VARIABILITY IN HUMAN TOOTH FORMATION: A COMPARISON OF FOUR GROUPS OF CLOSE BIOLOGICAL AFFINITY

By

CLARE MCVEIGH, B.A., M.Sc.

A Thesis

Submitted to the School of Graduate Studies

In Partial Fulfillment of the Requirements

For the Degree

Doctor of Philosophy

McMaster University

VARIABILITY IN HUMAN TOOTH FORMATION

DOCTOR OF PHILOSOPHY (1999) (Anthropology)	McMaster University Hamilton, Ontario

TITLE:	Variability in Human Tooth Formation: A Comparison of Four Groups of Close Biological Affinity
AUTHOR:	Clare McVeigh, B.A. hons., (University of Sheffield), M.Sc., (Universities of Sheffield and Bradford).
SUPERVISOR:	Dr. Shelley R. Saunders
NUMBER OF PAGES:	xiv, 241

Abstract

A considerable amount of research has been undertaken in the area of variability in the timing of human tooth formation. While many researchers acknowledge environmental influences have an impact on dental *eruption*, differences in the timing of human tooth formation are usually attributed to genetic factors. This thesis, however, investigates the impact of environmental and behavioural influences on the timing and duration of tooth formation by comparing four groups of close biological affinity.

Dental development of the Spitalfields sample (18th –19th century London) was found to be retarded in relation to the Poundbury (Romano British), Belleville (19th century Canadian) and Burlington (modern Canadian) samples. Furthermore, tooth size was significantly smaller for the Spitalfields sample. It is argued that these two factors are indicative of environmental stress in the form of poor nutrition and high pathogen load.

Acknowledgements

Having finally completed all the requirements for my Ph.D., I would like to take this opportunity to express my gratitude to the many individuals who have helped me to finally reach this point. First and foremost, I would like to thank my advisor, Dr. Shelley Saunders, not only for her guidance and support, but also for her patience and understanding over the last four years. To my other committee members, Dr. Aubrey Cannon and Dr. John Mayhall, I extend my sincere gratitude for their prompt and thoughtful advice throughout my entire program.

Many individuals at the anthropology department at McMaster University helped make the process of writing my thesis a positive experience. I would like to thank all the faculty members who helped create a warm and friendly working environment. This is also true of Janis Weir and Rosita Jordan and "Cookie" Brymer, and I would also like to thank them for dealing with my many traumas with efficiency and humour.

My research was carried out at the Palaeontology Department at the British Natural History Museum, and on material from the Burlington Growth Study at the Faculty of Dentistry, University of Toronto. I would therefore like to thank the many individuals at both institutions for their kindness to me while I was undertaking my fieldwork. My research was made possible by funding provided by the School of Graduate Studies at McMaster University and a Commonwealth Scholarship.

This section would not be complete without thanking my fellow graduate students who supported me over the last four years. They have not only been good colleagues, but also helped me settle in to a foreign county by their good natured tolerance.

Finally, I extend my heartfelt thanks to my husband Greig Parker. Without his uncompromising and untiring encouragement I would never have completed this thesis.

Table of Contents

Chapter 6: Conclusion.

List of Figures

List of Tables

Chapter 1
Introduction

1.1: The Research Question

This thesis addresses the question of variability in human tooth formation. While a considerable amount of research has been undertaken in this area, existing studies focus on either differences between groups of distant biological affinity (for example, Tompkins, 1996 and Owsley and Jantz, 1983), or on within-population variability in homogenous groups of modern, North American children of European origin (for example, Smith *et al.*, 1981; Garn, *et al.* 1965; and Demirjian *et al.*, 1973). A study by Harris and McKee (1990) however, found that differences in the timing of dental development were greater between two "white" samples than differences between "white" and black" populations. This highlights the need for further investigation into groups of close biological affinity, but from environmentally and culturally different backgrounds.

This thesis, therefore, tests the hypothesis that significant differences in the duration and pattern of tooth formation can be found between groups of close biological affinity. The value of such research is considerable, since it will highlight the impact of environmental and behavioural influences on dental development.

While many researchers acknowledge environmental influences have an impact on dental *eruption*, differences in the timing of human tooth formation are usually attributed to genetic factors (Hillson, 1996).

This thesis compares the duration and timing of human tooth formation of four samples of groups of close biological affinity; two are British, while two are Canadians of British origin. Furthermore, since one of the samples comprises modern children while the other three are derived from archaeological contexts, the life spans of the individuals in this study occur during a range of over 700 years. These factors have provided the opportunity to compare tooth formation in Europeans from diverse cultural and environmental conditions.

1.2: The Research Question in Context

1.2.1: Palaeoanthropology

The study of dental development plays a central role in the determination of phylogenetic relationships, life history, stature and behaviour of early hominids. Recent research has centred around an interest in the onset of an extended growth period and its implications (Smith and Tompkins, 1985; Lampl et. al. 1993). Such studies require accurate and reliable data on patterns of dental development in fossil hominids, apes and humans. At present, the data used for modern humans are based on modern North Americans of European origin. A more geographically and temporally diverse human sample would make a significant contribution to an assessment of the degree of variability and an understanding of the factors which influence it. Such a study may also shed light upon potential variables which may have influenced dental development amongst extinct hominid species.

1.2.2: Heredity and Environment

Dental development is subject to both environmental and genetic factors. The rate of human tooth formation, like other biological growth systems, is subject to a number of traits which show continuous variation. Continuous variation can be caused by the simultaneous effects of many genes (Niswander, 1963). A wide range of environmental influences are then superimposed on the genetic effects. By examining populations from different time periods, geographical locations, and cultural backgrounds, but with close biological affinity, the roles played by behaviour and environment are explored. The establishment of "stressed" and "affluent" patterns of human tooth formation will have practical applications as indicators of environmental stress, and would thus be of interest to biologists, archaeologists and environmentalists.

1.2.3: The Age Estimation of Archaeological Material

The observation of dental development, in the form of calcification stages, is the preferred method for the ageing of sub-adult skeletons (Smith 1991). Since such techniques can be used throughout the entire age spectrum of sub-adult remains, they are considered superior to other methods. In addition, tooth formation is believed to be less subject to genetic and environmental influences, and therefore the most accurate indicator of chronological age (Saunders, 1992; Smith, 1991; Moorees *et al.*, 1963; Ubelaker, 1989). As tooth formation standards are used as a basis for the calibration of other techniques, their accuracy is essential.

Since these standards are usually based on data from modern, European children and North American children of European origin, some researchers have questioned the validity of their use in archaeological contexts. Owsley and Jantz (1983) highlighted the problems of using modern standards to estimate the age of at death of material from an Arikara Indian population dating from A.D.1600-1835. A more recent study by Tompkins (1996) compared patterns of dental development in modern French Canadian, modern "Black" South African, and archaeological Native American samples. Again, significant differences in the patterns of development were discovered.

Both of these studies have used Native American samples as examples of archaeological groups. In the latter study, data from a number of archaeological sites were "pooled" and treated as a single population. A similar problem exists with the "black" sample, which is also assumed to be genetically, culturally and environmentally homogenous. This approach serves the advantage of increasing sample size, but also causes problems, as different biases are also pooled. For example, different cultural groups frequently eat different foods, live in different conditions, and are subject to different climates. All these variables may influence the rate of tooth formation. By pooling data from groups which are subject to such a variety of influences, the possibility of determining the causes of variation is lost.

In this thesis, the three archaeological and one modern sample are of close biological affinity, but from a broad temporal and environmental range. The comparisons of patterns of tooth formation in the samples will test the assumption that there is a European "standard" of dental development, which can be used to accurately determine age at death of subadult skeletons from different environments and cultures.

1.3 Organization of the Thesis

Chapter 2 reviews research which has been undertaken in the field of variability of human tooth formation and its controls. This includes an examination of developmental models, theories of evolutionary change, and variability in modern populations.

Chapter 3 contains background information about the four samples used in this study. This involves a comparison of the historical contexts and sample composition of each group in order to facilitate discussion regarding representativeness. This is necessary in order to validate comparisons of four samples from such diverse sources.

Chapter 4 presents the methodology, results and discussion of the investigation into variability in patterns of human tooth formation, while chapter 5 presents the same information for the investigation of correlates of dental development to tooth size and mandibular size.

The concluding chapter examines the contributions of these findings to different areas of study, highlighting biological and anthropological implications. In addition, suggestions for future research are outlined.

Chapter 2
The Development of the Human Dentition and its Controls

2.1: Introduction

The development of the human dentition and its controls are of interest to researchers in the fields of human biology and genetics. As teeth survive well over long periods of time, changes in the human dentition can be studied to gain knowledge about human evolution and the changing lifestyles of past populations, thus making the study of human dental development relevant to hominid palaeontologists, palaeoanthropologists, archaeologists and historians.

Although there is consensus in that most researchers agree that dental development is under genetic control upon which environmental influences are overlaid, there is disagreement regarding the actual mechanisms of control, the magnitude of environmental influences and the causes of evolutionary change. There is also disagreement regarding the degree of variation within and between human populations.

This chapter examines research in three areas: models of development, theories of evolution, and variability in modern populations. Rather than presenting an exhaustive review of the literature on these subjects, the aim is to critically evaluate some of the work which has been undertaken using representative studies as examples. The problems involved in each area of research are discussed, and possible solutions offered.

2.2: Developmental Models

2.2.1: Introduction

The development of the human dentition has been well documented (see Sperber, 1981; Berkovitz and Moxham, 1983; Hillson, 1986). This section, therefore, presents a brief description of four conceptual models which have been formulated to explain how and why the human dentition develops in a specific sequence. The validity of terming these conceptual models "theories" can be questioned. The difficulties involved in testing these models are discussed, citing research that has been undertaken in this area.

2.2.2: The Odontostichi Model

In 1919, Bolk (in Osborn, 1981) envisaged the human dentition as being derived from the first two rows of reptilian teeth. Polyphyodont animals such as reptiles replace their teeth in wavelike patterns, in that alternate teeth are replaced in waves that sweep forwards from the back of the jaw. This process has an advantage in that resorbing and erupting teeth are flanked on either side by mature, firmly established teeth thus preventing substantial gaps from arising in the dentition which would be a possibility if tooth replacement occurred on a random basis.

Bolk postulated that each row of alternate teeth could be considered as a biological unit, or odontostichos, and that reptiles possessed two types, one contributing to the replacement of odd numbered teeth, the other type to the replacement of even numbered teeth. The dentition of diphyodont animals such as mammals has evolved in such a way that only the first two odontostichi remain; one odd and one even. The teeth in these odontostichi have been pushed together so that the first contributes to the development of the deciduous dentition and permanent molars, and the second to the replacement teeth.

2.2.3: The Zahnreihe Model

This model was first proposed by Edmund in 1960 (in J.W.Osborn 1973). Unlike Odontostichi, Zahnreihe are units of consecutive, rather than alternate teeth. Edmund envisaged regular impulses originating in the front of the jaw towards the back, with teeth developing when the impulse reaches a given tooth position. After all tooth positions are filled, replacements will appear to be occurring from the back of the jaw.

In common with Bolk's Odontostichi model, the Zahnreihe model proposes that diphyodont animals possess only the first two Zahnreihe; one contributing to the deciduous teeth and permanent molars, the other to the replacement teeth.

2.2.4: The Clone Model

This model defines two biological units: the teeth and the dentition (J. W. Osborn 1981). Two homogeneous populations of mesenchymal cells contribute to the development of each unit: a dental clone which produces the teeth and the structures which support them, and the basal clone, which produces the jaws.

The dental lamina is initiated by the dental clone, with new teeth forming in available spaces. As each tooth is surrounded by an area which inhibits tooth growth, a clone must escape this area before it can initiate the development of a new tooth. J. W. Osborn proposed that there are three clones in each quadrant of the mammalian jaw, producing the incisors, canines and molars. This model accounts for heterodontic teeth, as gradients in size and shape are related to the direction of the growth of the clone. The ability of the clone to produce large complex teeth declines as the clone grows. For example, the first permanent molar is larger and possesses a more complex morphology than the second, which is in turn more complex than the third (J. W. Osborn 1981).

2.2.5: The Morphogenic Field Model

In this model all tooth primordia are identical, until their receptors become perfused with a given "morphogen" which dictates size and shape. Mammals have three different types of field substance, each giving rise to a different morphogenic field. Teeth at the centre of a morphogenic field will receive a more concentrated form of the morphogen, resulting in a large tooth of complex morphology. Teeth at the edge of a field will receive less of their own morphogen and also some morphogen from the neighbouring field. This will result in a reduction in size and decreased stability (J. W. Osborn 1981).

2.2.6: Discussion.

The Odontostichi and Zahnreihe models provide possible explanations for the sequence of replacement of reptilian teeth. Both are flawed in respect to diphyodontic dentition as neither explain the seemingly random but species specific sequence of mammalian tooth replacement (Hillson 1986).

J. W. Osborn (1970, 1973) has suggested that Zahnreihe need not have evolved to produce the mammalian form of tooth replacement, and that changes in sequence can be explained by changes in tooth morphology and the evolution of the heterodontic dentition. Put simply, the Zahnreihe model requires that all teeth take the same length of time to develop, in addition to remaining in the jaw for the same amount of time. This is the case in most homodonts. As this is not true of the more varied and complex dentitions of mammals, wave replacement of alternate teeth is no longer recognizable.

Neither the Odontostichi or Zahnreihe models attempt to provide an explanation for the development of the morphologically different classes of teeth in heterodontic dentitions. Both the Field and Clone models are used to attempt to provide such an explanation. It is difficult however to envisage ways in which these ideas could be tested. This limits their value as potential theories of dental development.

For example, work on fluctuating asymmetry (which will be discussed in more detail in section 4) appears to support the field model, as more distal teeth in fields appear to exhibit a greater degree of asymmetry between antimeres. This has been cited as a field effect by many researchers, as teeth furthest away from the centre of the field seem to be more developmentally unstable. It is not possible however to prove that this asymmetry is not a consequence of diminished "competence", as teeth are on the border of a clone.

Field theory suggests all tooth primordia are identical until perfused by a morphogen. Consequently, studies of embryonic dental development are of relevance. Some researchers have argued that extrinsic factors such as morphogens, chemical signals and impulses are not required for the development of different classes of teeth (J. W. Osborn, 1978; Lumsden, 1979; Lumsden, 1981; Schwartz and Langdon, 1991), while others have identified not three but two separate, functional segments in the jaws of modern humans.

Keiser and Groenenveld (1987) maintain that the anterior teeth (the incisors and canines) and the posterior teeth (the pre-molars and molars) are independent units, and that size of teeth in the two segments is negatively correlated. In addition, metric data derived from the teeth of fossil hominids also show two units of size co-variation, which are possibly responding to different selective pressures (Trinkaus, 1983)

deCastro and Nicolas (1987) argue that field theory does not account for all the variability found in the human dentition. They investigated the relationship between different teeth in the same class by measuring the third and fourth premolars of fossil hominids and modern humans. They discovered that the ratio between these teeth is a sensitive indicator of the *size* relationship between the anterior and posterior dentitions, as the third pre-molar appears to have been influenced by the reduction of the incisors and canines.

