological factors, for each Health Unit. Ranking comparison, previously described in Section 6.3, was again used for this purpose. Each Health Unit was ranked by three mortality rates (infant, neonatal and post-neonatal mortality), respectively, creating three ranks for each Health Unit. Each Health Unit was then ranked by those 19 ecological variables separately, creating a further 19 ranks for each Health Unit. The 19 eco-ranks for each Health Unit were then averaged to develop one mean eco-rank for that Health Unit. These data for the 21 Health Units was then plotted on an XY chart, with the X-axis representing the mean eco-rank for each Health Unit and the Y-axis the rank of Health Unit by infant, or

neonatal or post-neonatal mortality rate. These charts are Figures 16, 17 and 18 which display infant mortality, neonatal mortality, and post-neonatal mortality, respectively. These figures generally indicate a positive relationship between these two sets of ranks, indicating that higher infant mortality rates tend to be associated with the worst ecological settings, that is in those Health Units with the highest eco-ranks.

These relationships were then further explored using regression analysis. The analysis used mortality rates as dependent variables, which were assumed to be linearly related to all the 19 ecological factors which are the independent variables. In this manner the regression models show how much of the variance in mortality rates amongst the 21 Health Units can be attributed to each individual ecological factor, and all of these factors as a whole. Since there are so many ecological variables a stepwise regression approach was used in order to select those variables making the most significant contribution to the model. Each variable was introduced independently and its significance to the regression model assessed. It remained in the model only if it was statistically significant. The next variable was then introduced to the model, and the significance of each of the two variables was then assessed. Only the most significant variable remained. This selection method was continued until all the variables had been assessed. As a result, only the variable making the most significant contribution to the regression model remained, and was used to build the final regression model. Table 11 shows this step-by-step selection and associated R^2 values and statistical significance (p values).

In model one, the dependent variable being analysed is the Health Unit

Figure 16: Ranks of Health Units by Ecological Factors and Infant Mortality Rate From 1987 to 1996, British Columbia

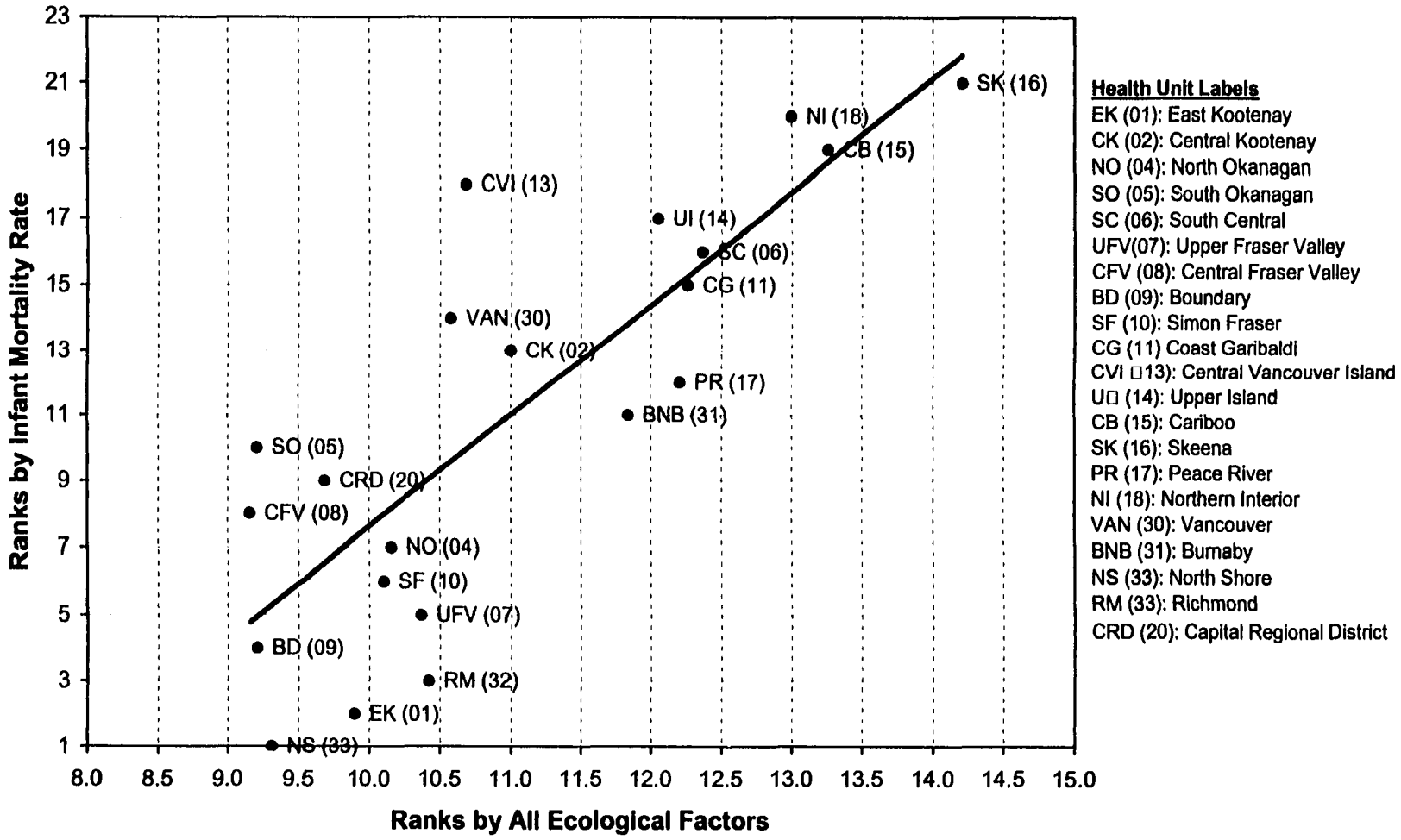


Figure 17: Ranks of Health Units by Ecological Factors and Neonatal Mortality Rate From 1987 to 1996, British Columbia

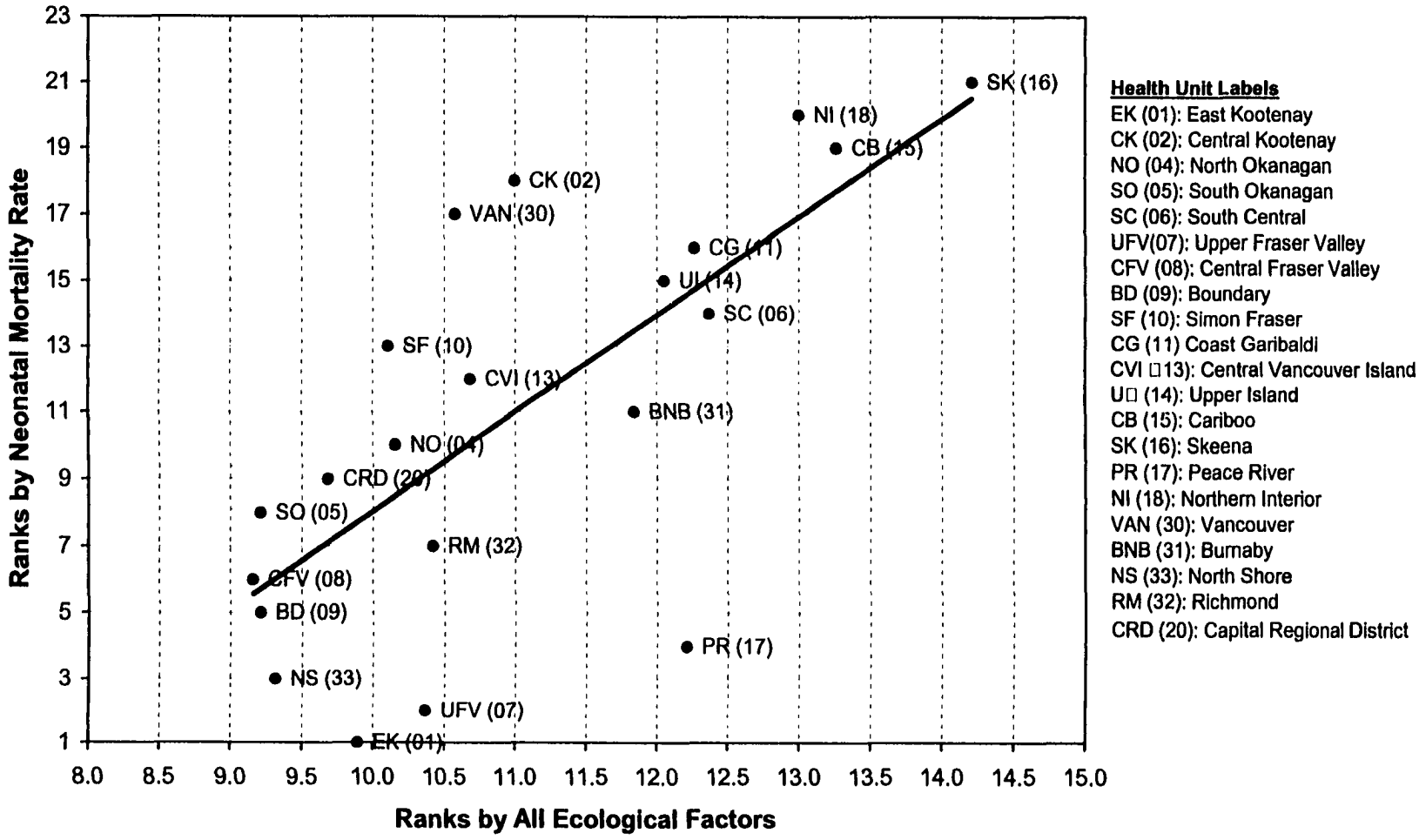
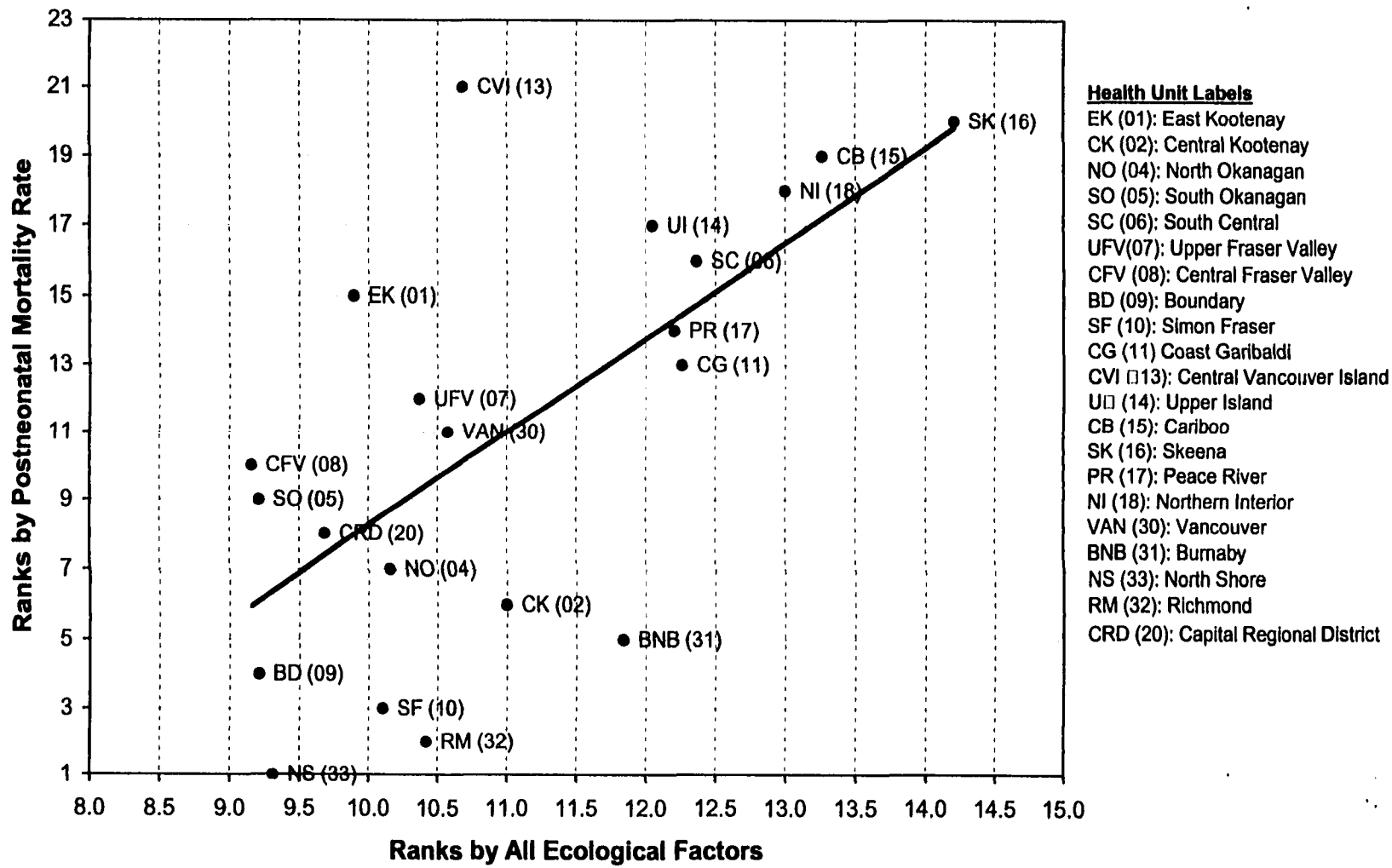