Chavez-Lomeli and colleagues (1996) investigated early dental development by looking at the role of innervation and maturity of bony structures. They maintain that, as nerve tissue is necessary for tooth formation from the bell stage onwards, a close developmental relationship exists between the teeth, nerves, and surrounding bone. By examining the mandibular canals of fetal skeletons, they were able to identify three separate origins or openings, each of which appears to be directed towards the three different classes of teeth classified according to field theory. They argue that these three openings carry three compounds of the mandibular nerve bundle to the three developmental fields and play a vital role in tooth growth. This concurs with the findings of Jakobsen and colleagues (1991) which show that the unilateral absence of the mandibular canal is associated with agenesis of teeth in the corresponding area.

In contrast, Lumsden's research into mice molars (1979) illustrates the way in which fully formed teeth within a morphological class can develop in a non-vascular, non-innervated medium. Three mouse molars were developed from a mass of cells from the region of the first molar in such an environment. These teeth were morphologically correct and developed in the correct sequence. This would appear to negate the validity of the field model which proposes that all primordial teeth are identical until perfused with the required morphogen. It could be argued however that such an infusion occurs at the initiation of the first tooth in each class (J. W. Osborn 1981).

Further research involving the dissection of mouse embryos led Lumsden (1981) to postulate that the induction of tooth development is aided in some way by pioneer nerves innervating the area where cells will eventually form the first molar. Can uniformitarian principles be applied to this idea to include the human dentition?

An examination of the adult human dentition would appear to support this notion of a role played by innervation, as the three morphological classes of teeth in the maxilla receive their nerve supply from three separate branches of the superior alveolar nerve, with the innervation at the molar/premolar border being variable between individuals. Schwartz and Langdon, (1991) however, argue that this anatomical evidence is misleading, and that ontogenetic research is required in order to examine innervation at the time of development.

Using 35 human fetal specimens, they found that the area of tooth germs is surrounded by, rather than penetrated by nerve branches. (Teeth are not truly innervated until the stage of root formation). In addition they discovered a variable configuration of innervation to the dentition, which they later simplified to form four main classes. The nerve supply as seen in the adult jaw does not appear to occur until much later in development, and is believed to be governed by proximity rather than by a blueprint that dictates a given tooth be innervated by a given nerve.

Schwartz and Langdon (1991) argue that if innervation does play a role in the induction of tooth formation, it is only in the case of stem progenitors, thus only involving the initial tooth in each class. It is argued that the following teeth are mitotic extensions of stem cells.

These studies illustrate how attempts can be made to test the validity of developmental models through rigorous research. The results of such studies however, are inconclusive. Are studies of the dentition of rodents, such as those undertaken by Lumsden (1979, 1981), relevant to the growth and development of humans? Does the evidence supplied by Schwartz and Langdon invalidate the morphogenic field model?

It must be remembered that studies of dental development are limited, in that only dead, non developing tissues are examined; it is not possible to observe the substructure without sacrificing the tooth (Dahlberg, 1971). The only longitudinal studies which are possible are those of formation; the study by Schwartz and Langdon can be considered cross-sectional, as a set of adult dissections are compared to a set of fetal dissections. This makes it impossible to investigate the relationships between different fetal configurations of innervation, with variability in the adult dentition.

2.3: Evolutionary Change

2.3.1: Introduction

There is a large body of research documenting the reduction of the human dentition. A summary of the literature dealing with evidence of reduction in the size, number and complexity of teeth can be found in a paper by Anderson and colleagues (1975). More recently, there has been an interest in the evolution of the timing of dental development (see Smith, 1986; Smith, 1995; Lampl *et al.*, 1993; and Mann *et al.* 1990). This section presents a critical evaluation of both areas of research.

2.3.2: Natural Selection

Before discussing changes in size and morphology, it is necessary to discuss the actual process of natural selection. R. H. Osborne defines natural selection as " a synonym for differential reproduction of different phenotypes". To constitute a selective advantage, a factor must relate to reproductive performance. Osborne presents the following argument regarding selective pressures and the evolution of tooth morphology.

> *"...it is uniformly found that attrition of both the deciduous and permanent dentition occurs early in tooth life and in chronological age. The fissures and cusp pattern of the molars are obliterated and the cutting edges of incisors removed before reproductive life, with nothing to suggest a reduction in either survival or reproductive performance of the individuals so affected." (R.H. Osborne, 1967:946)*

This argument is flawed on several counts. Firstly, as far as early populations are concerned, chronological age is unknown. Estimates are frequently based on formation standards which are derived from modern populations. Such standards may not be applicable to past populations (Owsley and Jantz, 1983; Tompkins, 1996), moreover establishing reproductive age is even more problematic. As we can ascertain neither variable with certainty, the premise upon which this argument is based is unsound. In addition, this argument could be used to support the counter argument; the fact that fissures and cusps disappear at an early age means that heavy attrition is present, and that the functional capacity of the dentition could cease before the end of the individual's reproductive life, thus diminishing reproductive capacity. R.H. Osborne (1967) ephasizeds the role of genetic drift due to migration. He argues that colonization rarely results in a population representative of its parent population as the colonizers are not normally a random sample. Instead, a group will usually comprise closely related family members, so the founder effect will play an important role.

2.3.3: The Selective Compromise Effect.

It has been argued that there needs to be a balance between tooth size and environmental demand (Calcagno and Gibson, 1988). Teeth that are small may be worn to the pulp relatively early in life, causing loss of function and risk of infection, which in some cases could be life threatening. Calcagno and Gibson state that the intuitive appeal of this scenario is responsible for its interest to advocates of The Probable Mutation Effect and Universal Pleiotrophy, and that the notion of selective pressure against large teeth have been ignored, as it is counter intuitive. (These theories will be discussed below).

Paradoxically, individuals with large teeth can sometimes have less functional area in the jaw than individuals with small teeth. Large teeth, for example, tend to have more pits and fissures, and are therefore prone to caries and abscesses which can result in tooth loss. In addition, infection can result in life-threatening conditions such as septicaemia, gangrene and osteomyelitis (Hillson, 1986). When both sides of this argument are considered it becomes apparent that both large and small teeth are prone to caries and ante-mortem tooth loss; however this does not help clarify the nature of selective pressures on teeth and it is more useful to examine tooth size in relation to jaw size. This will be discussed in detail in section 3.3.9.

2.3.4: The Somatic Budget Effect

Some researchers (for example Smith, 1982) maintain that the energy used in the development of unnecessary structures is wasted. In the context of dental development, large teeth are inefficient, and selective forces would favour individuals with smaller teeth, as they are more 'energy efficient.' Other researchers argue that dental reduction only saves a marginal amount of energy and materials, and believe it unlikely that this would become a factor in selection (Christensen, 1998).

3.3.5: Universal Pleiotrophy

Wright (1964) looked at structural reductions in terms of pleiotropy, a condition in which a singe mutation simultaneously affects several characters. Put simply, the direction of change in gene frequency depends on the most important of multiple components; if one character is no longer under selective pressure, then changes in gene frequency within a population will depend on the characters which are under selective pressure.

2.3.6: The Probable Mutation Effect

Brace (1963; Brace and Mahler, 1971) also examined the role of mutations when selective pressure on a given trait is relaxed. They argued that if a structure is no longer in use, the selective pressures which directed its morphology and size are relaxed. Mutations which would have previously been de-selected can now accumulate in the population. Eventually, the probable mutation effect will result in a decrease in structure's size and complexity (Brace, 1963; Brace, 1967; Brace and Mahler, 1971). Brace (1963) argued that the human dentition could be regarded as a structure no longer in use, and technological change in the form of tool use and food processing could be viewed as the conditions which resulted in the relaxation of selection.

More recently McKee (1984) has argued that structures controlled by multiple genes and high mutation rates would need only 40,000 years to decrease in size, and that such reductions are observable in the human dentition since the Upper Palaeolithic.

Calcagno and Gibson (1988) believe that McKee's high estimates are a result of the fact that they are based on a few very common genetic disorders and cannot be applied to the mutation rates in humans as a whole. They argue that other researchers have calculated far lower mutation rates than those of McKee. Using mutation rates calculated by Cavalli-Sforza (1971), they calculate that over twenty-five different loci would have to be involved in controlling tooth size, for a decrease to have occurred over the last 40,000 years. They find this contradicts the results of most researchers who believe that there are only between three and seven loci involved (Calcagno and Gibson 1988).

2.3.7: Increasing Population Density Effect

Machiarelli and Bondioli (1986) emphasize the role of environment rather than genetics on the development of human dentition. They argue that as population density increased during the Neolithic, general standards of nutrition and health standards declined. They view reduction in tooth size as part of a package which also includes smaller stature. They maintain that this stunting would also become re-enforced by selection for small body size as a response to limited nutritional resources.

Armelagos and colleagues (1989) tested this hypothesis using two large skeletal series from groups with differing nutritional status. Their findings did not fit the model created by Machiarelli and Bondioli (1986). A study by Coppa and colleagues (1995) also failed to find evidence to support this hypothesis, as their results showed a decrease in molar size accompanied by an *increase* in health and nutritional standards.

Christensen (1998) also tested Machiarelli and Bondioli's hypothesis (1986) using skeletal material from a broad temporal range. He posited that if the *Increasing Population Density Effect* was in operation, then reduction in tooth size would be generalized, and would also be accompanied by a reduction in stature and an increase in disease. In contrast, if the *Probable Mutation Effect* was responsible for dental reduction, then reduction would occur at an even rate over all time periods, unless there was an absence of any selective pressures in which case the rate would increase. The *Selective Compromise Effect* could be identified by different rates of reduction for different teeth.

The results from this study supported the *Selective Compromise Effect* hypothesis, as the greatest reduction occurred in teeth most susceptible to caries. In addition, the rate of reduction was greatest for the earliest period; the time of greatest technological change.

2.3.8: Heterochrony

Another area of research deals not with dental reduction, but with the altered timing of dental development. This work forms part of a larger body of research which deals with the evolution of an extended growth period in hominids; an issue which plays a central role in the determination of phylogenetic relationships, life history, stature and behaviour.

Mann and colleagues (1990) cite Gould's work on heterochrony as the mechanism responsible for the evolution of paedomorphism; the retainment of juvenile characteristics in adult forms. De Beer defines heterochrony as:

> *"phyletic change in the onset or timing of development, so that the appearance or rate of development of a feature in a descendant ontogeny is either accelerated or retarded relative to the appearance or rate of development of the same feature in an ancestor's ontogeny"* (in Gould 1977: 482)

Mann and colleagues argue that the results of heterochronic change are evident in the relatively late development of the human dentition. There has, however, been disagreement regarding the *timing* of change. Smith (1994) argued that the dental development of Australopithecines was similar in timing to that of modern apes, while Lampl and colleagues (1993) countered that Australopithecine dental development was similar to that of modern humans, and that this was part of a package, which also involved later sexual maturity and an extended learning period.

Unfortunately there are problems in determining the chronological age of fossil teeth. Attempts have been made to use dental microstructures to determine the rates of enamel growth in fossil Hominids (see Bromage and Dean, 1986 and Benyon and Wood 1987). As highlighted by Mann and colleagues (1991), considerable doubt has been cast upon the validity of such studies, since perikymata counts have been demonstrated to underage. However, determination of chronological age *can* be achieved by the thin sectioning of teeth, rather than the reliance on surface manifestations of incremental microstructures (FitzGerald

1995). Before this technique is applied to extinct hominid species, comprehensive comparative data for modern humans must be obtained.

2.3.9: Discrepancy Between Tooth and Jaw Size

If the teeth are too large for the jaw, then crowding causes serious problems, not only due to increased likelihood of caries and infection, but also due to impaction, which can in turn lead to resorption of alveolar bone and the roots of neighbouring teeth. Such crowding has been termed "tooth to denture base discrepancy" (Sakashita *et al.* 1997).

Bone continues to remodel throughout life and is susceptible to environmental pressures, whereas teeth are relatively buffered from the environment, and once developed, the enamel which is the interface with the environment cannot remodel. Calcagno and Gibson (1988) argue that if diet places little stress on the masticatory apparatus, it is possible that teeth will become too large for the jaws. This is because bone resorption will lead to shrinkage of the jaws, while the teeth remain the same size. The response therefore to a soft diet will be selective pressure in favour of small teeth.

Conversely, hard diets will place mechanical stresses on the jaw leading to bone deposition, resulting in large, robust jaws with sufficient space for large teeth. They cite data from the Mesolithic - Neolithic transition in Nubia which shows reduction in tooth size and accompanying reduction in jaw size.

A similar study was undertaken by Sakashita and colleagues (1997). When looking at skeletal material from Japan they found an increasing tooth to

tionship between mastication stress and the size and shape of the mandible (Hylander and Johnson, 1997). This supports the notion that more complex factors than merely a 'soft diet' are involved in the reduction of mandibular morphology.

Ward (1991) has questioned the role of masticatory stress in regard to determining the size and shape of the basal portion of the mandible. Citing experimental, biomechanical work by Knoell (1977), he argues that most of the stress caused by mastication is taken up by the relatively thin, porous alveolar bone. The forces experienced in this portion of the mandible are up to six times greater than those experienced at the basal portion where bone is thick and compact. For this reason, he argued that the mandibles of many primates, including fossil hominids, are "over designed" for the occlusal forces necessary for mastication.

Daegling and Hylander (1997) argue that the methodology of Knoell's work is flawed, as dry bone was used and the basal portions of the mandibles were supported as forces were applied. Their subsequent research used formalin suspended, non-supported specimens. In addition, twisting and bending forces were applied in order to provide a closer replication of chewing. The results of this study, while showing great stress at the alveolar bone, also showed greater stress at the basal portion, particularly on the medial aspect.

Koski also warned of the dangers of oversimplification in the interpretation of masticatory stresses, highlighting the fact that muscles other than those which actually attach to the mandible influence its growth and shape: "The whole soft tissue environment and the occluding dentition together with the bony jaw form the functioning unit" (1996: 545).

The studies discussed above highlight the complexities involved in the relationship between diet, dental reduction and mandibular morphology. The results of experimental work are often contradictory, suggesting that more rigorous research needs to be applied to the study of the biomechanics of mastication. There are obviously difficulties involved in designing such research, as studies of the human mandible must be limited to *in vitro* studies, while *in vivo* studies of non-human primates may be inappropriate and provide misleading results.

Corruccini and colleagues (1983) have discovered that reduction in the masticatory apparatus can occur in as little as one or two generations suggesting plasticity as a response to environmental factors, rather than adaptation in an evolutionary sense. As far as studies of human evolution are concerned, it is difficult to tell if temporal changes in the morphology of fossil hominid mandibles occurred as a result of adaptation in the evolutionary sense, or as a result of plasticity as a response to differing masticatory forces applied during a lifetime.

2.3.9: Discussion

There is still some uncertainty regarding the phylogenetic relationships of early hominid species making it difficult to document dental reduction with any degree of accuracy (Wood 1992). Additional problems arise due to the small sample sizes involved, making variability within and between species difficult to assess.

Researchers attempting to investigate developmental timing are presented with additional difficulties. Such study necessitates the estimation of stages of tooth eruption in the absence of any information regarding chronological age. The fact that it is the dentition itself which is believed to provide the closest approximation of chronological age presents serious problems.

Until the reliability of absolute ageing methods using incremental microstructures is thoroughly explored, hypotheses regarding the timing of prolonged childhood cannot be tested.

2.4: Variability in Modern Populations

2.4.1: Introduction

This section examines studies which have been undertaken to examine variability in the development of the human dentition of modern populations. The way in which researchers have attempted to investigate the controls of dental development is evaluated. An emphasis is placed on studies of the relative roles played by heredity and environment.

2.4.2: Variability in the Timing of Development

Evidence of the degree of variability in the timing of human development is contradictory. Some researchers maintain that there is normal variability as is expected in any biological growth system, while others conclude that the extent of variation is significant. A recent review of the literature on this subject has been undertaken by Tompkins (1996). Smith (1991) discusses the methodological problems involved in assessing variability in the timing of dental development, arguing that statistical treatment of the data is responsible for much reported variability.

Another aspect of research is investigation of the *causes* of variability. There are obvious difficulties involved in this type of research. It is difficult to assess the relative roles of heredity and environment in the absence of controlled breeding experiments. The remainder of this section will discuss ways in which researchers have attempted to overcome such difficulties.

Shapiro (1939) compared the dental development of children of Japanese immigrants in Hawaii with those of Japanese who had remained in Japan. Significant differences in the patterns of development were evident between the two groups, supporting the hypothesis that environment plays a large part in the development of the dentition.

Niswander (1963) studied the effects of inbreeding on the teeth of Japanese children. The more closely related the parents, the more likely the offspring to receive a pair of identical genes. In an inbred population (in the absence of selection) the frequency of a gene will not change. The frequency of a trait however will change due to increased homozygosity. Using data from 3,500 children of cousin marriages, and a control group of similar size, Niswander (1963) looked at dental development in terms of inbreeding and socio-economic group. He found that socio-economic status rather than recessive gene factors were responsible for variation within the population. Many other researchers have arrived at similar conclusions, citing environmental influences as the agents responsible for variability in the timing of development (see Lundstrom, 1948; Crossner and Mansfield, 1983; Mincer *et al.*, 1994).

Pelsmaekers and colleagues (1997) compared the dental development of monozygotic and dizygotic twins. Significantly more variation was found in the timing of development in dizygotic pairs than in monozygotic pairs, attesting to the importance of the genetic contribution to the timing of dental development. Pelsmaekers and colleagues, however, emphasized that there is a need to study monozygotic twins reared in different environments in order to fully understand the relative contributions and genetic and environmental factors.

A more detailed study of third molar agenesis was undertaken by Anderson and colleagues (1975). It was discovered that jaw size was related to skeletal size, but that neither of these variables were related to agenesis of the third molar. Contradictory results were reported by Garn and colleagues, who maintained that third molar agenesis is related to jaw length. They suggested that there is a relationship between absence of this tooth and delayed calcification of other post canine teeth. They further postulated that third molar agenesis is one end of a spectrum resulting from the expression of genetic factors responsible for delayed tooth formation.

Grüneberg's study of mice molars showed how the non-appearance of a tooth occurs when a given tooth fails to reach a certain threshold of size and can therefore be viewed as part of an underlying continuous variable (Saunders 1978). It is therefore not appropriate to view third molar agenesis as a discontinuous trait, nor is it sufficient to discuss this phenomenon in terms of timing alone.

2.4.3: Variability in Tooth Size

Some of the most interesting research into the relative roles of heredity and environment has been undertaken in the field of metric variation in human teeth. Garn and colleagues (1968a) examined the relationship between mesiodistal and buccolingual diameters. A high correlation would suggest the existence of a "size factor" affecting both diameters while a low correlation would suggest an autonomy of factors. Results suggested that the relationship was stronger in the post canine teeth. Sex differences in the relationship were also evident, with a higher correlation in males than females.

A 1967 study by Garn and colleagues examined sexual dimorphism in tooth size. They postulated that if tooth size was under relatively simple genetic control, the dimensions of brother/sister pairs would be similar. They also postulated that sexual dimorphism in tooth size was related to sexual dimorphism in body weight. This issue is of significance, because a relationship between these two variables could imply pleiotropy. Results supported both hypotheses. In addition, a "canine field effect" on sexual dimorphism was suggested, as the second incisors and first premolars were more dimorphic than their neighbours.

Another study by Garn and colleagues (1968b) found evidence to suggest a secular trend in tooth size over two generations. They cite supporting evidence from animal studies to suggest a nutritional cause for this phenomenon. As the father/son increase was greater than the mother/daughter increase, they suggest genetic drift involving the X chromosome. (Maximum differences would occur in the father/son relationship, due to lack of a shared X chromosome).

In contrast, Goose (1967) found no evidence for sex linked inheritance. Instead, low correlations were evident between both sexes and their parents. In common with the study by Garn and colleagues (1967), an increase in tooth size was evident over two generations. Again, improved nutrition was cited as a probable cause.

Family studies are not without problems as genetic variation is not being studied in isolation; usually, a family also shares a common environment. A study of Australian Aboriginal children by Townsend looked at half siblings from polygynous relationships who did not share a common environment (Townsend

1968 in Keiser 1990). Results showed no evidence of X linked inheritance. Townsend suggests that other studies have over-estimated the genetic control of tooth size due to shared environment of most families. Potter went a step further, (1968, in Keiser 1990) criticizing twin studies. Theoretically, a comparison of monozygotic and dizygotic twins should facilitate an assessment of the genetic component in tooth size. Potter argued that at best, we can only conclude that people with the same genotype react similarly to environmental influences.

In a 1979 study, however, Garn claimed that the results of twin studies involving tooth size may be compromised. His data showed that low birth weight babies develop smaller teeth than high birth weight babies. Garn suggests that dental reduction may be a consequence of the survival of low weight babies and that it is possible twin correlations are exaggerated due to small tooth size resulting from the low birth weight of most multiple birth babies.

2.4.4: Variability in Tooth Shape

The shape of teeth can be used to study not only evolution, but diversification and genetic development (Dahlberg 1971). Some researchers have attempted to look at movement of past populations by using dental traits to calculate biological affinity.

An early study by Hanihara (1967) investigated racial differences in crown characteristics. Characteristics were divided into two categories: inter-race variable characteristics which are different between races, and inter-race invariable characters, which are similar between races. Although Hanihara could present no explanation for the development of these differences, he proposed that researchers view them as racial complexes, suggesting they be used to study race histories. Unfortunately, until reasons for the development of these characteristics are understood, such classifications are unsafe. More recently, it has been suggested that the whole concept of "race" is outmoded, and should be regarded as a social construct rather than a biological reality.

Berry (1976) outlined some of the problems involved in the use of dental traits in population studies. She stated that bio-distance can only be investigated if the traits used are biologically determined and their presence is independent of sex and age structure. She also warned against comparing minor crown variants in past populations with those of the present as different selective forces have often played a role in the composition of samples (1976). Traits on the teeth of past populations for example, may have been obliterated by attrition due to an abrasive diet. In contrast, many modern populations have high caries rates which may destroy a different part of the crown, thus obliterating different features. Berry suggests that these two different agents of destruction affect different traits, and that this negates the validity of many comparisons (Berry 1976).

Mayhall and Kanazawa (1989) avoided such pitfalls in a three-dimensional analysis of the Canadian Inuit first molar. In this study only teeth with no attrition, caries, or dental restoration work were included. This facilitated comparisons with other ethnic groups, as different diets, cultural practices and demographic structures would not have a differential filtering effect on the sample. The results of this study revealed that there was a similarity between Japanese and

Canadian Inuit with regards to cusp height, intercuspal distances and degree of sexual dimorphism. In contrast, Dutch teeth showed less relief. As the Japanese and Inuit do not share a common environment or culture these factors are concluded to play only a minor role with the shape of the teeth being strongly genetically determined. This concurs with Hanihara's idea of a "Mongoloid" dental complex.

An earlier study by Turner II (1967) claimed that modern Eskimos can be considered "less Eskimo" than their ancestors as some non-metric traits are now diluted, bearing a closer similarity to American Caucasians. As not all traits were proportionately diluted, Turner suggested a number of possibilities including, sampling problems, complex modes of inheritance and admixing with Japanese and Chinese populations.

Dahlberg (1971) discussed the biological development of the crown morphology, emphasizing the way in which complex timing of dental development can dictate traits on the crown. Altered enzymes can cause biochemical blocks which affect the development of any organ. Such alterations can result from the change or loss of a single protein. This can cause change in the rate of growth or calcification, thus altering the original pattern. This can, in turn, lead to the denial of full development or the presence of ridges as a result of acceleration of growth. As these are all genetically controlled, environment plays only a minor role.

Butler's investigation of fetal development, shows how the timing of calcification affects morphology. The onset of calcification can freeze a stage of formation, altering its phenotype, therefore retarded calcification gives the cell more time to divide resulting in different morphology (Butler, 1969).

It is therefore micro differentiation of the timing of calcification of various parts of the crown which dictates morphological traits. Dahlberg (1971) suggests that environmental factors compete with phylogenetic forces, playing a relatively minor role in the final shape of the tooth.

2.4.5: Variability in Fluctuating Asymmetry.

Theoretically, adverse environmental conditions can alter the genetic blueprint of the tooth, which has dictated a symmetrical dentition, and result in what is termed fluctuating asymmetry. This type of asymmetry can be viewed as a failure of symmetry, as opposed to directional asymmetry, which is part of the design of an organ, for example the heart or lungs (Perzigian,1977). Fluctuating asymmetry is also referred to as random, or non-directional.

The study of fluctuating asymmetry has been used in attempts to investigate the degree of environmental stress within a population. A study by Perzigian (1977) compared environmental stress between archaeological groups, and found that populations with a high degree of fluctuating dental asymmetry also had a high prevalence of other growth stress markers. It was suggested that the degree of fluctuating asymmetry paralleled environmental conditions.

Unfortunately, problems exist when attempting to infer health status of skeletal populations. Different populations are subject to different archaeological filters. For example, Perzigian (1977) stated that the high infant mortality rate of the Indian Knoll sample is an indication of environmental stress. A high percentage of infant skeletons in a population however, can be an indication of a rising birth rate, and the presence of stress markers can be an indication that individuals were healthy enough to survive environmental stress long enough to develop stress markers (Wood *et al.* 1992).

Methodological problems arise when researchers do not correct for overall crown size, when comparing fluctuating asymmetry between populations. For example Suarez's (1974) conclusions regarding high levels of fluctuating asymmetry among Neanderthals may be a result of large tooth size, and therefore invalid.

Smith and colleagues (1982) presented a review of the literature of fluctuating asymmetry, and stressed the methodological problems involved in such studies. They state that most archaeological samples are unsuitable for analysis due to small sample size; if this is less than a hundred, then small differences will remain undetected. They suggest the study of samples with a high level of defined stress, such as fetal alcohol syndrome is necessary to study this phenomenon.

In summary, many researchers have used fluctuating asymmetry to infer the health status of past peoples. This may be inappropriate, due to methodological difficulties. Such studies may be premature because the biological processes which result in such asymmetry are not yet understood. More research into the relationship between fluctuating asymmetry and other supposed signs of dental developmental stress would help clarify matters.

2.4.6: Discussion

The problems involved in attempting to assess the magnitude of the role played by heredity in controlling dental development and size are significant due to difficulties in eliminating environmental variables. Populations which are genetically distant tend to come from different geographic areas or practice different cultures. As discussed above, even family and twin studies are not free from problems.

The difficulties involved in assessing variability must also be stressed. Small sample size, sample bias and statistical treatment of data may have contributed to much of the variability which has been documented. The contradictory results reported in most areas of research suggest these factors, accompanied by inter-observer error are playing a role. Ideally, consistency in methodology and researcher would eliminate some of these problems.

2.5: Conclusion

Research into dental development and its controls continues to advance as efforts are made to improve our understanding of human growth and development. There remain, however, several areas which require further investigation. Some of the main problems with this area of research are outlined below.

How useful are models of dental development? Although they may provide ways of visualizing developmental processes, they cannot be regarded as theories since it is not possible to formulate testable hypotheses to either support or refute them. It may be true that the difference between the clone and field model is so small as to be irrelevant. Research into fetal development using microscopic techniques will do far more to further knowledge of human dental development and its controls than academic discourse about the merits of field versus clone theory.

The main problem involved in research into evolutionary change is one of sample size. Attempts are being made to establish the causes and mechanisms of dental reduction when the data which documents this reduction are scanty. Samples of hominid fossils are rare and phylogenetic relationships between species unestablished. This makes it difficult to know when and how dental reduction occurred.

Additional difficulties arise when attempting to assess the role played by levels of masticatory forces on dental reduction. As discussed above, replicating the forces involved in human chewing in *in vivo* experiments is problematic, as is inferring such forces through observations of living, non-human primates.

Research into an extended period of childhood dependency is also problematic due to difficulties in estimating chronological age. More work on absolute ageing methods is required if the hypotheses formulated by Smith (1994) and Lampl and colleagues (1993) are to be tested.

Attempts have been made to explain variability in the absence of certainty about the extent of variability. Scientific research involves the design of methodologies which produce replicable results. Unfortunately, the constant refinement and improvement of techniques, methodologies and standards, produces results which cannot be compared with those of earlier studies.

In summary, more comparable data are required in all three areas of research before questions regarding developmental controls, evolutionary change, and causes of variability can be answered.

Chapter 3

The Samples in Context

3.1: Introduction

This study involves the comparison of four sets of data in order to assess the degree of variability in dental development both within and between different population groups. It is important therefore, to assess the composition of the samples since all four have been subjected to different biases.

Three of these data sets are derived from skeletal samples from archaeological contexts. The fourth data set is from a clinical study. Cemetery samples are used frequently to glean both cultural and biological information about past peoples. In common with all archaeological samples, human skeletal remains derived from cemeteries must be regarded as *samples* of populations. It must also be recognized that these samples are probably not representative of the populations from which they were derived. Clinical data are also subject to sampling biases. When the two types of data are combined in a study such as this, there is the possibility that these problems will become magnified.

It is recognized that the four data sets come from different geographical locations, time periods, and cultural backgrounds. This chapter therefore, places samples in their geographical, historical and cultural contexts in order to assess their degree of representativeness. Where possible, the direction of any biases is highlighted and discussed.

A concluding section evaluates the validity of comparisons used in this thesis, discussing the implications of various forms of bias. The potential degree to which the validity of interpretation may be compromised is also assessed.

3.2: Poundbury

3.2.1: Introduction

Poundbury, located outside the walls of Durnovaria (Roman Dorchester), is the largest Romano British cemetery to have been excavated to date (Farrell and Molleson, 1993). It yielded the skeletal remains of over 1000 individuals, 378 of which were sub-adults. Dental information for this study was gleaned from 186 sub-adult individuals who were sufficiently preserved for this purpose.