Figure 18: Ranks of Health Units by Ecological Factors and Postneonatal Mortality Rate From 1987 to 1996, British Columbia



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CVI □13): Central Vancouver Island
U□ (14): Upper Island
CB (15): Cariboo
SK (16): Skeena
PR (17): Peace River
NI (18): Northern Interior
VAN (30): Vancouver
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mortality rate. Included in the six selected independent variables from the original 19 ecological factors were female life expectancy, percent of the population with employment income, crude death rate, percent of the population who were Native Indians. The variable, percentage of families with two parents, was initially selected, but was finally removed because it became insignificant, once other variables were introduced. The adjusted R^2 for the model was some 80 percent with p values remaining at the significant level, 0.0001. The ecological variables included in the final regression model, which was established based on the six selected ecological variables, were percentage of the population with employment income (β=-1.7599, p=0.0140), female life expectancy (β=-11.4092, p=0.0005) and the percentage of the population receiving unemployment insurance (β=2.1449, p=0.0826). The model's R^2 was 73 percent and the p value 0.0001. This result indicates that the 73 percent of the variations in infant mortality rate, amongst the 21 Health Units, can be attributed to different incidences of these ecological factors.

Model two represents the relationship between neonatal mortality rate and the selected ecological factors. The dependent variable in this model is the Health Unit neonatal mortality rate. Only three independent variables amongst the original 19 ecological factors were selected to explain this, namely female life expectancy, the percentage of children with two parents, and the percentage of population receiving unemployment insurance. The adjusted R^2 of the model was 37 percent, with p values remaining at 0.01 significant level. However, all three ecological variables were not statistically significant if assessed using 0.05 or 0.01 p values, although they were all marginal. For instance, the significance of the β value (-3.4953) of

TABLE 11:
Multiple Regression Analysis - Stepwise Selection
Infant, Neonatal, and Postneonatal Mortality Rates and Ecological Factors
By Health Units with 10 Years Aggregated Data (1987-1996)

Selection Steps	Independent Variables Added/Removed in the Regression After each Selection Step	R-Square Value	p Value
	Model I: Dependent Variable = Health Unit Infant Mortality Rate		
1	Initial Selection: Percent population under unemployment Insurance.	0.4821	0.0003
2	Added: Percent of families with two parents.	0.6394	0.0001
3	Added: Female life expectancy.	0.7048	0.0001
4	Added: Percent population with employment income.	0.7403	0.0001
5	Added: Crude death rate.	0.7795	0.0001
6	Removed: Percent of families with two parents.	0.7670	0.0001
7	Added: Percent population of Native Indians.	0.7973	0.0001
	Ecological variables included in the final regression model:		
	PCTICEMP - Percent population with employment income:	Beta=-1.7599	p=0.0140
	FLIFEEXP - Female life expectancy:	Beta=-11.4092	p=0.0005
	PCTPOPUI - Percent population under unemployment insurance:	Beta=2.1449	p=0.0826
	R-Square of the model= 0.7297; p-value=0.0001.		
	Model II: Dependent Variable = Health Unit Neonatal Mortality Rate		
1	Initial Selection: Female life expectancy.	0.2703	0.0131
2	Added: Percent of children with two parents.	0.3585	0.0147
3	Added: Percent population under unemployment insurance.	0.4678	0.0087
	Ecological variables included in the final regression model:		
	FLIFEEXP - Female life expectancy:	Beta=-3.4953	p=0.0911
	PCTKID2P - Percent of children with 2 parents:	Beta=-1.4196	p=0.0668
	PCTPOPUI - Percent population under umemployment insurance:	Beta=1.5113	p=0.0784
	R-Square of the model= 0.3743; p-value=0.0116.		
	Model III: Dependent Variable = Health Unit Postneonatal Mortality Rate		
1	Initial Selection: Percent population under unemployment Insurance.	0.4474	0.0007
2	Added: Female life expectancy.	0.5364	0.0007
	Ecological variables included in the final regression model:		
	FLIFEEXP - Female life expectancy:	Beta=-3.4625	p=0.0791
	PCTPOPUI - Percent population under umemployment insurance:	Beta=2.2440	p=0.0086
	R-Square of the model= 0.4854; p-value=0.0010.		

Notes:

1. Whether a variable is removed and/or added is controlled by a pre-set probability that indicates the significance of the variable's contribution to total variance of mortality rates by Health Unit. The default probability in SAS program to keep a variable in the regression model is 0.15. Therefore, not all variable finally kept in the model are significant at 0.05 level in their contributions to total variance of the mortality rates by Health Units.
2. The R-Square values indicate the percent of the total variances of mortality rates by Health Unit explained by those independent variables included in the regression models.
3. The p-values indicate statistical significance of the model as a whole, not each independent variable.
4. The ecological variables included in the final regression model were selected after introducing those remained variables selected in stepwise regression to regression model.

female life expectancy was 0.0911, that for the β value (-1.4196) of percentage of children with 2 parents was 0.0668, and that for the β value (1.5113) of percentage population receiving unemployment insurance was 0.0784. The final regression model based on these three variables was statistically significant, with a p value of 0.0116. These data suggest that ecological factors do not contribute as much to neonatal mortality rate as they do to infant mortality rate. Geographical inequality in neonatal mortality rate does not seem to be as closely associated with regional ecological settings as it does to infant mortality rate.

Only two independent variables were selected for modelling post-neonatal mortality rates by initial stepwise regression analysis. These were the percentage of the population receiving unemployment insurance and female life expectancy. However, these two variables explained 54 percent of the total variance of the regional inequality of this mortality rate. Indeed, the final model included only one variable, the percentage of the population receiving unemployment insurance. This variable alone contributed, significantly, some 50 percent of the total variance of post-neonatal mortality rate amongst the 21 Health Units.

These multiple regression analysis of ecological variables and infant mortality rates at the Health Unit scale revealed that two, out of the original 19 ecological variables, were the major determinants of regional inequalities in the three infant mortality rates. These two variables were the percentage of the population with employment income and the percentage of the population receiving unemployment insurance. Both these variables were a general measure of the economic status of the Health Unit. The results as a whole indicated that the regional economic status

seems more closely related to post-neonatal mortality rate than to neonatal mortality rate.