Using both historical and archaeological sources, this section provides a brief overview of what is known of the Late Roman period in Britain, the period from which this cemetery can be dated. In order to facilitate a discussion regarding the representativeness of this sample, information regarding the site itself and the excavation is provided.

3.2.2: Historical Context

The main Poundbury cemetery has been dated from the mid third to mid fourth century AD, placing it within the Late Roman period. Britain had been invaded by Julius Caesar four centuries earlier in 54 BC, but the systematic conquest of the province did not begin until the Claudian invasion of AD 43 (Frend, 1968).

In spite of often violent resistance, which at times culminated in full scale revolt, Briton remained part of the Roman Empire until AD 410. During these centuries the army maintained a stronghold in the province, exposing the natives to Roman technological and cultural influences in the form of language, religion, architecture, engineering, and legal and political systems. Archaeological evidence however, suggests that only the native nobility and urban dwellers adopted Roman culture and that rural inhabitants maintained a traditional lifestyle with minimal Roman interference (Collingwood and Richmond, 1971).

Archaeological evidence reveals three main classes of Romano British settlement: villas, homesteads and towns. Villas can be described as Romanized farms varying in size from small, two-room 'cottages' with a few acres of land, to grand country houses with substantial arable or pastoral lands. In contrast, homesteads were 'native villages' subsisting by means of agriculture. Archaeological evidence indicates that such communities experienced a very low degree of Romanization; although there are some signs of Roman farming innovations, there is little sign of Roman culture. Over 700 such settlements have been found in Great Britain and it is thought that there are probably many more which have yet to be located (Collingwood and Richmond, 1971).

Roman towns also varied in size, the largest being typified by Londinium and Verulanium, which acted as administrative centres for the province. More typical are the tribal capitals such as Durnovaria, the capital of the Duritriges tribe. These tribal capitals were created by the Romans in order to maintain the tribal structure of Briton, thus easing the transition to full Romanization. The tribal nobility were easily persuaded to build such towns after defeat, and were granted privileges by the Romans. The nobility performed an administrative function for the Roman empire, with tribal leaders becoming administrative officials for the empire.

In the case of the Duritriges, the nobility were moved to Durnovaria after the defeat at the Iron Age hillfort at Maiden Castle several miles away. The tribal leader became a "vice-Emperor," with the local nobility forming a senate. Durnovaria, in common with other tribal capitals, served no real economic purpose, forming a parasitic relationship with the surrounding countryside. It did however, perform an important cultural and political role by linking the remainder of the native population to the rest of the Roman Empire (Collingwood and Myers, 1949).

Such towns had an open forum which served as a market place, shops, basilica and other government buildings, bath houses and residences in the form of individual houses of varying size. Archaeological evidence indicates that the streets were well paved and drained, and the houses well built.

The population size of such towns is difficult to assess as there are no extant statistical records relating to such parameters. Based on archaeological evidence, the population of Durnovaria is estimated to be somewhere between 1000 and 2000 (Collingwood and Richmond, 1971). Such figures must be treated with caution, however, as the population of tribal capitals probably fluctuated throughout the period. Much has been written about population decline throughout the Roman Empire during its latter years, but in the case of marginal provinces this may be misleading. There is evidence of urban decay in most tribal capitals throughout Britain from the 3rd century AD onwards. It can be debated, however, whether this reflects abandonment in favour of a more traditional, rural lifestyle or an actual decrease in numbers on a regional scale.

3.2.3: Site and Excavation

The excavations at Poundbury comprise two main sites; Poundbury camp, a series of earthworks spanning a 2000 year period, and Poundbury cemetery. The latter contains Bronze Age and Late Iron Age/Early Roman burials, but the main part of the cemetery dates to the Late Roman Period.

The first discovery of skeletal material occurred during construction of a prisoner of war camp in 1915. Full excavation of the cemetery however, was not undertaken until trial excavations in 1966 hinted to the extent and importance of the site. Work continued until 1986, when the excavation was finally completed (Farrell and Molleson 1993).

Although the cemeteries yielded one Bronze Age burial and 59 Late Iron Age/Early Roman burials, the vast majority (over 1100) date from the Late Roman period. Men, women, and children appear to have received similar burials (typically, east-west alignment, supine, with no grave goods) and are randomly distributed throughout all burial areas. Analysis of non-metric traits suggests that burials often occur in family rows. All these factors have led researchers to designate Poundbury as a Christian cemetery (Farrell and Molleson 1993).

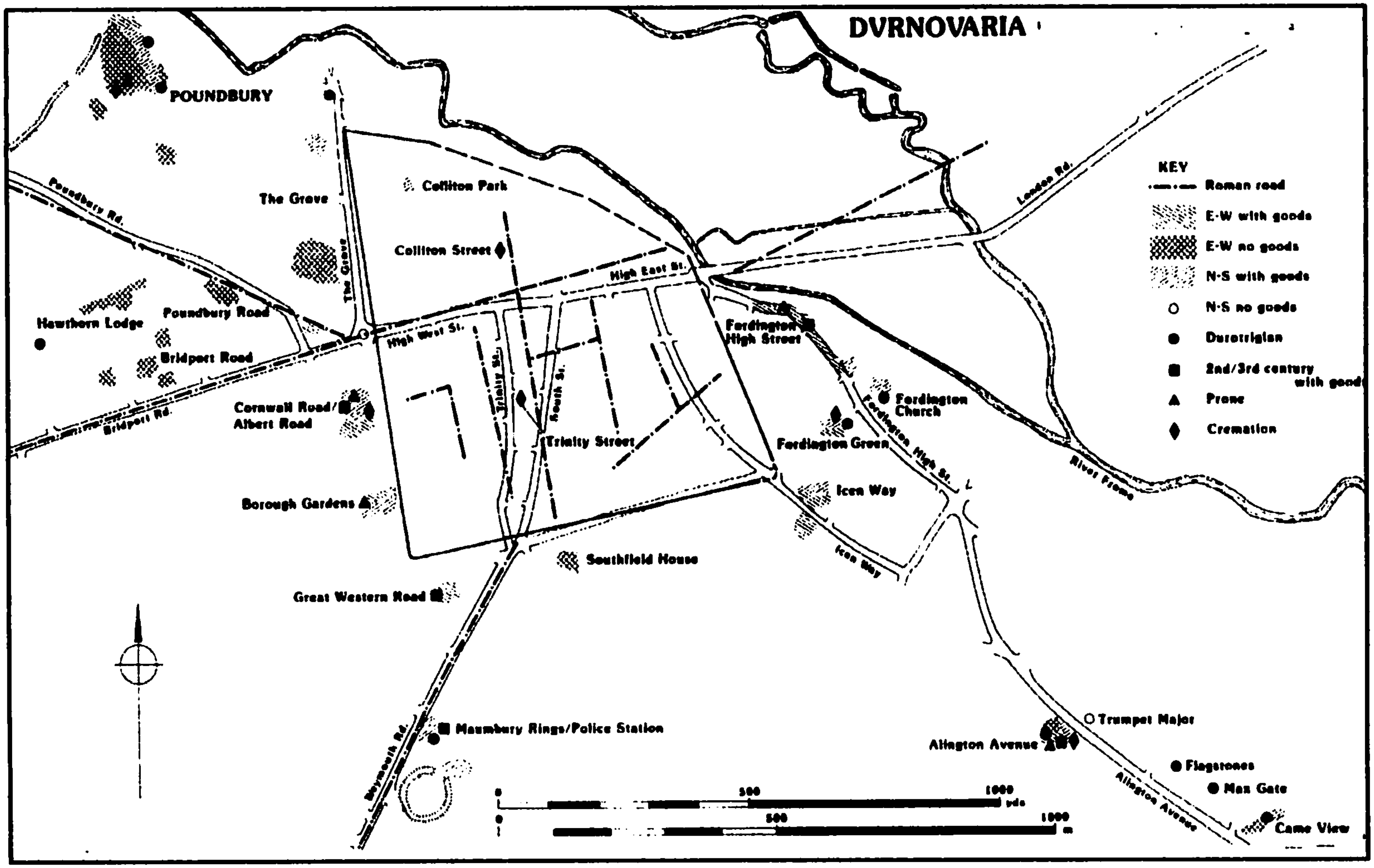


Figure 3.1: Spatial relationships between Durnovaria and surrounding burial sites.

(From Farrell and Molleson, 1993)

The large amphitheatre at nearby Maumbury Rings, which provides far more room than the population of Roman Dorchester required (Collingwood and Myers, 1949), suggests that the area was a centre for ceremonial activities for both the urban *and* rural Romano British population. It can therefore be argued that the cemetery would also be likely to have served both sections of the population. What can be suggested with a degree of certainty is that the inhabitants of the "native villages" would not have been represented. There is evidence, in the form of shallow graves outside village ramparts, that this section of the population retained their traditional Iron Age funerary rites (Collingwood and Myers, 1949).

As stated above, population numbers for this period are difficult to estimate. This makes it difficult to assess what proportion of Romanized Britons from the area were buried there. Based on the period of cemetery use and a population estimate of 1,500 for Poundbury, the Crude Death Rate is 4.9. This is far too low for a non-industrialized population (McVey and Kalbach, 1995), suggesting that some form of cultural selection was involved. It must be remembered however, that Poundbury is only part of a larger complex of settlements and cemeteries (see figure 3.1). Evidence suggests that some families from the area were buried at Poundbury, while others were buried in nearby cemeteries (Farrell and Molleson 1993). Unfortunately, it is not possible to assess whether those buried at Poundbury were of higher or lower status than those buried in the smaller cemeteries.

In conclusion, Poundbury contains individuals from the more privileged, Romanized sections of society; the poorer, native population is not represented.

3.3: Spitalfields

3.3.1: Introduction

Built in the 18th century, Christ Church, Spitalfields is located in the east of London, England. An excavation of its vault yielded the remains of 143 sub-adults with sufficiently preserved teeth to be used in this study. It was possible to trace the parish records of 383 of the total of 800 burials which were excavated. This makes the Spitalfields collection a valuable resource, not only for studying post-medieval London, but for the testing the precision and accuracy of sexing and ageing techniques (Molleson *et al.* 1993).

3.3.2: Historical Context

The life-spans of the individuals buried in the vault of Christ Church, Spitalfields occurred during the period ranging from 1642 with the birth of the first individual buried to 1842 with the death of the last. These two centuries were a time of great social, economic and technological change. Events such as the London plague, the fire of London, wars with America and France, the Luddite riots, and changes in the corn laws, all contributed to fluctuations in diet and health standards (Molleson *et al.*, 1993).

In spite of the social and political events mentioned above, the population of the country as a whole had risen by two-thirds to 10.7 million by the middle of the 18th century. During the following fifty years, it doubled to over 20 million. Earlier marriages, innovations in agriculture resulting in improved diet, the decline of chickenpox and social reform have all been cited as factors contributing to this increase (Floud and McLosky, 1981).

The population of the capital city of London rose from 0.5 million in 1701 to 2.5 million in 1851. At this time, Great Britain was the first country in the world to have more urban than rural inhabitants. This increasing urbanization was fuelled by economic change caused by innovations in the steel, mining and textile industries, which resulted in London becoming the centre of industry and commerce (Floud and McLosky, 1981). These changes affected people throughout the country, and the inhabitants of the London parish of Spitalfields would have been no exception.

Many churches had been destroyed in the fire of London in 1666 which, combined with the increasing population of the city, resulted in plans to build 72 new churches throughout the city. This number was later modified to 50, although only 12 were actually built. Christ Church, Spitalfields was one such church, providing a place of worship for Anglican members of the 20,000-strong population of the parish.

The parish had a thriving silk industry with many wealthy merchants and master-craftsmen residing there in high quality housing. A large proportion of the population, however, was extremely poor; the parish contained a workhouse, four almshouses and a charity school. The minute book of Christ Church Vestry described the parish as "inhabited almost entirely by poor persons" (Sheppard, 1957:7 quoted in Molleson *et al.*, 1993).

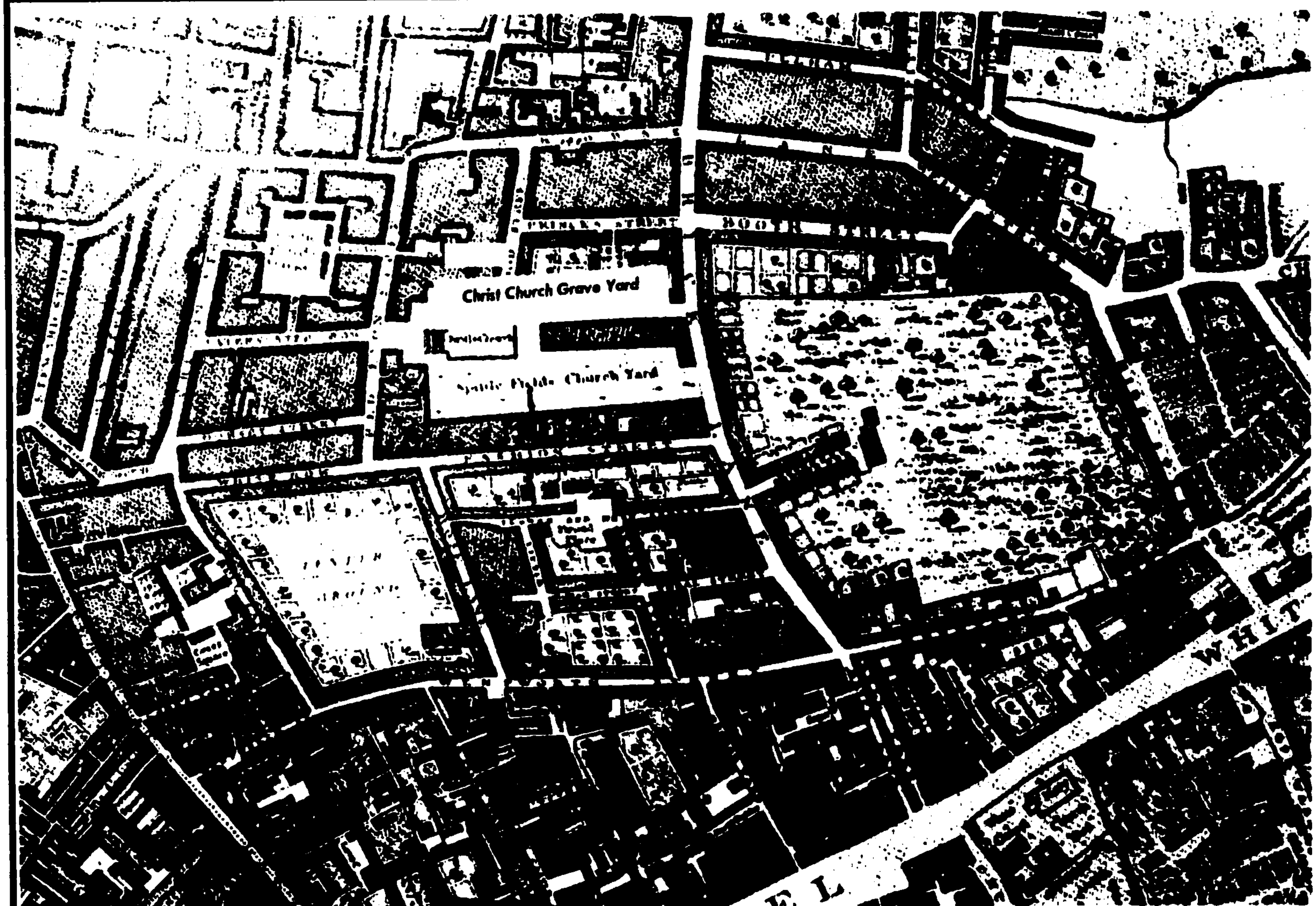


Figure 3.2: the Site of Christ Church, Spitalfields, 1730.

The parish also included a large number of Huguenot refugees and their descendants. This section of the population was part of a million strong emigration from France who had fled the country due to religious persecution following the revocation of the Edict of Nantes by Louis XIV in 1685. The majority of individuals who found refuge in Spitalfields were engaged in the silk industry in some capacity. Parish records show that these individuals ranged in socio-economic status from very poor artisans to members of wealthy families who ran successful businesses. Other occupations included gold and silver smiths, and watch and clock makers. (A summary of occupations for the named sample is given in table 3.1).

Category	Percent	Category	Percent
Artisan	47.9	Merchants	5.2
Master Craftsmen	31.8	Wholesalers	2.5
Professionals	11.0	Independently Wealthy	2.1

Table 3.1: Distribution of occupations for the named sample (data from Molleson et al., 1993)

3.3.3: Site and Excavation

Christ Church, Spitalfields was consecrated in 1729, followed by the first vault burial later in the year. The crypt extends under the entire building and contains seven bays which correspond to the seven aisles of the church itself. The burial register of the church contains records of 68,000 burials in all. A total of

983 were recovered from the vault, and comprise 98% of all vault burials. The remainder are believed to be buried in the church yard (Molleson *et al.* 1993).

The excavation of the vault of Christ Church, Spitalfields was undertaken as a result of plans for restorations to the building in the late 1970's. A proposal in 1973 resulted in the commencement of archaeological excavations in the following year. The aims of the project were as follows:

1. To improve archaeological methodology for this type of excavation,
2. To improve understanding of human biology,
3. To add to knowledge of post medieval archaeology,
4. To improve understanding of site formation.

These aims were achieved to a great extent, as many coffin plates providing age, sex, and date of birth and death were surviving in situ. Post excavation analysis of the skeletal material began in October 1983 and was undertaken by members of the anthropological team from the British Museum: Natural History.

3.3.4: The Cemetery Sample

The excavation of the vault yielded the remains of 983 individuals, with males and females occurring in equal numbers. The remains of 215 sub-adults were included in this number. Preservation of the skeletal material varied from dry and firm, to wet, soft and damaged. Bone tissue was compromised in many cases due to chemical degradation. In some cases the remains of hair, ligaments and skin were preserved.

The named sample (those for whom records survive) comprises 383 individuals. Birth dates span 200 years, with 75% of individuals born during the 18th century. In the named sample, 3.4% of individuals are recorded as being from outside England, but French names suggest a Huguenot origin for 42%. Metric data, however, shows that Huguenot and non-Huguenots were not physically different (Molleson *et al.*, 1993).

There is a high prevalence of dental caries and a low rate of attrition, indicating a soft diet which is high in sugar. Obesity is suggested in many cases by the preservation of adipocere and evidence of DISH*. Documentary evidence for the period shows that the "well off" existed on a diet high in alcohol, and animal protein and fat, with very little fibre. The diet of the poor, although deficient in calories, was healthier in content, with high levels of cereal and fish (Drummond and Wilbraham, 1991).

3.3.5 Discussion

The Spitalfields collection provides valuable information relating to the lives of those who were buried there. Can this information be applied to the population of England as a whole, or even to those living in the parish of Spitalfields? As discussed above, 18th and 19th century England can be regarded as an urban, industrialized country, so the parish of Spitalfields was by no means unusual. It is debatable, however, whether the skeletal collection can be used to portray life in the Spitalfields in general. As only the vault has been excavated, little is known of

* Diffuse idiopathic skeletal hyperostosis

the majority of those buried in the parish. The church vault can be regarded as exclusive and prestigious as only the well off could have afforded the high cost incurred by this type of burial. Prestige, the desire to be close to a source of spiritual power, and a horror of earth and decay would have prompted those with the financial means to invest in a vault burial (Molleson *et al.* 1993).

Parish records confirm that while not of the aristocracy, the Spitalfields collection represents successful wage earners and provides no information relating to the thousands of poor and destitute residing in London in the 18th and 19th centuries.

3.4: Belleville

3.4.1: Introduction

St. Thomas's Anglican Church is located in the town of Belleville in southern Ontario. An excavation of the churchyard yielded a skeletal collection comprising the remains of 577 individuals who had been buried during a 53 year period between 1821-1874 (Saunders *et al.*, 1995a). The teeth of 229 of the sub-adult skeletons were sufficiently preserved for the purpose of this study.

This section provides a brief outline of Belleville during this period, allowing the skeletal collection to be placed in an appropriate temporal and social context. A brief description of the excavation and skeletal collection are also included, followed by a discussion regarding the representativeness of the sample.

3.4.2: Historical Context

The town of Belleville was founded by Captain Meyers in 1816. Situated at the mouth of the Moira river, Belleville was strategically placed as part of the water system which includes the Bay of Quinte, Lake Ontario and the River Trent. At this time it was a small settlement of loyalist farmers and trades-people, the town plan recording only 45 buildings, including frame-dwellings, stores, stables, a distillery and a post office (Mika and Mika, 1973).

By the 1830's, the town's population had reached 1700. The number of private dwellings now exceeded 200, with the town now spreading over both sides of the river. A breakdown of business buildings is listed in table 3.2. Further advances occurred over the following decades, with improved communications in the form of gravel roads, steam ships, and rail travel. The 1861 census records the town's population as 6,000. By 1876, this figure had risen to 11,120 (Mika and Mika, 1973).

No.	**Building**	**No.**	**Building**	**No.**	**Building**
5	Churches	2	Flour Mills	1	Paper Mill
1	Axe and Sythe Factory	6	Taverns	4	Sawmills
1	Patent Pail factory	1	Iron Foundry	7	Blacksmith Shops
2	Tinsmith Shops	20	Merchant Businesses	6	Medical Offices
5	Schools	3	Saddle and Harness Factories	1	Watchmaker
2	Hatters	3	Cabinet Makers	2	Chair factories
10	Shoemakers	7	Tailors	3	Bakers
3	Butchers	3	Potasheries	6	Dock Warehouses
10	Groceries	2	Drugists	7	Milliners
3	Breweries	3	Carriage and Wagon Factories	4	Cooper Shops
1	Distillery	3	Tanneries	2	Brickyards
1	Lathe Factory	4	Lawyers offices	1	Notary Public
1	Land Registry				

Table 3.2: Buildings of Belleville, 1834 (Data from Mika, 1973)

3.4.3: Site and Excavation

The excavation of the burial ground of St. Thomas' Anglican Church was prompted by plans to develop a parish hall on adjacent land. The 577 graves unearthed by Northeast Archaeological Associates during the summer of 1989 made St. Thomas' Cemetery one of the largest historical skeletal collections in North America (Saunders *et al.*, 1995b).

3.4.4: The Cemetery Sample

It is estimated that the 577 burials are one third of the total interments which took place at St. Thomas' between the years 1821-1874 and so are a sub-set of the cemetery population as a whole (Hoppa, 1996). Preservation of the 577 individuals was excellent due to the well-drained sandy soil and elevated location. This facilitated age and/or sex estimates for 97% of the burials (Saunders *et al.*, 1995a).

The observable sample of 558 comprises 276 adults and 282 sub-adults, 148 of whom are aged under one year. It was possible to gain exact information regarding the demographic parameters of 72 of the 558 observable skeletons since surviving coffin plates could be matched to entries in the parish register (Saunders *et al.*, 1995a).

Parish records indicate that although Belleville escaped the cholera epidemic which prevailed in Upper Canada at this time, infectious disease did pose an environmental problem for the inhabitants of Belleville, with children exposed to smallpox, measles, meningitis, scarlet fever and whooping cough. Skeletal evidence suggests that it was these acute infections rather than chronic infection and malnutrition, which had the greatest impact on the mortality and morbidity of the population (Saunders *et al.* 1995b).

3.4.5: Discussion

The congregation of St Thomas's, was Anglican, a denomination making up one quarter of the town's population at the time (Saunders *et al.*, 1995b). Beyond this, the boundaries of the contributing population are difficult to define. At the beginning of the period of cemetery use, the population was probably largely rural, becoming more urbanized towards the end of the period (Herring *et al.*, 1991). This situation was typical of Upper Canada in the 19th century, which was a time of great expansion, and technological and social change. Comparison of the skeletal data and parish records show that the Belleville Collection is representative of the parish of St. Thomas'. Saunders and colleagues (1995a) suggest a study comparing the St. Thomas's parish records with the decennial censuses for 1851-1881 in order to assess the representativeness of the Anglican population. Until this work is completed, the relationship between the individuals in the skeletal sample and the population of Belleville as a whole remains uncertain.

3.5: Burlington

3.5.1: Introduction

Modern clinical data from the Burlington Growth Study plays an important role in this study as it is used to represent modern children of European origin. The longitudinal data of 1300 children in the study provided cross-sectional data for 207 individuals. This section provides information relating to the time period in which these data were collected, allowing it to be placed in a historical and social context. A brief account of the growth study itself is also provided, followed by a discussion examining the representativeness of the sample.

3.5.2: Historical Context

The children of the Burlington Growth study were born in the late 1940's, placing their early growth period at the beginning of the post war period. In Canada, this was a time of great optimism, as the federation had just been completed, and the country was experiencing a great post war economic boom. Oil supplies in Alberta and iron ore deposits in Northern Quebec and Labrador had recently been discovered, adding to the county's mineral wealth. Technology was advancing as uranium sources were developed in Northern Ontario, and hydro-electric power stations were developed throughout Canada. Manufacturing both expanded and diversified, and the GNP of Canada rose from $12 billion in 1946 to $30 billion in 1957. Much of Canada's resources were invested in the health and welfare of the population. The arrival of 1.5 million immigrants, chiefly from the United Kingdom, is indicative of the mood of optimism and expansion during this period.

Burlington was ideally located to benefit from this expansion being situated on the north shore of Lake Ontario between Hamilton and Oakville. The town was incorporated into the communication and transportation network running around the Golden Horseshoe from Toronto to Niagara Falls. By 1967, 48% of the town's 19,000 work force was employed in the town itself, the remainder working in Oakville, Hamilton and Toronto. 'White collar workers' comprised 65% of the workforce, the majority of whom were employed as clerks and managers in either the steel or car industry. Of the remainder, 16% were skilled, 6% semi-skilled and 10% unskilled (C.C.C., 1967).

Throughout the period, wages were rising well above the rate of inflation, but the increased standard of living was accompanied by longer working hours and more responsibility. Although the town was experiencing rapid growth and the majority of its population enjoying an affluent lifestyle, some social problems persisted. Many families were facing large debts, and the lack of low cost housing was a problem for the low paid, unskilled members of the community.

The 5,000 strong population of 1952 rose to 33,000 in 1958, and by 1966 this figure had doubled to 66,000. The 1961 census shows that 70% of this population was of British origin and 27% was of European origin other than British. An above average income was earned by 70% of the population of Burlington, with only 20% being classed as 'low income families' (C.C.C., 1967).

Although good medical care was provided for the residents of Burlington, facilities and agencies catering for the social welfare of the citizens were less comprehensive. Social concerns at the time include alcoholism, underage drinking, increasing juvenile delinquency, and lack of sex education in the home (C.C.C., 1967).

3.5.3: The Burlington Growth Study

In 1952, the Burlington Growth Study was initiated by Doctors Moyers, Grainger and Mitton and was administered by the University of Toronto. The study continued until 1971 when most of the children had reached age 20. Initially, the sample comprised 75 children between the ages of 3 and 12. In the following year, the longitudinal study of 1,380 children began.

The original objectives of this research were defined as follows:

1. To evaluate the provision of early orthodontic treatment
2. To accumulate growth records which would help in the early diagnosis of malocclusion
3. To evaluate the roles of general practitioners and consultant services
4. To assess the contribution of inheritance in malocclusion by conducting family studies.

These objectives were then extended to include the collection of anthropometric data in order to investigate the relationship between malocclusion and general physical development, and also to investigate the relationship between periodontal disease and faulty occlusal stresses.

The following information was collected from the 1300 children who participated in the study. (These children represented 85-90% of their age co-horts for the 1953 population of Burlington). An inventory of the total sample of children included in the clinical study is summarized in table 3.3.

Orthodontic records

1. Case History
2. Clinical examination
3. Cephalometric radiographs: 3 laterals, 2 obliques and 1 anterior posterior
4. One hand and wrist radiograph
5. Impressions for dental casts
6. Height and weight records

Anthropological Data

1. Measurements of somatic growth: height, weight, length of trunk and limbs, breadth of shoulders and hips, girth of chest and abdomen, bone thickness, handgrip strength
2. Medical examination and history: family and health history, nutrition, congenital defects, postural problems, progress of puberty
3. Behavioural data: interests, activities and vocational interests

Periodontal data

1. Intra-oral examination and recording of: gingival colour, debris and plaque, calculus and pockets
2. Bite wing radiographs or interproximal alveolar bone record

3 Year Serial Experimental Group

Age	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23
Males	132	167	161	154	149	144	141	141	136	127	125	122	35	116	86	101	32	68			
Females	100	136	132	130	126	122	121	117	116	115	113	109	0	103	47	86	31	57	1		
Total	232	303	293	284	275	266	262	258	252	242	238	231	35	219	133	187	63	125	1		

C-6 Serial Control

Age	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23
Males				168	2		138	3	3	133	3	91	3	105	1			53	4		
Females				129	1		107	1	1	101	1	70		76	2			49	2	1	
Total				297	3		245	4	4	234	4	161	3	181	3			102	6	1	

C-8 Control

Age	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23
Males						96				1	4	12	1	20				24	2		
Females					1	123	3	1		6	3	17	2	25				21	8		
Total					1	219	3	1	0	7	7	29	3	45	0	0	0	45	10		

C-10 Control

Age	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23
Males								111		1				19	3			4			
Females								106	2			1	2	12				1			
Total								217	2	1		1	2	31	3			5			

C-12 Control

Age	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23
Males									1	113	1			1	2	1		56	8		
Females									1	99	1			1	2			37	4	1	1
Total									2	212	2			2	4	1		93	12	1	1

Family Study: Siblings

Age	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23
Males		2	4	19	8	28	29	31	7	35	5	36	1	33	1	3	3	3	1		
Females	1	3	4	25	11	37	43	45	3	44	8	43	3	43	1		1	3	1		
Total	1	5	8	44	19	65	72	76	10	79	13	79	4	76	2	3	4	6	2		

Family Study: Parents

Males = 151 Females = 161 Total = 312

Table 3.3: Inventory of data collected for the Burlington Growth Study

3.5.6: Discussion

As stated above, the Burlington Growth Study contains data which are derived from 85-90% percent of the children of Burlington, making it the most representative of all the samples discussed in this chapter. It appears however, that ethnic minorities are under-represented, suggesting that many children from such backgrounds are part of the missing 10-15%. Although this is useful for the purpose of this study, which involves an investigation of variability between individuals of European origin, it does compromise the representativeness of the sample, as does the fact that the population of Burlington enjoyed a standard of living above the national average (CCC, 1967). In summary, the Burlington Growth Study represents individuals from Middle Class families of European origin; the native population, recent non-European immigrants, and low income white families are not represented.