A significant problem with the previous multiple regression analyses is that the original 19 ecological variables are themselves inter-correlated (Table 15). Unfortunately, such correlations between independent variables may invalidate the assumptions on which regression analysis is based because this statistical technique requires that there is no correlation between independent variables. Using a set of inter-correlated independent variables to conduct multiple regression can result in multicollinearity, which produces misleading coefficients for intercorrelated variables. It is necessary, therefore, to remove the intercorrelations amongst the original 19 variables (Clark & Hosking, 1986). This can be achieved by the use of factor analysis. Factor analysis can be used to group together the original ecological variables on the basis of their intercorrelations. A representative factor can then be extracted from each group of variables. As a result, these extracted factors are not correlated. Table 12 summarizes the results of the factor analysis performed using the original 19 ecological variables.

Several steps are involved in factor analysis. In step one, as showed at the top of Table 12, the eigenvalue of each factor is calculated based on the correlation matrix of the original variables. Simply put, the eigenvalue indicates how many original variables this factor is equivalent to. For instance, the eigenvalue of factor 1, in table 12, is 5.93. This indicates that factor 1 is equivalent to almost 6 of the original ecological variables, that is factor 1 contains the full information represented by six of the original ecological variables. Therefore, whether a factor from the

correlation matrix is used in further analysis depends on its eigenvalue. Theoretically, there can be as many factors as original variables. However, significant factors are those whose eigenvalues are greater than 1. Based on this criterion, a total of 4 new factors were selected. These explained about 85 percent of total variations within the original 19 variables. That is, these 4 factors contain 85 percent of the information measured by the original factors and were the equivalent of 10 original variables. This illustrates significant intercorrelations between the original 19 ecological variables, since 85 percent of the information they contained can be explained by 4 newly formed factors.

After these 4 new factors were identified, the next step was to build a factor pattern or structure. The middle section of table 12 displays this factor pattern. The value in each cell gives an estimated strength of association between a factor and one original variable. For instance, the value 0.8986, located in the first cell of the middle section, indicates that there is a close association between factor 1 and the original variable, capacities for child care in 1993. A comparison of all values in each column identifies which original variables are mostly closely represented by each factor. The values in the last column, Final Communality, represents the percentage of one of the original variables represented by those selected factors. To illustrate, the figure 0.8877, the first value in Final Communality column, indicates that almost 89 percent of the information contained in the first variable, Capacities for child care in 1993, has been explained by these four factors. In general, the middle section of table 12 indicates that most of the original ecological variables can be represented by the four selected factors. However, there is no

TABLE 12:
Factor Analysis for Ecological Variables
By Health Unit, British Columbia

Step One: Eigenvalues of the Correlation Matrix

Factors	Eigenvalue	Difference Between Successive Eigenvalues	Proportion of Variation	Cumulative Proportion of Variation	Variance Explained by Each Factor
1	5.9314	2.9901	0.3954	0.3954	5.9314
2	2.9413	0.8879	0.1961	0.5915	2.9413
3	2.0534	0.9568	0.1369	0.7284	2.0534
4	1.0966	0.3261	0.0731	0.8015	1.0966
5	0.7706	0.0975	0.0514	0.8529	...
...	...	...	...	...	...

** 4 factors will be retained by the Min-Eigenvalue criterion (eigenvalue>1).*

Step Two: Factor Pattern and Communality - Without Rotation

Original Variables	Factor				Final Communality
	1	2	3	4	
Capacities for child care in 1993	0.8986	-0.1093	0.2474	0.0840	0.8877
Percent of children with one parent	0.8295	0.3786	-0.2129	-0.2942	0.9633
GP FTEs per 1,000 population in 1991	0.7936	0.2905	-0.1386	0.1877	0.7686
Number of grade 12 enrollment in 1992	0.7732	-0.2011	0.2433	0.1548	0.7214
Percent of population speaking English	-0.5712	0.1784	-0.5097	0.2415	0.6762
Percent of income from employment	-0.7219	0.1498	0.5934	0.0743	0.9012
Percent of children with two parents	-0.8222	-0.3908	0.2201	0.2974	0.9655
Acute care beds per 1,000 population in 1991	0.3523	0.7189	0.1058	0.3508	0.7751
Population receiving unemployment insurance	-0.5348	0.6904	-0.1977	0.0635	0.8058
Percent of Native Indians population	-0.4746	0.6356	0.2000	-0.2390	0.7264
Percent of grade 12 graduation in1992	0.2375	-0.5556	0.1037	0.4272	0.5583
Average number of children per family	-0.5856	0.2940	0.7057	0.1000	0.9373
Average individual income	0.3316	-0.4839	0.5124	-0.1637	0.6335
Percent of families with two parents	-0.5646	-0.4852	-0.5839	0.2276	0.9469
Extended care beds per 1,000 population in 1991	0.4706	0.4383	0.0752	0.5799	0.7555

Step Three: Factor Pattern and Communality - Orthogonal Factor Rotation (Varimax)

Original Variables	Factor				Final Communality
	1	2	3	4	
Percent of income from employment	0.8977	0.0231	-0.0706	0.2997	0.9012
Average number of children per family	0.8831	-0.0964	0.1122	0.3683	0.9373
Percent of children with two parents	0.8113	0.3284	-0.3712	-0.2484	0.9655
Percent of children with one parent	-0.8099	-0.3389	0.3699	0.2360	0.9633
Percent of population speaking English	0.1163	0.8067	-0.0616	0.0907	0.6762
Percent of families with two parents	0.0851	0.7256	-0.4951	-0.4099	0.9469
Number of grade 12 enrollment in 1992	-0.3001	-0.5830	0.3409	-0.4186	0.7214
Capacities for child care in 1993	-0.4083	-0.6637	0.4077	-0.3380	0.8877
Average individual income	0.0844	-0.7060	-0.1822	-0.3077	0.6335
Extended care beds per 1,000 population in 1991	-0.0970	-0.0165	0.8539	-0.1287	0.7555
Acute care beds per 1,000 population in 1991	-0.0826	-0.0047	0.8393	0.2528	0.7751
GP FTEs per 1,000 population	-0.5770	-0.2120	0.6163	-0.1043	0.7686
Percent of Native Indians population	0.3489	0.1489	0.0770	0.7593	0.7264
Population receiving unemployment insurance	0.2207	0.5921	0.2125	0.6012	0.8058
Percent of grade 12 graduation in 1992	0.0699	-0.1539	0.0404	-0.7267	0.5583

TABLE 13:
Multiple Regression Analysis - Stepwise Selection
Relationships between Selected Etiological Factors and Ecological Factors
By Health Unit, British Columbia

Selection Steps	Independent Variables Added/Removed in the Regression After each Selection Step		R-Square Value	p Value
Dependent Variable = Health Unit Preterm Birth Rate				
1	Initial Selection:	Number of GP FTEs per 1,000 population in 1991.	0.4209	0.0011
2	Added:	Average number of children per family.	0.5178	0.0001
3	Added:	Percent population with employment income.	0.6315	0.0004
4	Added:	Crude death rate.	0.7002	0.0002
5	Added:	Female life expectancy.	0.7528	0.0002
6	Added:	Number of extended care beds per 1,000 population in 1991.	0.7937	0.0002
7	Added:	Number of acute care beds per 1,000 population in 1991.	0.8359	0.0001
8	Added:	Percent of grade 12 graduation in 1991.	0.8745	0.0001
Final	Added:	Crude birth rate.	0.8993	0.0001
Dependent Variable = Health Unit Teenage Birth Rate				
1	Initial Selection:	Percent population receiving UI.	0.7150	0.0001
2	Added:	Percent population speaking English.	0.7817	0.0001
3	Added:	Percent families with two parents.	0.8404	0.0001
4	Added:	Average number of children per family.	0.8597	0.0001
5	Added:	Percent population with employment income.	0.9093	0.0001
6	Added:	Percent of grade 12 graduation in 1992.	0.9296	0.0001
7	Added:	Number of acute care beds per 1,000 population in 1991.	0.9436	0.0001
8	Removed:	Percent families with two parents.	0.9422	0.0001
9	Added:	Number of extended care beds per 1,000 population in 1991.	0.9533	0.0001
Final	Added:	Male life expectancy.	0.9624	0.0001
Dependent Variable = Health Unit Maternal Complications Rate				
Final	Initial Selection:	Number of GP FTEs per 1,000 population in 1991.	0.5233	0.0001

Notes:

1. Whether a variable is removed and/or added is controlled by a pre-set probability that indicates the significance of the variable's contribution to total variance of mortality rates by Health Unit. The default probability in SAS program to keep a variable in the regression model is 0.15. Therefore, not all variable finally kept in the model are significant at 0.05 level in their contributions to total variance of the mortality rates by Health Units.
2. The R-Square values indicate the percent of the total variances of mortality rates by Health Unit explained by those independent variables included in the regression models.
3. The p-values indicate statistical significance of the model as a whole, not each independent variable.

TABLE 14:
Multiple Regression Analysis
Relationships between Infant Mortality Rates and Rotated Ecological Factors
By Health Unit, British Columbia

Dependent Variable	Independent Variables (Rotated Factors Extracted from 15 Original Ecological Variables)	R-Square Value	p Value
Infant Mortality Rate (1/1,000)	Factor 1 (Representing Family Structure - Number of Children, Parents, etc.)	1.208	0.6615
	Factor 2 (Representing Regional Social Profile - Grade 12 graduation, etc.)	-1.013	0.6910
	Factor 3 (Representing Health Care Services and Resources - GP FTEs, etc.)	4.0493	0.1435
	Factor 4 (Representing Population Status - Percent Native Indians, UI, etc.)	6.2641	0.0090
Final Regression Model: R-square = 0.2859, p-value = 0.0435.			
Neonatal Mortality Rate (1/1,000)	Factor 1 (Representing Family Structure - Number of Children, Parents, etc.)	-0.1897	0.9148
	Factor 2 (Representing Regional Social Profile - Grade 12 graduation, etc.)	-1.0860	0.5101
	Factor 3 (Representing Health Care Services and Resources - GP FTEs, etc.)	1.4017	0.4214
	Factor 4 (Representing Population Status - Percent Native Indians, UI, etc.)	3.3953	0.0240
Final Regression Model: R-square = 0.1411, p-value = 0.1635.			
Postneonatal Mortality Rate (1/1,000)	Factor 1 (Representing Family Structure - Number of Children, Parents, etc.)	1.3973	0.4584
	Factor 2 (Representing Regional Social Profile - Grade 12 graduation, etc.)	0.0723	0.9663
	Factor 3 (Representing Health Care Services and Resources - GP FTEs, etc.)	2.6496	0.1584
	Factor 4 (Representing Population Status - Percent Native Indians, UI, etc.)	2.8687	0.0635
Final Regression: R-square = 0.1413, p-value = 0.1633.			