3.6: Conclusion

As this study involves comparisons of four different samples in order to assess variability, it is important that the populations represented by these samples are of a comparable nature. The main sources of bias which could present potential problems in this regard, are as follows:

A: Length of Time.

The children involved in Burlington Growth Study were all born at the same time and lived at the same location. They can therefore be regarded as a true biological population. The other three samples are not comparable in this regard. The period of cemetery use ranges from 53 years in the case of St. Thomas' Anglican Church Belleville, to 200 years in the case of Christ Church Spitalfields. It is possible that a secular trend is involved, as these collections comprise individuals of several generations. If more variability is evident in the collections comprising individuals whose lifespans range over a longer time period, then this must be taken into account.

B: Socio-economic status.

If some of the collections involved in this study are representative of the highest socio-economic groups while others are representative of the entire contributing population, then comparisons of the biological data from these collections may be invalid. As discussed above, the Poundbury, Spitalfields, Belleville and Burlington samples, all provide information about the relatively well off members of society, and so are comparable in this regard. Dangers would arise if biological information was used to make inferences regarding health status for the various time periods in general, but such work is beyond the scope of this study.

C: Mortality bias.

The Burlington Growth Study provides information about living children, whereas the three archaeological samples can only provide information about dead children. It is possible that such archaeological samples would provide biological information which is unrepresentative of the *living* children of the populations from which they are derived. This is because individuals who die young are often the weakest, most frail members of society. (Wood *et al.*, 1992).

Information from the parish records at St. Thomas's, Belleville, however, shows that acute infections, rather than chronic conditions were responsible for the majority of sub-adult deaths. In this instance therefore, the skeletal sample is not comprised of children whose health had been compromised over the long term. Information of this nature for Poundbury and Spitalfields is unavailable, so some problems could occur when using these data. For this reason, caution must be used when interpreting any comparisons.

In summary although the biases outlined above may present some problems, a knowledge of the extent and direction of these biases will do much to alleviate their seriousness.

Chapter 4

Patterns of Human Tooth Formation:

Is there a Significant Difference in Patterns of Tooth Formation Between Populations of Close Biological Affinity?

4.1: Introduction

There has been much research into human dental development, the results of which have often been contradictory. Some researchers claim that the rate and pattern of human tooth formation has remained constant since the emergence of modern humans (see Holz 1959 and Smith 1989), while others believe there is great variation both within and between populations, and that differences between populations of distant biological affinity can be observed (Crossner and Mansfield 1983; Owsley and Janz 1983; Lampl *et.al.* 1993, Tompkins, 1996).

Since these differences have been attributed to genetic rather than environmental differences, there is a need for research into differences in dental development between populations of close biological affinity existing in different environments. This chapter addresses this research problem by comparing the dental development of the populations described in chapter 3.

Existing research has not demonstrated conclusively either variability or consistency in the timing and rate of human tooth formation. This is because it is not possible to determine if the apparent variability reported by some researchers is due to true developmental variation rather than methodological variation. Put simply, comparisons between samples have been frequently subjected to different sampling biases, inter-observer differences and different statistical analyses, and are therefore not comparable. By eliminating methodological factors as potential sources of variation, this chapter provides a more accurate assessment of the degree of inter-population variation in dental development.

4.2: Methodology

4.2.1: The Samples

In order to test the hypothesis that there is a significant difference in the timing of dental development between populations of close biological affinity, the four samples described in chapter three are compared.

Sex and age of the Burlington children is known. For Belleville and Spitalfields however, this information is available for less that half of the individuals. In the case of Poundbury this information is unavailable for all individuals. This necessitated the creation of a method of analysis which facilitates the comparison of patterns of development in the absence of chronological age (This will be discussed further in the following section). Sample sizes range from 143 in the case of Spitalfields, to 229 from Belleville (see table 4.1). In the archaeological populations, however, not all skeletons retained full dentitions due to ante-mortem tooth loss. This resulted in reduced sample sizes for comparisons using the archaeological sam[ples.

Population	*Sample Size*
Spitalfields	143
Poundbury	186
Belleville	229
Burlington*	207

Table 4.1: Sample Sizes

In the archaeological samples, sub-adult individuals whose dental development ranged from crown formation during the peri-natal period to the completion of the roots of the second molar were included in this study. Informa-

*As explained in section 3.5.2, although the Burlington Growth Study involved a study of 1300 children, these data were longitudinal. In order to make the sample comparable to the archaeological populations, only 207 individuals were used. These were divided equally between all age categories).

tion on the deciduous dentition was not available for the Burlington growth study, since only children over 3 years old participated.

4.2.2: Formation standards

Only lower teeth were examined since radiographs of the mandibular dentition show less distortion than those of the maxillary (Tompkins, 1996). The mandibular dentition was examined using posterior-anterior and lateral radiographs in order to assess stages of dental development for each individual tooth. In cases where the image was not clear enough to determine a developmental stage with confidence, the individual was eliminated from the study.

Differences in the development of isomeres were tested by checking the formation stages of left and right teeth in random samples from all four samples. These samples represented 10% of the total number of individuals in each population. Developmental stages differed in less than 5% of cases for all samples. Furthermore, these differences were not directional. Only the left teeth, therefore, were assessed for the purpose of this study. In instances where the left teeth were missing, or where radiographs were unclear, right teeth were used.

Since this study avoids the use of chronological age, each tooth was merely assigned to a developmental stage with no attempt made to determine age. Eight developmental stages corresponding to those devised by Demirjian and colleagues (1973) were used for this purpose. A description of these stages, and an illustration of how they correspond to those devised by Moorrees and coworkers (1963a) can be found in figures 4.1- 4.4.

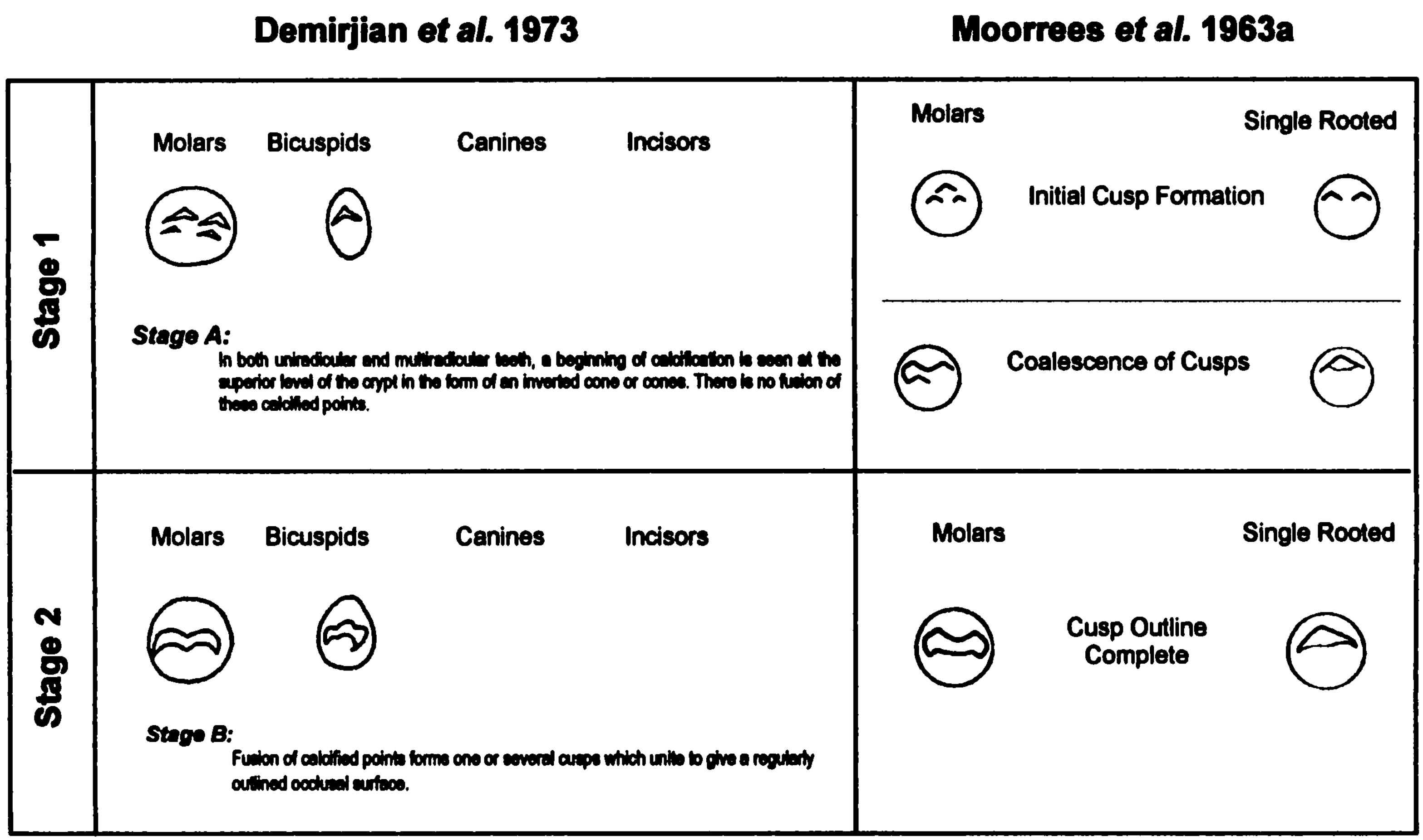


Figure 4.1: Calcification Stages 1 to 2.

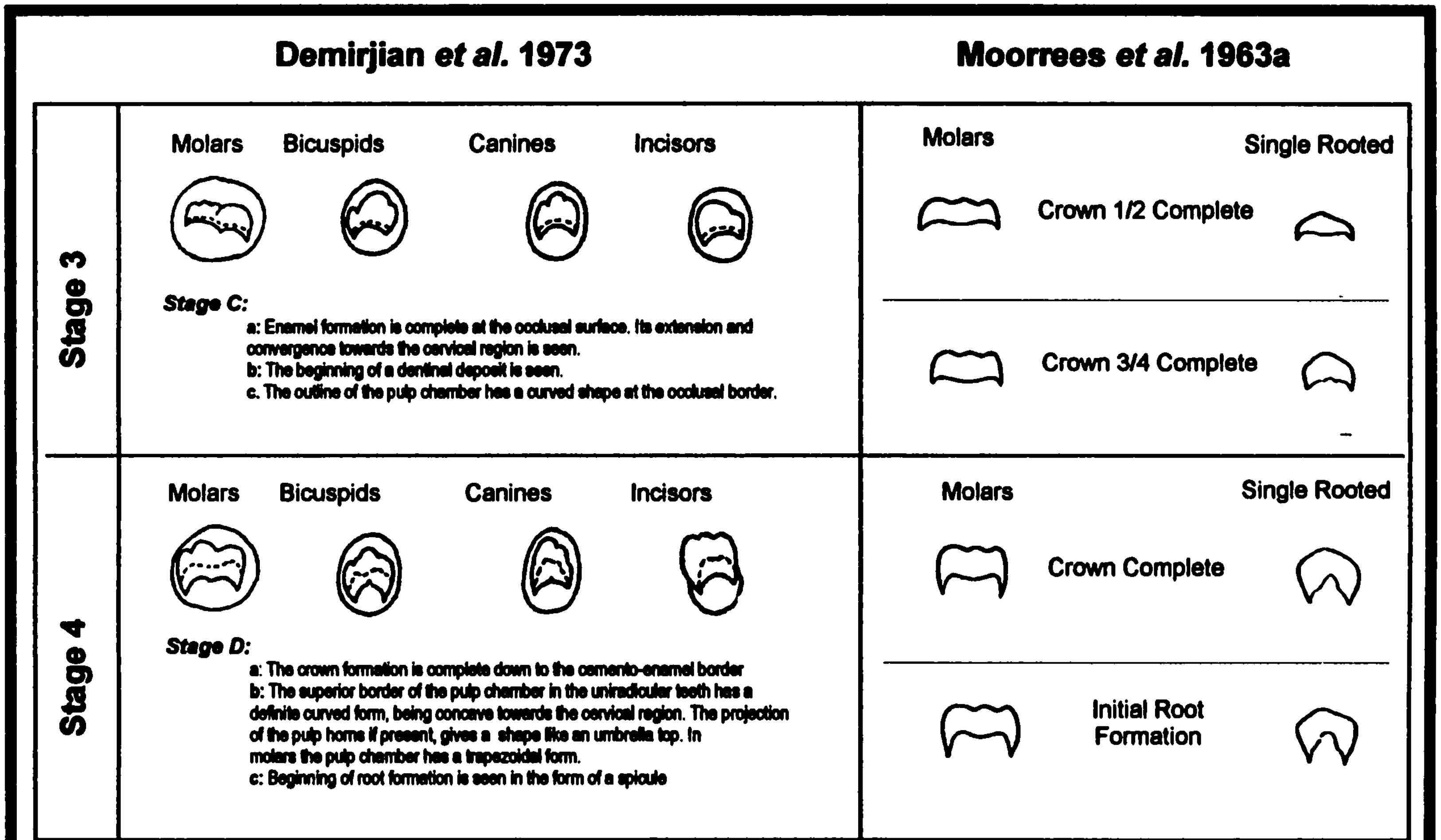


Figure 4.2: Calcification Stages 3 to 4.

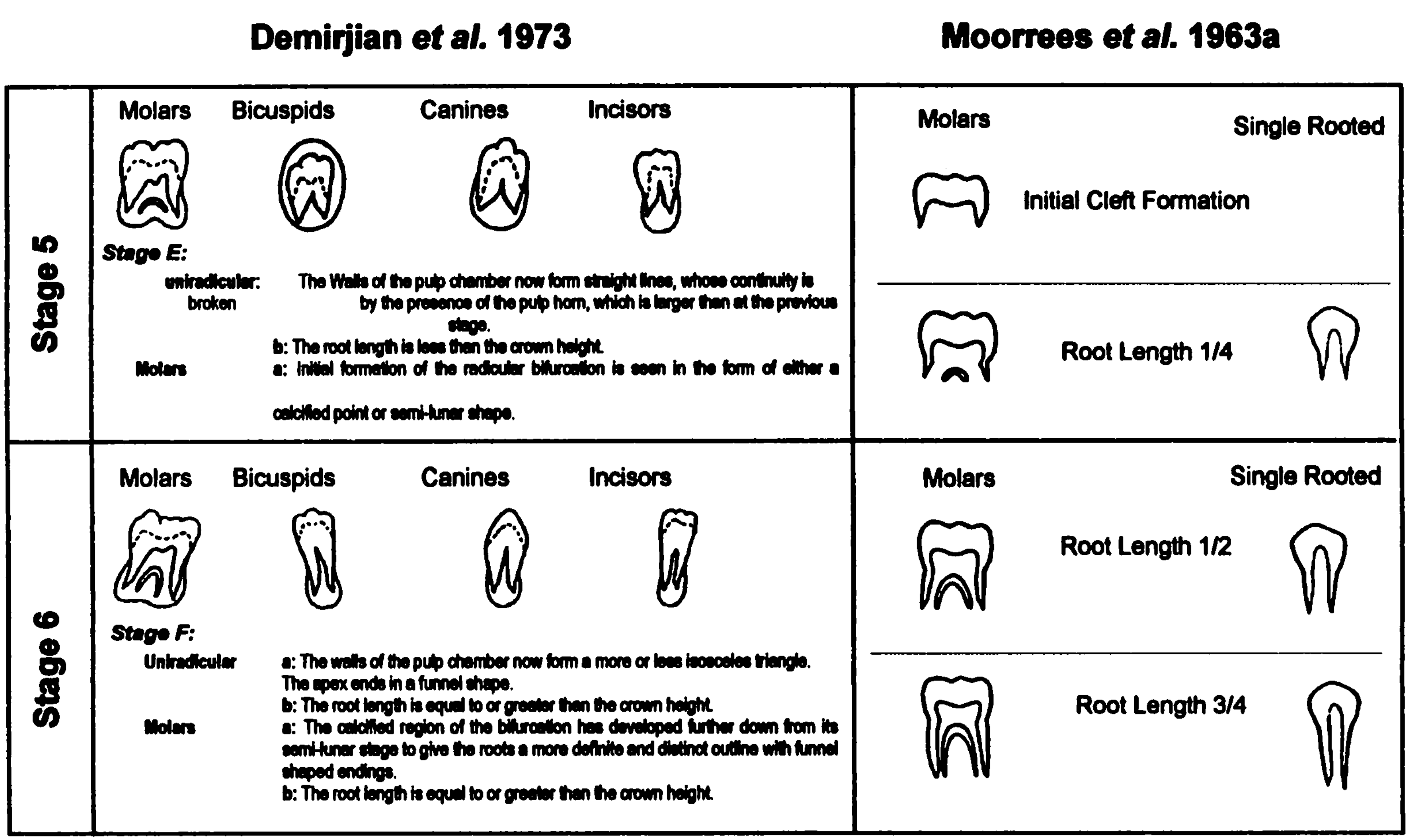


Figure 4.3: Calcification Stages 5 to 6.

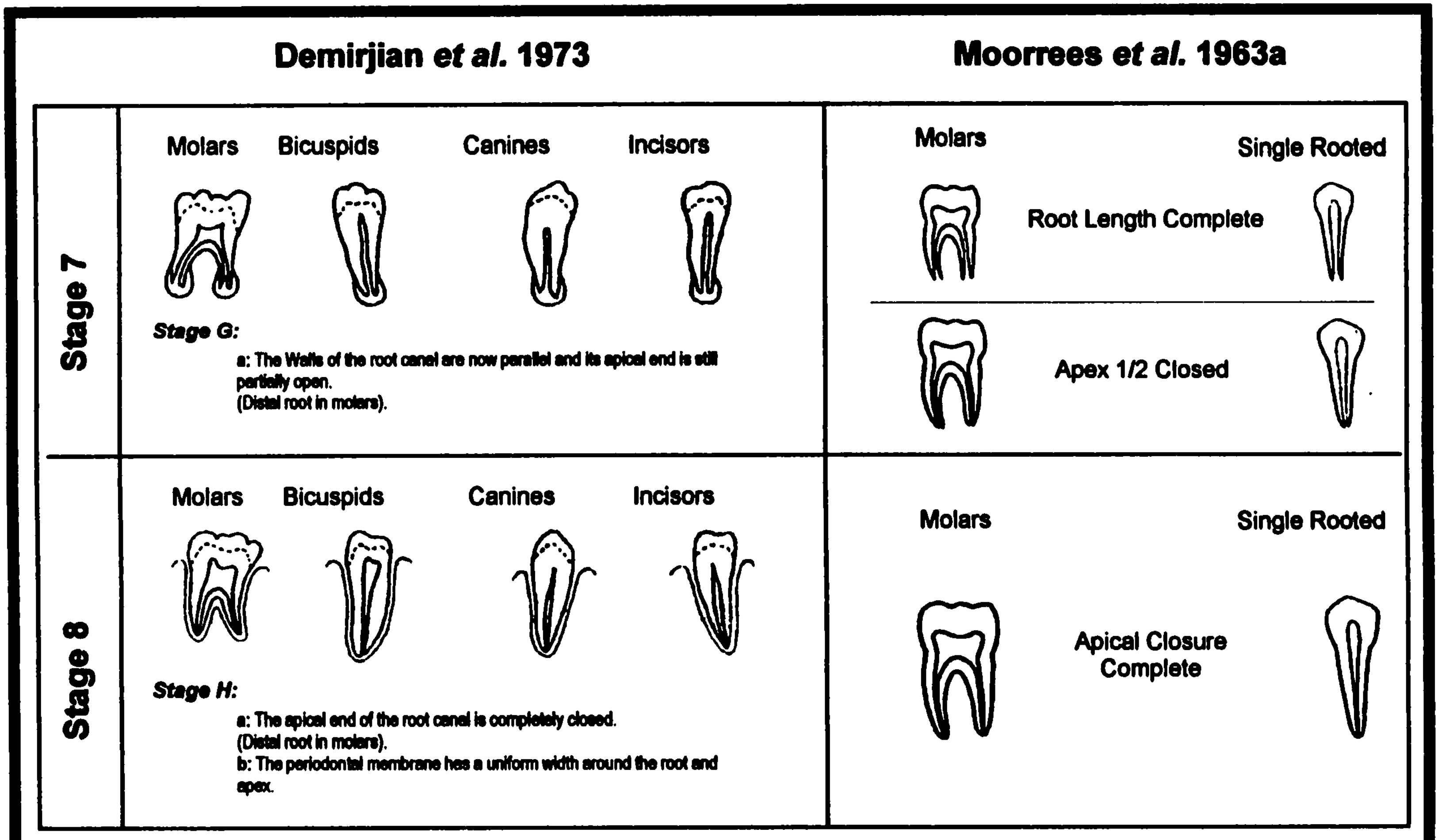


Figure 4.4: Calcification Stages 7 to 8.

4.2.3: Recording

The development of each tooth was observed and then noted on a recording sheet. This information was later entered into an Excel spreadsheet and double checked for data entry errors. The data were then imported into an Access database in order to facilitate cross referencing with the data described in the following chapters.

4.2.4: Intra observer error

The entire dentitions of forty individuals were reassessed three months after the initial data collection. This involved an re-examination of 280 teeth. In 96.4% of cases, there was found to be no difference in the two sets of observations. This was considered an acceptable margin of error, in concurrence with the recommendations of Demirjian and colleagues (1973).

	Stages Identical	One Stage Different	Two Stages Different
Uniradicular Teeth	96.5%	2.5%	1.0%
Molars	96.3%	3.8%	0.0%
Total	96.5%	2.9%	0.7%

Table 4.2: Intra Observer Error

4.2.5: Condensed/expanded index

Initial analysis was designed to give a broad picture of the developmental patterns of each population. Each population was divided into developmental categories since chronological age is unknown for many individuals. Each individual was then assigned a score according to how condensed or expanded their dentition appeared. Put simply, the mandible of an individual with extremely

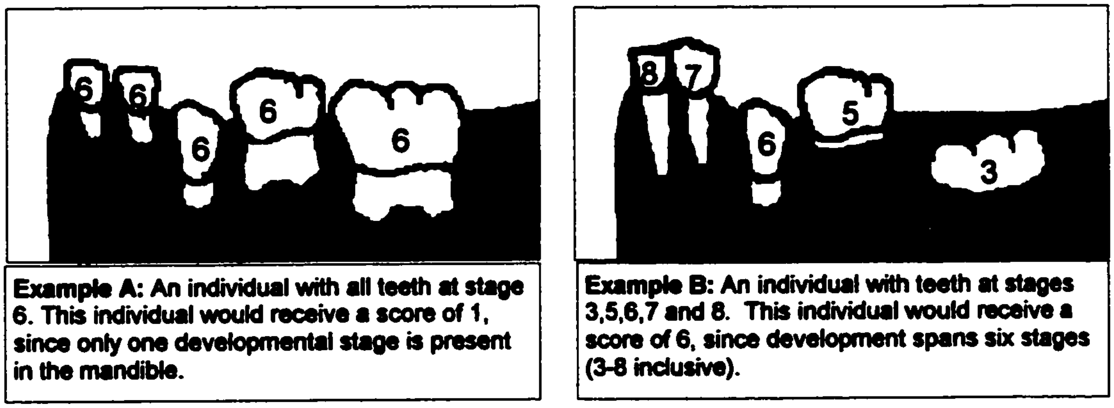


Figure 4.5: Explanation of evaluation of a condensed and an expanded dentition.

densed dental development will exhibit only one developmental stage, since all teeth will be growing in unison. At the other extreme, an individual whose dental development is taking place over a long period of time (expanded) will exhibit 7 developmental stages, since there will be a temporal gap between the develop-

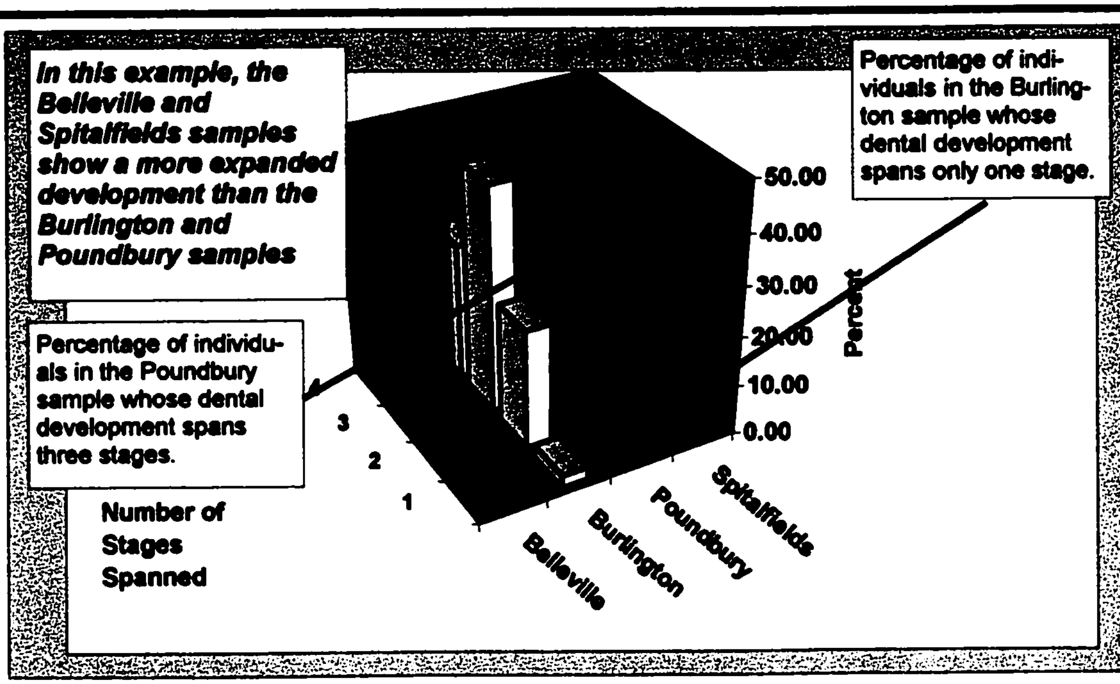


Figure 4.6: Explanation of bar charts used to illustrate comparisons of distributions of CEI.

ment of different teeth within the mandible (see figure 4.5). This is referred to as the condensed/expanded index (CEI). Frequency distributions were then calculated for each population. For ease of visual comparison, bar charts were then constructed based on percentages in order to illustrate similarities and differences between CEI distributions in each population (see figure 4.6).

4.2.6: Paired comparisons

Although the method described above gives a clear picture of the overall differences in *duration* of development, it does not identify which teeth are accounting for the differences. In order to get a more detailed picture of the similarities and differences between the four samples, a modification of the methodology used by Tompkins (1996) was used. Initially, this involves comparing frequency distributions of calcification stages of all deciduous and permanent teeth. Each tooth, in turn, is used as a reference tooth held constant at different stages of development, and compared separately to distributions of all other developing teeth (see figure 4.7). By comparing each tooth at all developmental stages to all the other teeth in turn, a detailed picture of the comparative development of the human dentition can be obtained.

4.2.7: Statistical Analysis

It was decided that, due to the nature of the data, non-parametric statistical methods were most appropriate since such methods do not rely on the assumption that the variables have normal distributions (Colton, 1974). For both sets of analyses (for the condensed/expanded distributions and the more detailed,

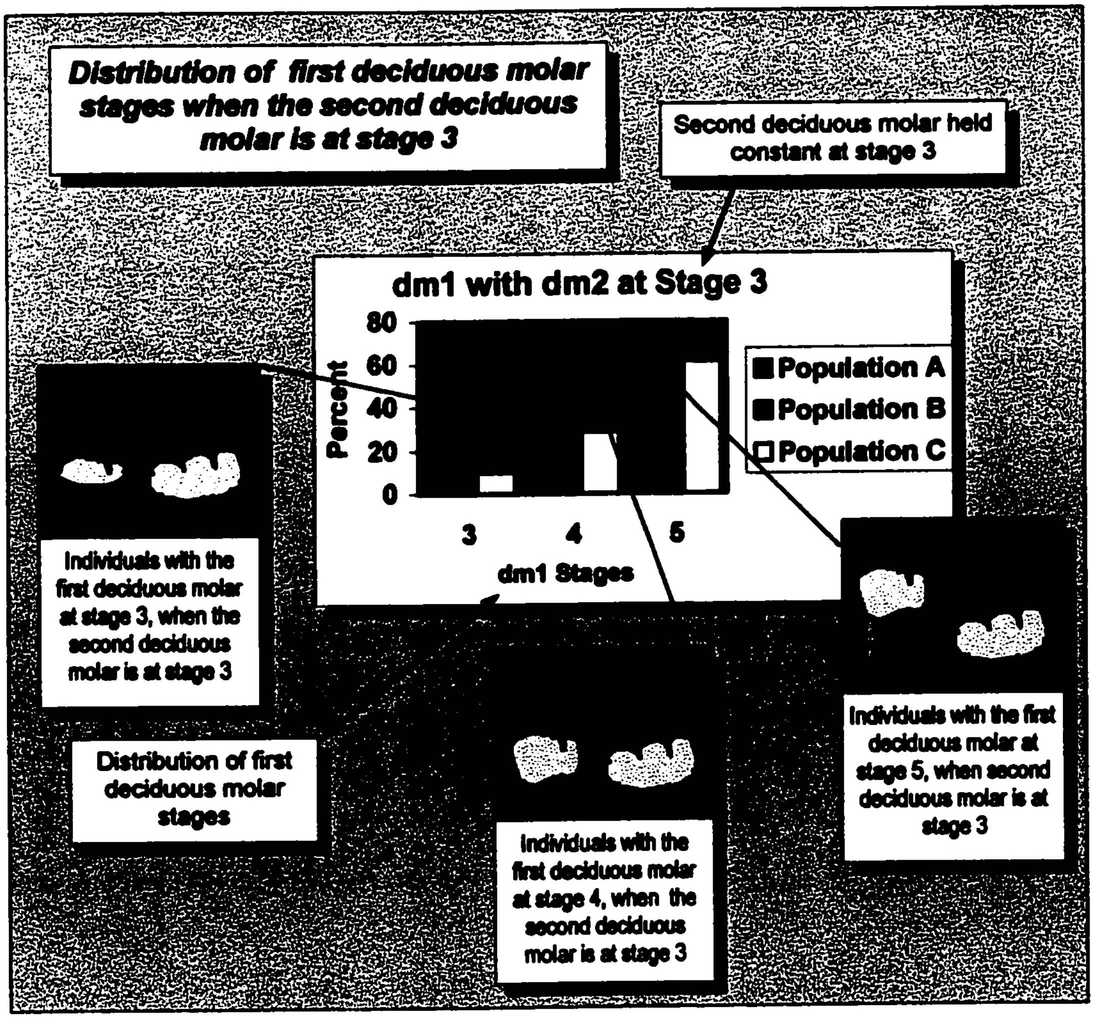


Figure 4.7: Explanation of paired comparisons.

paired comparisons), frequency distributions were initially tested for differences using the Kruskall-Wallis H statistical test. This test is a non-parametric equivalent to the ANOVA, and tests whether several independent samples have the same, underlying distribution (Anderson and Sclove, 1986). Although this test cannot be used to distinguish which or how many of the four samples accounts for the differences, the sheer volume of the tests which needed to be performed necessitated the initial use of this "shorthand" method in order to gain a clear picture of which comparisons required further investigation.