Notes:

1. Factor 1 has major loadings on the original variables of percent of income from employment, average number of children per family, percent of children with two parents, and percent of children with one parent.
2. Factor 2 has major loadings on the original variables of percent of population speaking English, percent of family family with two parents, number of grade 12 enrollment in 1992, capacities for child care in 1993, average individual income.
3. Factor 3 has major loadings on the original variables of number of extended care beds per 1,000 population in in 1991, number of acute care beds per 1,000 population in 1991, number of GP FTEs per 1,000 population in 1991.
4. Factor 4 has major loadings on the original variables of percent of Native Indians population, percent of population receiving unemployment insurance, percent of grade 12 graduation in 1992.

TABLE 15: Correlation Analysis amongst 19 Ecological Variables by Health Unit, British Columbia

Ecological Variables	Ecological Variables																		
	ACRT91	AVGINDIC	PCTICEMP	AVGNOKID	CPCC1993	CRUDEBR	CRUDEDR	ECRT91	G12ENR92	G12GRD92	GFRT1991	PCENTNA	FLIFEEXP	MLIFEEXP	PCNTENGL	PCNTTWOP	PCTKID1P	PCTKID2P	PCTPOPUI
01. ACRT91	1.0000	-0.1611	-0.0887	0.0986	0.2316	-0.0189	0.0730	0.5591	0.1072	-0.1312	0.5730	0.1559	-0.1239	-0.4842	-0.0922	-0.5247	0.4158	-0.4207	0.2975
	0.0000	0.4853	0.7023	0.6707	0.3123	0.9353	0.7531	0.0084	0.6438	0.5709	0.0068	0.4998	0.5927	0.0201	0.6909	0.0146	0.0608	0.0576	0.1903
02. AVGINDIC	-0.1611	1.0000	-0.0136	-0.0769	0.4013	0.0737	-0.1129	-0.0661	0.3108	0.3055	0.1695	-0.2122	0.2124	0.3020	-0.4129	-0.2491	0.0172	-0.0095	-0.6746
	0.4853	0.0000	0.9534	0.7405	0.0714	0.7508	0.6261	0.7760	0.1704	0.1781	0.4625	0.3558	0.3553	0.1834	0.0628	0.2762	0.9409	0.9675	0.0008
03. PCTICEMP	-0.0887	-0.0136	1.0000	0.8592	-0.5144	0.8322	-0.9049	-0.1327	-0.4497	-0.2430	-0.5929	0.4657	-0.5541	-0.5814	0.2005	-0.0108	-0.6765	0.6740	0.3281
	0.7023	0.9534	0.0000	0.0001	0.0170	0.0001	0.0001	0.5665	0.0408	0.2884	0.0046	0.0334	0.0092	0.0057	0.3836	0.9628	0.0008	0.0008	0.1464
04. AVGNOKID	0.0986	-0.0769	0.8592	1.0000	-0.3383	0.8401	-0.8822	-0.0742	-0.2681	-0.1787	-0.4900	0.5679	-0.4060	-0.6916	0.0536	-0.2069	-0.5479	0.5504	0.3912
	0.6707	0.7405	0.0001	0.0000	0.1337	0.0001	0.0001	0.7493	0.2400	0.4383	0.0242	0.0072	0.0679	0.0005	0.8174	0.3681	0.0101	0.0097	0.0795
05. CPCC1993	0.2316	0.4013	-0.5144	-0.3383	1.0000	-0.1704	0.4286	0.4209	0.9476	0.2669	0.6279	-0.4214	0.1483	0.0561	-0.5516	-0.5528	0.6195	-0.6048	-0.5922
	0.3123	0.0714	0.0170	0.1337	0.0000	0.4602	0.0526	0.0574	0.0001	0.2422	0.0023	0.0571	0.5212	0.8092	0.0095	0.0093	0.0027	0.0037	0.0047
06. CRUDEBR	-0.0189	0.0737	0.8322	0.8401	-0.1704	1.0000	-0.8112	-0.0281	-0.0851	-0.1022	-0.4672	0.4868	-0.4466	-0.6802	-0.0385	-0.2282	-0.4999	0.5018	0.2392
	0.9353	0.7508	0.0001	0.0001	0.4602	0.0000	0.0001	0.9037	0.7137	0.6593	0.0327	0.0252	0.0424	0.0007	0.8685	0.3198	0.0210	0.0205	0.2964
07. CRUDEDR	0.0730	-0.1129	-0.9049	-0.8822	0.4286	-0.8112	1.0000	0.1849	0.3595	0.2234	0.5498	-0.5601	0.4212	0.5003	-0.1235	0.1196	0.6063	-0.6037	-0.3554
	0.7531	0.6261	0.0001	0.0001	0.0526	0.0001	0.0000	0.4223	0.1094	0.3304	0.0098	0.0083	0.0573	0.0209	0.5937	0.6057	0.0036	0.0038	0.1139
08. ECRT91	0.5591	-0.0661	-0.1327	-0.0742	0.4209	-0.0281	0.1849	1.0000	0.3279	-0.0283	0.5623	-0.0663	-0.0188	-0.3653	-0.0562	-0.3803	0.3801	-0.3832	-0.0498
	0.0084	0.7760	0.5665	0.7493	0.0574	0.9037	0.4223	0.0000	0.1468	0.9031	0.0080	0.7751	0.9357	0.1034	0.8090	0.0890	0.0892	0.0864	0.8303
09. G12ENR92	0.1072	0.3108	-0.4497	-0.2681	0.9476	-0.0851	0.3595	0.3279	1.0000	0.3592	0.4444	-0.4157	0.1542	0.0208	-0.4733	-0.4248	0.4726	-0.4535	-0.5129
	0.6438	0.1704	0.0408	0.2400	0.0001	0.7137	0.1094	0.1468	0.0000	0.1098	0.0435	0.0609	0.5045	0.9285	0.0302	0.0550	0.0305	0.0390	0.0174
10. G12GRD92	-0.1312	0.3055	-0.2430	-0.1787	0.2669	-0.1022	0.2234	-0.0283	0.3592	1.0000	0.0596	-0.4130	0.3376	0.0821	-0.2528	0.1000	-0.0981	0.1046	-0.3140
	0.5709	0.1781	0.2884	0.4383	0.2422	0.6593	0.3304	0.9031	0.1098	0.0000	0.7975	0.0628	0.1344	0.7235	0.2689	0.6663	0.6722	0.6519	0.1657
11. GFRT1991	0.5730	0.1695	-0.5929	-0.4900	0.6279	-0.4672	0.5498	0.5623	0.4444	0.0596	1.0000	-0.2234	0.1133	0.0932	-0.3028	-0.4195	0.6887	-0.6914	-0.1838
	0.0068	0.4625	0.0046	0.0242	0.0023	0.0327	0.0098	0.0080	0.0435	0.7975	0.0000	0.3304	0.6249	0.6879	0.1821	0.0584	0.0006	0.0005	0.4252
12. PCENTNA	0.1559	-0.2122	0.4657	0.5679	-0.4214	0.4868	-0.5601	-0.0663	-0.4157	-0.4130	-0.2234	1.0000	-0.3652	-0.4716	0.3170	-0.1613	-0.1242	0.1103	0.6845
	0.4998	0.3558	0.0334	0.0072	0.0571	0.0252	0.0083	0.7751	0.0609	0.0628	0.3304	0.0000	0.1036	0.0309	0.1615	0.4848	0.5917	0.6342	0.0008
13. FLIFEEXP	-0.1239	0.2124	-0.5541	-0.4060	0.1483	-0.4466	0.4212	-0.0188	0.1542	0.3376	0.1133	-0.3652	1.0000	0.5656	-0.2382	0.3133	-0.0103	0.0142	-0.4348
	0.5927	0.3553	0.0092	0.0679	0.5212	0.0424	0.0573	0.9357	0.5045	0.1344	0.6249	0.1036	0.0000	0.0075	0.2985	0.1667	0.9646	0.9513	0.0489
14. MLIFEEXP	-0.4842	0.3020	-0.5814	-0.6916	0.0561	-0.6802	0.5003	-0.3653	0.0208	0.0821	0.0932	-0.4716	0.5656	1.0000	-0.1402	0.4103	0.1129	-0.1208	-0.4700
	0.0261	0.1834	0.0057	0.0005	0.8092	0.0007	0.0209	0.1034	0.9285	0.7235	0.6879	0.0309	0.0075	0.0000	0.5444	0.0647	0.6260	0.6019	0.0318
15. PCNTENGL	-0.0922	-0.4129	0.2005	0.0536	-0.5516	-0.0385	-0.1235	-0.0562	-0.4733	-0.2528	-0.3028	0.3170	-0.2382	-0.1402	1.0000	0.5929	-0.3305	0.3315	0.4699
	0.6909	0.0628	0.3836	0.8174	0.0095	0.8685	0.5937	0.8090	0.0302	0.2689	0.1821	0.1615	0.2985	0.5444	0.0000	0.0046	0.1434	0.1421	0.0316
16. PCNTTWOP	-0.5247	-0.2491	-0.0108	-0.2069	-0.5528	-0.2282	0.1196	-0.3803	-0.4248	0.1000	-0.4195	-0.1613	0.3133	0.4103	0.5929	1.0000	-0.6255	0.6219	0.0753
	0.0146	0.2762	0.9628	0.3681	0.0093	0.3198	0.6057	0.0890	0.0550	0.6663	0.0584	0.4848	0.1667	0.0647	0.0046	0.0000	0.0024	0.0026	0.7456
17. PCTKID1P	0.4158	0.0172	-0.6765	-0.5479	0.6195	-0.4999	0.6063	0.3801	0.4726	-0.0981	0.6887	-0.1242	-0.0103	0.1129	-0.3305	-0.6255	1.0000	-0.9986	-0.1557
	0.0608	0.9409	0.0008	0.0101	0.0027	0.0210	0.0036	0.0892	0.0305	0.6722	0.0008	0.5917	0.9646	0.6260	0.1434	0.0024	0.0000	0.0001	0.5003
18. PCTKID2P	-0.4207	-0.0095	0.6740	0.5504	-0.6048	0.5018	-0.6037	-0.3832	-0.4535	0.1046	-0.6914	0.1103	0.0142	-0.1208	0.3315	0.6219	-0.9986	1.0000	0.1399
	0.0576	0.9675	0.0008	0.0097	0.0037	0.0205	0.0038	0.0864	0.0390	0.6519	0.0006	0.6342	0.9513	0.6019	0.1421	0.0026	0.0001	0.0000	0.5453
19. PCTPOPUI	0.2975	-0.6746	0.3281	0.3912	-0.5922	0.2392	-0.3554	-0.0498	-0.5129	-0.3140	-0.1838	0.6845	-0.4348	-0.4700	0.4699	0.0753	-0.1557	0.1399	1.0000
	0.1903	0.0008	0.1464	0.0795	0.0047	0.2964	0.1139	0.8303	0.0174	0.1657	0.4252	0.0006	0.0489	0.0316	0.0316	0.7456	0.5003	0.5453	0.0000

Detailed Descriptions of Ecological Variables

01. ACRT91: Acute care beds per 1,000 population in 1991.
02. AVGINDIC: Average individual income.
03. PCTICEMP: Percent of population with employment income.
04. AVGNOKID: Average number of children per family.
05. CPCC1993: Capacities for child care in 1993
06. CRUDEBR: Crude birth rate.
07. CRUDEDR: Crude death rate.
08. ECRT91: Extended care beds per 1,000 population in 1991.
09. G12ENR92: Grade 12 enrollment in 1992.
10. G12GRD92: Grade 12 gradulation in 1992.
11. GFRT1991: GP FTEs per 1,000 population in 1991.
12. PCENTNA: Percent of population with Native Indian status.
13. FLIFEEXP: Female life expectancy.
14. MLIFEEXP: Male life expectancy.
15. PCNTENGL: Percent of population speaking English.
16. PCNTTWOP: Percent of families with two parents.
17. PCTKID1P: Percent of children with 1 parent.
18. PCTKID2P: Percent of children with 2 parents.
19. PCTPOPUI: Percent of population under UI.

: indicating significant correlations at 0.05 or 0.01 level.

clear indication of which of the original ecological variables are represented by any one specific factor. This problem is overcome by step three of the factor analysis.

In this step, the selected four factors are rotated. As a result, each factor can specifically represent certain original variables. The bottom section in Table 12 displays the orthogonally rotated factor pattern or structure. From this pattern, it is clear that the first factor largely represents four original ecological variables, specifically the percentage of income from employment, the average number of children per family, the percentage of children with two parents, and the percentage of children with one parent. The second factor is mainly loaded by five original variables. These are the percentage of the population speaking English, the percentage of families with two parents, the number of grade 12 enrollments in 1992, the capacities for child care in 1993, and the average individual income. The third factor has major loadings on three original variables, extended care beds per 1,000 population in 1991, acute care beds per 1,000 population in 1991, and the number of full-time equivalent general practitioners per 1,000 population. The last factor largely represents three original variables, namely the percentage of Native Indians in the population, the percentage of the population receiving unemployment insurance and the percentage of grade 12 graduations in 1992.

It can been seen in summary that the first factor mainly measures family status, the second mainly the population characteristics of the Health Unit, the third factor chiefly represents health care resources and their availability within the Health Unit, and the last factor represents the employment and education status of the Health Unit. Table 14 then presented the results obtained from multiple regression

analysis derived from the three infant mortality statistics and the four selected ecological factors. Amongst the four factors, only one, factor 4, representing the percentage of Aboriginals in the population and level of unemployment, is significantly correlated to all three mortality statistics. This relationship confirms the results from previously described multiple regression analyses (Table 11), which indicated that the unemployment status of the population made a significant contribution to regional variations in all three mortality rates. In this factor regression analysis, the R^2 values obtained were 0.2856, 0.1411, and 0.1413 for infant, neonatal, and post-neonatal mortality rates respectively. It seems, therefore, that Aboriginal population, unemployment, and education level are the three major ecological variables which make the largest contributions to geographical inequalities in infant mortality rates at the Health Unit level. This contribution, however, is relatively modest, only some 28 percent.

6.5 Interactions between Ecological and Etiological Variables

The main objective of this study is to investigate geographical inequalities of infant mortality rates amongst all 21 Health Units. As discussed previously, it is clear that geographical inequalities in all three infant mortality statistics, measured by weighted coefficients of variation (WCV), have not followed the declining trends experienced by the three corresponding provincial infant mortality rates, during the past three decades. It is hypothesized that this phenomenon occured because those etiological causal factors for infant mortality rates vary significantly among these 21 Health Units. Such variations in the prevalence rates of etiological factors

are likely to be determined, partially, by the specific ecological setting of each Health Unit. If this is true, there must exist a very close correlation between the prevalence rates of etiological factors and the specific ecological characteristics of each Health Unit. In order to illustrate the existence of such relationships between these two sets of factors, analyses which are able to identify such interactions must be performed. One such method is canonical correlation. This type of correlation differs from simple one-dependent variable correlation analysis in that it can be used to perform correlation analysis on two groups of variables. To illustrate, it can be used in correlations between etiological and ecological variable groups. In canonical correlation, a pair of factors, canonical variables, are extracted from the two original variable sets, and correlation performed between them. Each canonical variable has a linear association with all of the original variables in each variable group. This relationship is represented by correlation coefficients between this extracted factor and all original variables in each group. Then, a second pair of factors are extracted from the two original variable sets, and correlation is performed. It should be noted that the second factor is only extracted from the original variables after the first factor has been removed. Such extraction continues until all canonical variables are extracted. Theoretically, the number of canonical variables is equal to the number of original variables. The advantage of canonical variables is that each of them represents "pure" characteristics, intrinsically included in each of the original variables. The correlation between each pair of canonical variables, therefore, identifies the most common aspects of the two variable groups, and, as a result, is more meaningful in explaining relationships between the original

variable sets.

Table 16 illustrates the results obtained from canonical correlation between the etiological and ecological variables. The top portion of the table presents the canonical structure, and correlations between the original and canonical variables. Five canonical variables have been created from the five original etiological variables. However, only the first three canonical variables appear to be meaningful. The first such variable has a strong correlation with the original variable of teenage birth rate (r=0.9327). The second has strong correlations with the other four original variables, that is with low birth rate (r=0.7949), preterm birth rate (r=0.9210), perinatal conditions rate (r=0.7135), and the maternal complications rate (r=0.6915). The third canonical variable displayed relative moderate correlations with low birth rate (r=0.5994), perinatal conditions rate (0.5236), and maternal complication (r=0.6679). All these correlations are positive. As suggested, the structure shows that the first canonical variable represents the original teenage birth rate, and the second and third canonical variables are actually representative of a combination of the other four original etiological variables.

Although there are 19 original ecological variables, only five canonical variables were generated, indicating that these five canonical variables have loaded most of the information provided by the 19 original ecological variables. In particular, the first canonical variable displays strong correlations with two original variables, namely percentage of the population receiving unemployment insurance (r=0.8502) and average individual income (r=-0.7206), and moderate correlations with another four variables, including percentage of the population speaking English (r=0.6105),

TABLE 16: Canonical Correlation Analysis
Etiological and Ecological Variables by Health Unit

1. Canonical Structure: Correlations between Original and Canonical Variables

Original Variable Group	Original Variable	Canonical Variable I	Canonical Variable II	Canonical Variable III	Canonical Variable IV	Canonical Variable V	Original Variable Descriptions
Etiological	LOWBRATE	0.0647	0.7949	0.5994	-0.0384	0.0570	Low Birth Weight Rate (1/1,000 live births)
	PRETRATE	-0.0931	0.9210	0.3718	0.0430	-0.0558	Preterm Birth Rate (1/1,000 live births)
	TEENRATE	0.9327	0.2956	-0.1078	0.1728	-0.0354	Teenage Birth Rate (1/1,000 live births)
	PERIRATE	0.1126	0.7135	0.5236	0.4329	0.1293	Perinatal Condition Rate (1/1,000 live births)
	MARTRATE	-0.0875	0.6915	0.6679	0.1379	-0.2216	Maternal Complications Rate (1/1,000 live births)
Ecological	ACRT91	0.3995	0.3245	0.4327	-0.1183	0.1555	Number of Acute Care Beds per 1,000 Population
	AVGINDIC	-0.7206	0.0917	-0.1290	0.1066	0.2590	Average Individual Income
	PCTICEMP	0.3433	-0.1458	-0.4581	0.0355	0.4842	Percent of Income from Employment
	CPCC1993	-0.5814	0.5362	0.4165	-0.2169	0.0892	Capacities for Child Care in 1993
	CRUDEDR	-0.2968	-0.0770	0.4462	-0.0333	-0.3598	Crude Death Rate
	ECRT91	-0.0832	0.3210	0.4931	0.2299	0.0030	Number of Extended Care Beds per 1,000 Population
	FLIFEEXP	-0.3701	-0.2292	0.3480	0.2239	-0.2893	Female Life Expectancy
	G12GRD92	-0.3334	-0.3010	0.2229	-0.1942	-0.0922	Percent of Grade 12 Graduation in 1992
	GFRT1991	-0.2186	0.4453	0.6160	0.0392	-0.0017	Number of GP FTEs per 1,000 Population in 1991
	MLIFEEXP	-0.5140	-0.2797	0.1154	0.1145	-0.2355	Male Life Expectancy
	PCENTNA	0.5376	0.4253	-0.5792	0.2018	0.0125	Percent of Native Indians to Population
	PCNTENGL	0.6105	-0.1163	-0.1063	0.5531	-0.0187	Percent of Population Speaking English
	PCNTTWOP	0.1584	-0.5851	0.0030	0.4503	0.0184	Percent of Family with Two Parents
	PCTKID1P	-0.2254	0.4841	0.2236	-0.2691	-0.3520	Percent of Children with One Parent Family
	PCTPOPUI	0.8502	0.1536	-0.1872	-0.0950	-0.0757	Percent of Population under Unemployment Insurance

2. Canonical Correlation Coefficients:

Pair of Canonical Variables	Canonical Correlation Coefficient	Squared Canonical Correlation	Approximate F Value	Degree of Freedom	Statistical Significance (Pr > F)	Proportion of Eigenvalue	Cumulative Proportion
1	0.9990	0.9981	2.4728	75	0.0709	0.8527	0.8527
2	0.9933	0.9866	1.3916	56	0.2971	0.1221	0.9748
3	0.9624	0.9262	0.7593	39	0.7428	0.0208	0.9956
4	0.8284	0.6862	0.3913	24	0.9641	0.0036	0.9992
5	0.5706	0.3256	0.2195	11	0.9830	0.0008	1.0000

capacities for child care in 1993 (r=-0.5814), percentage Native Indian population (r=0.5376), and male life expectancy (r=-0.5140). The second and third factors all had relative moderate correlations with the other original variables, while the fourth and fifth ones had relative weak correlations with them.

Canonical correlations between canonical variables based on etiological and ecological variables were fairly strong. As shown towards the bottom of Table 16, the correlation coefficient between the first etiological and ecological canonical variables was 0.9990, with a marginal statistical significance (p=0.0709). However, the proportion of the eigenvalue of the first pair canonical correlation is over 85 percent. Although the second to fourth pair canonical correlations are also fairly large (r between 0.8 or 0.9), they are not statistically significant, and their total contributions to the proportion of the eigenvalue is only about 12 percent. These figures indicate that the association between the original etiological and ecological variable sets is mainly explained by the first pair of canonical variables. As can be seen from the canonical structure of this pair of variables, it is clear that the teenage birth rate of the etiological variable set is strongly correlated with two economic variables of the ecological variable set, namely the percentage of the population receiving unemployment insurance, and the average individual income.

Canonical correlation analysis, therefore, demonstrates a tendency for interaction between etiological and ecological variables. This interaction may in turn determine the high infant mortality rates that are associated with unfavourable ecological settings in certain Health Units. In order to explore further such possible interrelationship between etiological and ecological variables, regression analyses

were performed between each of the etiological variables and all of the ecological variables. In such regressions, etiological variables are viewed as dependent variables while ecological variables are independent ones. This allows an investigation of how much, if any, regional differences in the incidence of etiological variables at the Health Unit scale can be attributed to the distinct ecological Health Unit characteristics.

In multiple regression analysis between infant mortality rates and etiological variables, three etiological factors appeared to have made significant contributions to mortality. As shown in Table 10, teenage birth rate, preterm birth rate and maternal complication rate are the three significant etiological variables. These three variables were, therefore, analysed against all ecological variables. Table 13 presents the results derived from stepwise selection regression analysis. In the model in which preterm birth rate was the independent variable, there were 9 ecological variables which made significant contributions to the regional variation in preterm birth rate. The R^2 value of this model which reached 0.8993 was clearly statistically significant ($p=0.0001$). However, the variable which made the greatest contribution to the preterm birth rate was the number of full-time-equivalent general practitioners per 1,000 population, a measure of the availability of the health care resource. This factor alone explained over 40 percent of the total variance, that is it accounted for almost half of the variance explained by all selected ecological variables. In summary, regional inequality in preterm birth rate at the Health Unit scale in British Columbia appears to have been largely determined by corresponding regional difference in the availability of primary care resources.

A total of 10 ecological variables appear to have made significant contributions to variances in teenage birth rate. These independent variables together contributed over 96 percent of the total variance of teenage birth rate by Health Units. However, the most important variable, that is the highest contributor to the variance of teenage birth rate, was percentage of the population receiving unemployment insurance. This variable, alone, explained over 70 percent total variance. For the maternal complications, the most important variable was once the again number of full-time-equivalent general practitioners per 1,000 population. Therefore, the Health Unit specific availability of the primary care resource and employment status were the two major ecological factors contributing to regional differences in the prevalence rates of three important etiological factors, namely teenage birth rate, maternal complications rate, and preterm birth rate. These etiological factors in turn were the main contributing factors to regional inequalities in infant, neonatal, and post-neonatal mortality rates. By comparing the R^2 values derived from regression analyses performed between etiological factors and infant mortality, and between ecological and etiological factors, as shown in Table 11, these interaction between etiological and ecological factors can be clearly demonstrated. To illustrate, the total contributions to the variance of infant mortality rates by ecological variables are between 46 and 80 percent (R^2 values between 0.46 and 0.80), while those to etiological variables ranged from 50 to 96 percent. Such differences seem to indicate that the relationships between etiological and ecological variables are closer, or more direct, than between mortality rates and ecological variables.

CHAPTER SEVEN

DISCUSSION AND CONCLUSIONS

This study set out to explore those etiological and ecological factors which are generally associated with and/or contribute to geographical variations in infant mortality found in British Columbia at the Health Unit scale. Comprehensive and multi-dimensional methodologies have been used for this purpose. These have included time trend analysis of infant mortality of the province as a whole, cross-sectional and time trend analysis of geographical inequalities of mortality at the Health Unit scale, the identification of the etiological factors and ecological determinants of such geographical inequalities, as well as the development of geographical profiles of Health Units which have distinct mortality rates. These analytical techniques show that infant health status in British Columbia as a whole has improved enormously during the past 30 years. These improvements are illustrated by the fact that provincial infant mortality rates, including neonatal and post-neonatal mortality rates, have been declining consistently over this time period. The past 30 years have seen, for example, an 81 percent reduction in infant mortality, an 84 percent decline in the neonatal mortality rate, and a 74 percent fall in the post-neonatal mortality rate.

However, regional inequalities in infant health status, at the Health Unit scale, do not appear to have followed a similar declining trend. For this reason, research into the underlying causal factors of such regional inequalities clearly was required. Such explorative analyses provided in this thesis identify differences in the teenage birth rate amongst British Columbia's 21 Health Units as the major

determinant of this regional disparity. Limitations and/or restrictions implicit in the data used in this study, however, prevent explicit determination of exact causes of regional inequality of infant mortality in the province. In addition, some of the statistical methods employed in this study have limitations that mean that results must be cautiously interpreted. It is important, therefore, to discuss these issues further and to clarify attempts that have been made to overcome these restrictions and their associated limitations.

7.1 Data Quality Restrictions and Limitations

As previously mentioned, most of the information on infant health status used in this study, including mortality and other etiological indicators, was collected from the annual reports of British Columbia's Vital Statistics Agency. A few other data sources were provided by the same agency as the results of special requests. To illustrate, information on ecological determinants and other related socio-economic characteristics were derived from the Health Planning Database of the British Columbia Ministry of Health and Ministry Responsible for Seniors. Some of the information from this database has been used previously in the Provincial Health Officer's Report of the Ministry, to describe socio-economic profiles of Health Regions (British Columbia Provincial Health Officer, 1996). A common feature of both data sources is that most of the information they contain was provided at the Local Health Area and/or Health Unit or Health Region levels. This implies that such information is population, or area-based data. A major advantage of such sources is that access to them is cheap. However, the use of such area-based data has

limited the depth and width of the analyses applied in study. This is because certain statistical methodologies, such as Pearson regression analysis, especially when a cause-effect relationship is intended to be derived from such an analysis, can not be applied to such population and area-based data.

As described in Chapter Five, one of the major goals of this dissertation was to identify those etiological factors which seem mainly responsible for higher infant mortality in some Health Units, and to investigate which area-specific ecological factors determine their prevalence. In order to achieve this goal, individual-specific, rather than population-based, data is required. Such data is infant specific. It allows identification of investigated causal factors, such as birth weight and gestational age, that are essential in establishing a case-control design which would permit a comparison between infants with such causal factors and infants without them. As a result, an odds ratio, which measures the degree of the contributions that a causal factor has made to an infant death, can be developed. Such information is always critical in determining whether prior exposure to a particular environmental factor is etiologically important (Lilienfeld & Stolley, 1994). Unfortunately, a lack of such individual-based information forced the etiological analyses to be performed only on area-based data. As described in Chapter Six, therefore, the incidence rates of the five etiological factors, namely low birth weight, preterm birth, teenage birth, births with perinatal conditions, and births with maternal complications that were developed, in this study, were not specific to either "exposed" or "non-exposed" individual infants, but rather to the total infant population for each Health Unit. In a similar manner, infant mortality rates were also based upon the total newborn

population of each Health Unit. As a result, the resultant etiological analyses were, therefore, performed between the incidences of etiological factors and mortality rates associated with each Health Unit. Such analyses, therefore, converted an individual-based etiological study into a population-based ecological analysis (Comstock, 1988). As a consequence, the results of this etiological study may not be completely reliable indicators of cause-and-effect relationships. This is because exposures, measured at the community/population level, may not represent individual exposures. Similarly, confounding factors associated with each individual, within each Health Unit, may differ significantly, so that a discovered positive or negative association between these two set rates, at the Health Unit level, might be statistically spurious (Comstock, 1988). As a result, cause-effect relationships between etiological factors and infant mortality cannot be fully established on the basis of such analytical findings. This is because that data restricts the methodology, which, in turn, determines the applied value of study (Pocock, Shafer, Powell, et al., 1987).

Despite such methodological limitations, relationships found from population-based research can still be valuable if two factors are plausibly biologically-related and a regression coefficient, not a pure correlation coefficient, is developed which is statistically significant (Comstock, 1988). To illustrate, in this study both simple and multiple regression analyses between infant mortality rates and each of the incidence rates of all five etiological factors have been performed. The established relationships are both biologically plausible (Kramer, 1987, United Nations, 1985; Wang et al., 1992), and consistent with other publications (Kierans & Hu, 1995;

Kierans, 1992; Hu, 1995, McCormick, 1985). It can be concluded, therefore, that Health Units with higher teenage birth rates tend to experience elevated infant mortality, especially post-neonatal mortality, although no cause-effect relationship between teenage birth rate and infant mortality rate can be established absolutely as yet.

A similar lack of direct linkage between infant mortality and etiological variables is another limitation resulting from the nature of the data used in this study. The available data is limited to the number of infant deaths, as a percentage of the total number of live births for each Health Unit or Local Health Area, and values of five etiological factors in the same Health Units. What was not available was the proportion of infant deaths within each of the sub-groups of infants exposed, or not exposed, to each of these five etiological factors. Mortality rates, therefore, can only be developed for the total infant population and not from infants that experience, or do not experience, particular etiological factor(s). The base mortality rate, which is normally developed from the infant population without taking such etiological factors into consideration, can not be obtained. As a result the relative contribution to infant mortality of each or group of etiological factors in each Health Unit cannot be established, since there is no base rate for comparison. The etiological factor (EF) of each of the five etiological causes cannot be calculated either. As a consequence, the strength of the association cannot be developed (Kramer, 1987; Kline & Stein, 1988).

As Bradford Hill (1964) pointed out, a causal inference cannot be made unless two studied events satisfy a series of conditions, which include strength of

association, consistency of the observed association, specificity of the association, temporal sequence of events, dose-response relationship, biological plausibility of the observed association, and experimental evidence (Lilienfeld & Strolley, 1994). In the current study, as a result of the limitations of the data, the strength of association between infant mortality and etiological factors could not be measured since the base mortality rate is unknown. Similarly, the specificity of the association cannot be defined because of a lack of one-to-one relationships between infant deaths and etiological factors; neither can dose-response relationships be established. As a consequence of these limitations, the author makes no attempt to draw any causal conclusions. Emphasis is placed on describing and comparing mortality trends in Health Units which have high incidences of the five etiological factors which tend to occur where there are increased infant mortality rates. These trend relationships appear biologically plausible and are consistent with the results of other published studies (Kierans & Hu, 1995; Kierans, 1992; Hu, 1995, McCormick, 1985). They also show a temporal sequence, since these etiological factors predate the infant death being examined.

Another goal of this study was to investigate the interactive relationship between etiological factors and ecological determinants. The author's original intent was to test his hypothesis that in a region with unfavourable ecological characteristics infants are exposed to a higher prevalence of negative etiological factors leading to an increased probability of infant death. If this is true, a close relationship must exist between these two sets, that is between ecological characteristics and etiological factors. The ideal data format to conduct this type of

analysis is, once again, a one-to-one matched record between ecological characteristics and etiological factors specific to each infant. Ideally, for such an analysis involving any individual infant, what is needed is data on its own health status (dead and alive), and also on its mother and family and on areal characteristics. If all these data are available, a complete infant profile could have been developed. This profile could then have been used for comparisons between etiological and ecological factors. The data available for this study, unfortunately, are population-based, which has restricted analyses to those that can only be performed between Health Units as a whole, rather than between sub-groups of infants with specific data records. To illustrate the problem, although an individual infant and its family might not necessarily be poor, if the Health Unit in which it was born has an average household income lower than the provincial mean, it would be assumed that the dying infant was from a poor household. From this point of view, the area-based ecological data is a reasonable surrogate measure of local ecological settings and has been used for this purpose elsewhere (British Columbia Provincial Health Officer Report, 1996; United Nations, 1985; Foster, 1993; Foster, 1992).

The author accepts that individual-specific matched data would have been more valuable to use in the preceding analyses if they had been available. However, ecological studies, such as the one conducted here, are also valuable if they are used appropriately. Indeed, there is an increasing trend towards the use of ecological analysis based on population data in medical geography and epidemiology, because they are cheap to access and rely on readily available and

routinely-collected information. This approach has been widely used as a first step analysis, when a hypothesis needs to be tested (Comstock, 1988). The results from such ecological analysis can usually identify which type of more specific and detailed studies are required to further test particular hypotheses. One of the goals of this research was the identification of a target region, a local region with unusually high infant mortality rates, where greater attention needs to be paid to potentially causal etiological and ecological characteristics. This goal has been achieved.

7.2 Methodological Considerations

As discussed in Section 7.1, the restrictions imposed by the data used in this study have determined the statistical analyses which can be appropriately applied to achieve the principal goal of this dissertation, an investigation of regional inequality in infant mortality rates at the Health Unit level. The statistical methods employed in this thesis include three types: descriptive/observational analyses of the temporal trend of the provincial infant mortality rate over time, regional inequalities of mortality rate over a time period relationship analyses between mortality statistics and etiological and ecological factors, and interactive analyses between etiological and ecological factors and their associations with regional inequalities in infant mortality.

7.2.1. Temporal Trend Analysis of British Columbia Infant Mortality Rates

To describe the temporal trend of the provincial infant mortality rate over the

last 30 years, the number of infant deaths that had occurred amongst the total number of live births each year was used to calculate an annual provincial infant mortality rate. These annual rates were then tabulated for each year from 1965 to 1996, allowing a comparison of changes. Several factors may confound the validity of such long term temporal trend comparisons. Definitions of infant health status may have undergone significant change during the decades involved, so that different definitions used in different time periods may reduce the comparability of various annual mortality rates. To illustrate, since 1915 when infant health statistics were first available in British Columbia, the definition of "stillbirth" has been modified three times. The very first definition of a stillbirth, used until June 30, 1962, was "the birth of a viable foetus after at least 28 weeks' pregnancy in which pulmonary respiration does not occur, whether death occurs before, during or after birth". This definition means that some stillbirths, prior to 1962, would now be reported as infant deaths. As a result, infant mortality rates for the years after 1962 may be artificially elevated (Cronin & Danderfer, 1994). The earliest year analysed in this study, however, was 1965. As a result, this definitional change has no impact on the results of analyses. Fortunately, the final two re-definitions of stillbirths had no direct impact on infant death statistics. It should be pointed out that the impact of the first change in stillbirth definition would be to increase the recorded infant mortality rates in later years. As a result, temporal analysis would tend to under-estimate any annual reduction in infant mortality, not over-estimate it.

A second factor, which may influence temporal analysis, is the significant increase in live births in recent years. This growth in the number of births may, to

some degree, render older and more recent annual infant mortality rates less comparable. To illustrate, the total number of live births in 1996 (45,883) was an increase of about 37 percent over that of 1965 (33,669). Since it is based on a smaller number of births, the infant mortality rate for 1960s might be less stable than rates for the 1990s. If this is true, the annual reduction in infant mortality rates, developed from temporal trend analysis, may be either an over or an under estimate. Nevertheless, since the numbers are very large for the recent time period, this seems very unlikely to be an issue.

Thirdly, the infant mortality rate developed for use in this study is an overall infant death rate, rather than one illustrating a specific mortality related factor's impact. Temporal trend analysis of the infant mortality rate gives only an overview of how the health status of the provincial infant population has improved during the past 30 years. Causes of infant death are so complicated because so many factors can directly or indirectly impact on the development of a fetus, leading to the birth of either a healthy or unhealthy infant (Kramer, 1988). Ideally, because this study focuses on regional inequalities of infant mortality rate at the Heaith Unit scale from both an etiological and an ecological perspective, those factors which may confound this comparison should be controlled, or adjusted, before temporal comparison is performed. For instance, maternal age and the number of single or multiple births, therefore, can influence infant mortality rate. It would be more meaningful, therefore, if such a temporal comparison was undertaken using maternal-age-specific mortality rates rather than an overall rate. In addition, women are now tending to give birth later in life, so mothers tend to be older. However, it is known that older mothers'

infants tend to experience higher mortality rates (Cronin & Danderfer, 1994). Since the general trend of the infant mortality rate over the past 30 years has been one of decline, such adjustment for maternal age would probably steepen this declining trend, rather than reduce it. The impact of maternal age at birth, therefore, may not be critical to the conclusions drawn from the descriptive temporal trend analysis of the British Columbian infant mortality rate, over the last three decades.

7.2.2. Description of Geographical Patterns of Infant Mortality Rates

In order to describe regional inequality in infant mortality within British Columbia, the geographical distribution pattern of the infant mortality rate at the Health Unit level should first be described. Trends in inequality can then be investigated. To achieve this objective the expected number of infant deaths in each of the 21 Health Units in a given year was derived based on the annual mortality rate of the province as a whole. Then, a ratio between the actual and expected infant deaths was calculated for each Health Unit. This ratio indicates how close the number of infant deaths occurring in any Health Unit were to the expected number, that is to the provincial norm. When applying this method, however, annual infant deaths (especially when subdivided into neonatal, and post-neonatal mortalities) in certain Health Units were too limited to allow meaningful comparisons to be made with the relatively small total number of infants delivered in that year, in these Health Units. One common method used widely to overcome this problem is to calculate the accumulated mortality ratio, either by aggregating area/population size, or by accumulating several years of data before analysis (Foster & Edgell, 1992). In this

study, in order to ensure enough infant deaths for regional comparison, data from the ten years, 1987 to 1996, were aggregated to develop the total observed, over expected mortality ratio, for each Health Unit. In other similar studies the time periods used in mapping health status by geographical regions have used aggregated data for a single year (Danderfer, 1997; 1998); 10 and 11 years (Pickle et al., 1987); or even 28 years (Band et al., 1989). The length of time period used for data aggregation, therefore, varies in studies with different objectives of distinct specific events, such as death or morbidity. It should be noted that short time periods may result in a limited number of mortality observations, so that reliable statistical measures are difficult to develop. Long time periods, however, also are fraught with problems since they may obscure real trends and are complicated by improvements in diagnoses and in medical technologies over time (Foster & Edgell, 1992).

The nature of the data sources causes another weakness in describing the geographical patterns of infant mortality at the Health Unit scale. According to the annual report of the British Columbia Vital Statistics Agency, live births and infant deaths which occur outside the province are historically not included in these reports and, therefore, neither are they analysed in this study (Danderfer, 1997; 1998). It is known, however, inhabitants of British Columbia living close to the boundary with Alberta often go to that province to seek medical care. This is allowed by the BC Medical Services Plan. Such an outflow of services utilization may result in a significant number of live births being delivered outside British Columbia, in addition to a significant number of infant deaths occurring outside the province.

When comparing the provincial infant mortality rate over an extended time period, such out-of-BC infant deaths and live births may not be statistically significant because it is reasonable to assume that the proportion of BC residents seeking medical care out of the province remains relatively consistent over time. When comparing infant mortality rates amongst Health Units, however, this outflow of patients may cause significant regional rate inconsistencies. As a result Health Units bordering Alberta, such as East Kootenay, may present significant lower infant mortality rates. This problem can be solved by collecting out-of-province live birth and infant death data, if available. In this study, however, no attempt has been made to address this problem. It has been assumed that the infant death rate amongst those live births delivered outside of BC for any Health Unit is comparable to that of infants delivered inside of BC. If this assumption is accepted, the individual Health Unit mortality rates would not be seriously impacted by such a patient outflow.

7.2.3. Measuring Geographical Inequality of Infant Mortality Rates

This thesis has examined overall geographical inequality in infant mortality, and has included a detailed analysis of specific differences in relation to the ecological profiles of Health Units and to the etiological factors of the infant population as a whole. Comparisons between geographical areas can prove extremely useful when it comes to identifying more disadvantaged areas where government intervention programmes may be needed (Lardelli 1993). However, such spatial methodologies do not provide the kind of overall appraisal which would

allow conclusions to be drawn about the extent to which a particular Health Unit was progressing towards the goal of equal-health for infants (Vazquez-Vizoso et. al, 1993). In another words, a cross-sectional description of regional differences in infant mortality provides a static picture of the problem under the study. The primary goal, however, must be to investigate changes in regional inequality over time, especially if they are moving in the same direction as the change in the provincial infant mortality rate as a whole. To achieve this goal a single index, which is able to describe geographical inequality in infant mortality in the province as a whole, needs to be developed. This index, in turn, should be used to portray the temporal trend of geographical inequality in infant mortality amongst the 21 Health Units, in comparison to the trend of the provincial infant mortality rate.

Statistical concepts that measure the dispersion of a variable come very close to modelling this social concept of inequality, suggesting a single measurement capable of quantifying inequality. For this reason it is usual to use a dispersion-related measure when seeking to arrive at an overall quantification of geographical inequality (Illsley & Svensson, 1990). However, not every such measure is equally suited for this purpose and it is possible that the application of all of these measures will not produce the same conclusions. To illustrate, dispersion measures can be divided into two groups, namely those that quantify dispersion in absolute terms and those which do so in relative terms (Vázquez-Vizoso et al, 1993). Among the former, range, standard deviation and variance are commonly relied upon; the latter category includes the ratio between maximum and minimum distribution values, the ratio between top and bottom deciles, and the

coefficient of variation. Range is probably the measurement of dispersion most frequently used to quantify health differences between population groups (Centres for Disease Control, 1991). Its chief drawback lies in the fact that it contains information only on the highest and lowest values of any distribution. While standard deviation and variance do take into account all the values of any distribution, values depend not only on the degree of dispersion but also on the magnitude of the variable so rendering comparison impossible between distributions having different magnitudes or, what amounts to the same thing, different means. Finally, measurements of absolute dispersion have units that stem from the variable on the basis of which they have been calculated which rules out any comparison of variability in the case of distributions having different units (Wagstaff, Paci, van Doorslaer, 1991). The weighted coefficient of variation (WCV), however, used in this study has a unique advantage. It weighs the coefficient of variation of Health Unit infant mortality rate by live births born in each Health Unit. As a result, every infant death from any Health Unit weighs the same, so that the mean of the Health Unit rates, the average weighted rate, coincides with the mean provincial mortality rate. Therefore, the contribution of unstable rates, from Health Units having the lowest number of live births to overall variability is minimized (Wing, Casper, & Davis et al., 1990). In a comparative review of the various techniques available for assessing health inequalities across socioeconomic groups Wagstaff et al. (1991) listed three characteristics of a good measure of inequality. These characteristics are all present in the weighted coefficient of variation as an indicator of geographical inequalities in health: (1) it reflects the geographical dimension of inequalities in health; (2) it

takes into account the data of all geographic areas; and (3) it is sensitive to time changes in the distribution of the population at risk across the areas. Because of these three advantage, the weighted coefficient of variation, it has been employed elsewhere to analyse time trends in the differences recorded for distinct population groupings with respect to a diversity of social and economic variables, and cardiovascular disease mortality (Wing, Casper, & Davis et al., 1990; Vázquez-Vizoso et al, 1993).

7.2.4. Relationship Analysis

As discussed previously, the limitations and restrictions of the data used in this study have ensured that the statistical methodologies applied were, to a large extent, the population and/or area based analyses typically used in observational epidemiology (Beaglehole, Bonita, & Kjellstrom, 1993). In evaluating the relationship between infant mortality and etiological causal factors, correlation and regression analyses have been performed between the rates of the two sets of variables across Health Units. This type is recognized as ecological correlation/regression analyses (Clark & Hosking, 1985). Generally, there are three types of erroneous inferences that can occur if a researcher attempts to generalize from one level of investigation to another. The first is individualistic fallacy, which is the attempt to impute macro-level (aggregate) relationships from micro-level (individual) relationships. It is the classic aggregation problem first examined by economists. The second one is cross-level fallacies, which can occur when one makes inferences from one sub-population to another at the same level of analysis. The

last one is the ecological fallacy, which is the opposite to the individualistic fallacy and involves making inferences from higher to lower levels of analysis. Although the ecological fallacy has been widely discussed and publicized, it is a common error in studies involving causal inference (Clark & Hosking, 1985). However, ecological relationship analysis is appropriate when the variables are functions of some common underlying causal structure inherent not in the individuals themselves but in the properties of the areas. To illustrate, social conflict, cultural conflict, and other sociological variables may be the underlying structures for some individual level analyses. Ecological analysis is also accepted when one aggregate variable is related to another and an individual correlation would be impossible (as, for example, in the correlation between the number of physicians per capita and the infant death rate) (Clark & Hosking, 1985). In this particular study, the author did not attempt to draw causal inferences between any of the etiological and the ecological factors and infant mortality at the Health Unit scale. Rather he attempted to describe structural characteristics of each Health Unit in association with different levels of infant mortality rates. Although there is some implied inference when making such analysis, no explicit cause-effect inference is drawn from his study.

Another methodological issue which has been addressed in the last decade in geographical studies is spatial autocorrelation. Spatial autocorrelation refers to the correlation of the residuals from a regression equation. For spatial data of infant mortality by Health Units, for instance, the mortality rate in one Health Unit is likely to be similar to that in adjacent Health Units. This autocorrelation usually violates the assumption of statistically independent error terms in regression analysis. The

effect of this violation is that although the estimates of β_0 and β_1 (the intercept and regression coefficient in a regression equation) from ordinary least squares are unbiased, the confidence intervals around these estimates are very large. As a result the estimators are inefficient and *t* and *F* tests are invalid (Clark & Hosking, 1985). Compared to time-series autocorrelation, spatial autocorrelation is more complicated because it involves multi-direction autocorrelation. In addition there is no effective statistical method available to solve this problem, and the spatial autocorrelation needs to be considered only if statistical inference is made based on the estimated β_0 and β_1 (Clark & Hosking, 1985). In this study, however, the author did not try to measure quantitatively how much the geographical inequality of infant mortality rate at the Health Unit scale would have been reduced, if both etiological and ecological factors could be controlled to a certain extent. Rather, the author tried to qualitatively scale Health Units into certain groups which appear to correspond to the Health Unit ranking, based on the infant mortality rate. Therefore, spatial autocorrelation, if it has any impact, should not significantly influence the validity of the relationships analysed in this study.

7.3 Directions in Policy Making

Despite all the weakness in the methodologies used in this study, two major variables have been discovered that provide a biologically plausible explanation for regional inequalities in infant mortality rates at the Health Unit scale. These variables are teenage birth rate and family income level. To confirm further whether or not these two variables really play causal roles in regional variations in infant

mortality, detailed information on maternal age and family income level specifically associated with individual infant health status must be collected. Once such information is available, a case-control research design can be established to test the hypothesis further. Such a research design would be able to determine the extent to which maternal age and family income level contribute to infant mortality. If the hypothesis, suggested by this research, proves correct, it would have major policy implications for provincial Ministry of Health. Since this study strongly suggests that elevated teenage births are responsible for much of the regional difference in infant mortality, reducing the teenage birth rate might be a goal for local health authorities in all Health Units.

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