In order to distinguish which samples are accounting for the differences, pair-wise, two-tailed Mann-Whitney U tests were used. The Mann-Whitney U test is the non-parametric equivalent to the *t* test, and is used to determine if two independent samples have the same underlying distribution (Smith, 1991). Like the Kruskall-Wallis H test, it uses the *ranks* of cases. When values are sorted in ascending order, the U statistic is the number of instances in which a value from group A precedes a value from group B. The ranks should be randomly mixed between sample A and sample B, if they are both from the same location. Pair-wise comparisons were only undertaken when the sample size for each group was equal to, or exceeded, 5.

P-values of 0.05 were accepted as significant for both the Kruskall-Wallis H and Mann-Whitney U tests. It is likely, however, that due to the large number of tests carried out in this chapter, significant results occurred by chance (Rice, 1989). In order to minimize this problem, results were regarded on a table-wide, rather than on an individual basis. This was achieved by the use of the sequential Bonferroni test as devised by Holm (1979). This involves dividing the selected significance level (0.05), by the total number of tests in a table (k). If the smallest *P*-value (P_1) is less than or equal to this figure, it can be considered significant. Next, if the second smallest P value (P_2) is smaller than or equal to $0.05/(k\text{-}1)$, it too can be considered significant. This procedure is then repeated for the third *P*-value which must be smaller than or equal to $0.05/(k\text{-}2)$. This procedure is repeated until the *P*-value fails to meet the required level.

4.3: Results

4.3.1: Over view: condensed/expanded index

As described in section 4.2.5, each individual was given a condensed/ expanded score, and then assigned to a category according to developmental stage. This section presents the results of comparisons of the populations for the four developmental categories.

A: Comparison of individuals with development of di1 at stages 3-5

This developmental category comprises the youngest individuals in the samples. At this developmental stage, the development of the first deciduous incisor falls anywhere between stage 3 and stage 5 (figures 4.2-4.3). Although the chronological age of the individuals in these samples is unknown, their ages probably fall within the peri-natal period. (see Steele and Bramblett, 1988). As explained above, children under the age of 3 were excluded from the Burlington growth study, limiting comparisons to Spitalfields, Poundbury and Belleville.

	Spitalfields $^{n=17}$	Poundbury $^{n=13}$	Burlington $^{n=0}$	Belleville $^{n=17}$
Spitalfields	-	0.509	-	0.634
Poundbury	0.509	-	-	0.341
Burlington	-	-	-	-
Belleville	0.634	0.341	-	-

Table 4.3: P-values for Mann-Whitney U tests (comparisons of distributions of CEI for individuals with di1 at stages 3-5)

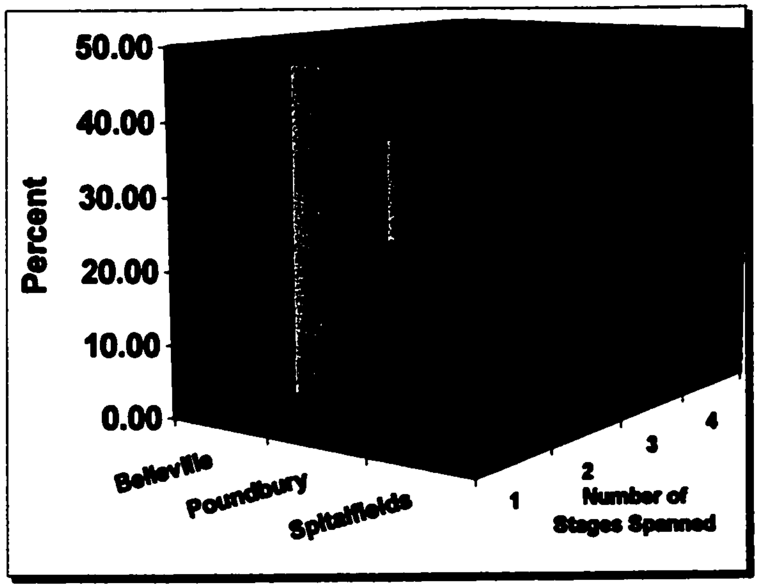


Figure 4.8: Comparison of CEI distributions for individuals with dil development at stages 3-5

Although the dentition of some individuals from the Poundbury sample span only one developmental stage, the mode is 4. The mode for the Belleville and Spitalfields samples are 2 and 3 respectively.

Although the distributions illustrated in figure 4.8 suggest that the deciduous dental development of the Belleville sample follows a more condensed pattern than those of Poundbury and Spitalfields, the differences are not statistically significant (see table 4.3).

B: Comparison of individuals with development of M1 at stages 4-5

Again, comparisons for this developmental stage are limited to those between the archaeological samples: Spitalfields, Poundbury and Belleville. Although chronological ages for the individuals in this category are unknown, the

Table 4.4: P-values for Mann-Whitney U tests (comparisons of distributions of CEI for individuals with M1 at stages 4-5)

	Spitalfields $^{n=11}$	Poundbury $^{n=6}$	Burlington $^{n=0}$	Belleville $^{n=21}$
Spitalfields	-	0.078	-	0.000*
Poundbury	0.078	-	-	0.001*
Burlington	-	-	-	-
Belleville	0.000*	0.001*	-	-

** Significant at the table-wide level*

dental development displayed here probably occurred during the first four years of life, and therefore, in contrast to the previous category, represents early, post-uterine development.

Unlike the differences apparent in the previous category, the differences between the three distributions in this comparison are statistically significant, except for those between Poundbury and Spitalfields (table 4.4). In this instance, the more condensed pattern of development evident in the Belleville sample is significantly different. The expanded pattern of development evident in the Spitalfields sample illustrates that dental development is slower than that of the Belleville sample, since there is a larger developmental gap between the early and later developing teeth. The Poundbury sample falls somewhere between these extremes, and is significantly different from the pattern of development for Belleville, but not from that of Spitalfields.

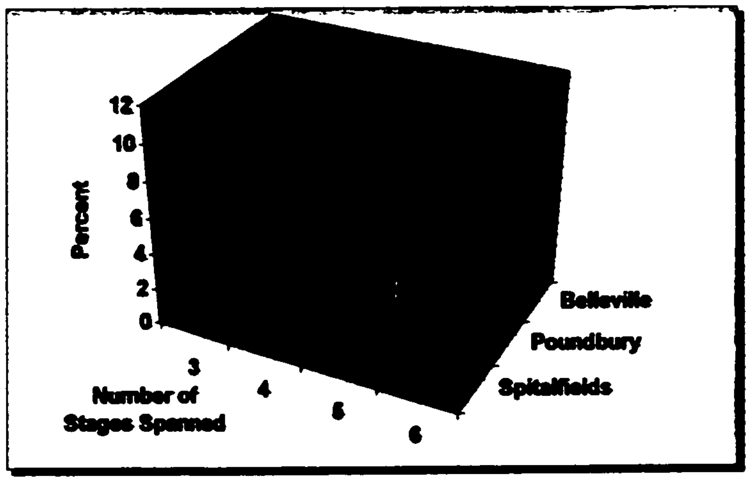


Figure 4.9: Comparison of CEI distributions for individuals with M1 development at stages 4-5

C: Comparison of individuals with development of M1 at stages 6-7

For individuals in this developmental category, the only statistically significant difference at the table wide level, is between Belleville and Burlington. The former shows a more expanded pattern of development than the latter in this instance.

	Spitalfields n=10	Poundbury n=14	Burlington n=73	Belleville n=20
Spitalfields	-	0.036	-	0.530
Poundbury	0.036	-	0.521	0.036
Burlington	0.012	0.521	-	0.008*
Belleville	0.530	0.036	0.008*	-

** significant at the table-wide level*

Table 4.5: P-values for Mann-Whitney U tests (comparisons of distributions of CEI for individuals with M1 at stages 6-7)

It is interesting to note that the Belleville sample, in contrast to the previous two developmental categories, now shows an expanded pattern of development indicating retardation in the dentition of children who died at this developmental stage.

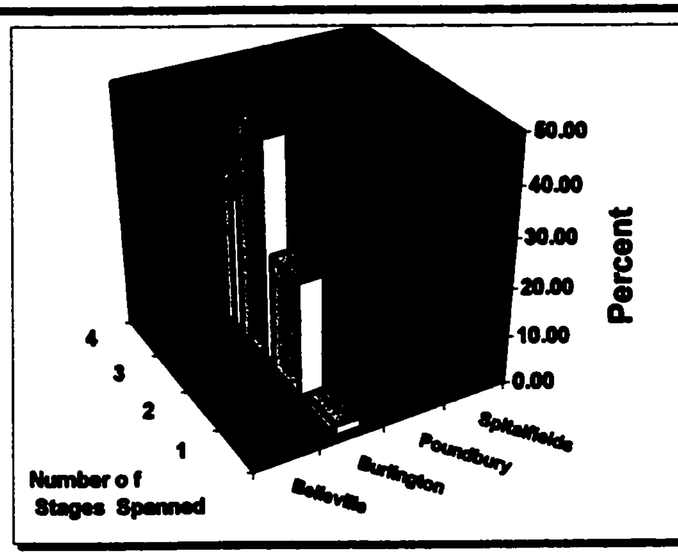


Figure 4.10: Comparison of CEI distributions for individuals with M1 development at stages 6-7

D: Comparison of individuals with development of M2 at stages 6-7

	Spitalfields $^{n=6}$	Poundbury $^{n=21}$	Burlington $^{n=63}$	Belleville $^{n=10}$
Spitalfields	-	0.722	0.914	0.278
Poundbury	0.722	-	0.516	0.306
Burlington	0.914	0.516	-	0.038
Belleville	0.278	0.306	0.038	-

Table 4.6: P-values for Mann-Whitney U tests (comparisons of distributions of CEI for individuals with M1 at stages 4-5)

For this developmental category, no test yielded results which were significant at the table-wide level. By this stage, all the archaeological samples show patterns of development similar to those of the modern Canadian sample (Burlington).

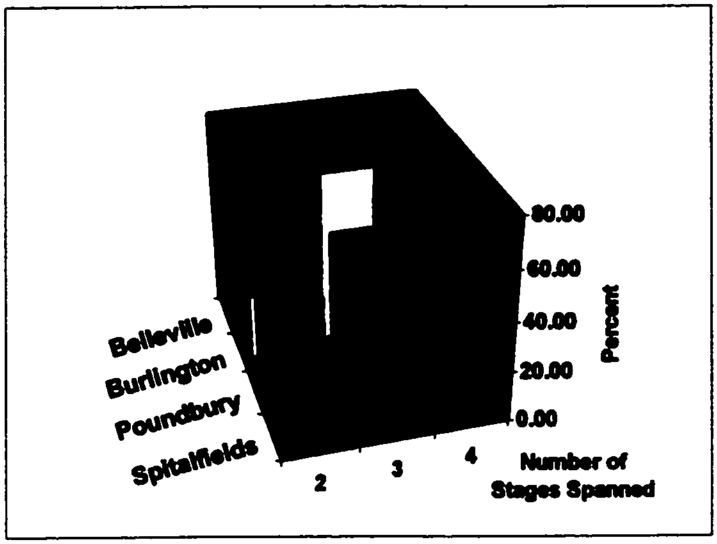


Figure 4.11: Comparison of CEI distributions for individuals with M2 development at stages 6-7

E: Summary

In summary, the developmental pattern of the Belleville sample is condensed, except for the category *M1 at stages 6-7*. The Spitalfields sample shows an expanded pattern of development for all categories. In the latter two developmental categories (M1 at stages 6-7 and M2 at stages 6-7), the Poundbury sample displays a developmental pattern similar to that of Burlington. Unfortunately, the absence of Burlington data for the early categories makes it impossible to assess if this similarity is evident in the early developmental categories, where the developmental pattern of the Poundbury sample falls somewhere between that of the Spitalfields and Belleville samples.

Comparisons of individuals in the oldest and youngest developmental categories do not, however, reach table-wide significance.

4.3.2: Paired comparisons

This section presents the results of the pair-wise comparisons between the four samples for distributions of tooth formation of all deciduous and permanent teeth at all stages of development. Each tooth is in turn used as a reference, and held constant at each stage of development. Since this involved the creation of over 1100 distribution charts with corresponding tables, and over 850 pair-wise comparisons, it is impractical to reproduce all the tables and bar charts in this chapter. As an alternative, all frequency distributions are placed in tabular form in Appendix I.

For ease of discussion, results are grouped by tooth with selected bar charts displayed alongside the text to illustrate similarities and differences in development. Tables of results of Mann-Whitney U two-tailed tests are also reproduced in the body of the text as tables 4.10-4.22. Tables 4.7 and 4.8 show a summary of all *statistically significant* results for the Mann-Whitney U tests at both the individual and table-wide level.

	Spitalfields & Poundbury		Spitalfields & Burlington		Spitalfields & Belleville		Poundbury & Burlington		Poundbury & Belleville		Belleville and Burlington	
	Tests with p=>0.05	No. of tests in table	Tests with p=>0.05	No. of tests in table	Tests with p=>0.05	No. of tests in table	Tests with p=>0.05	No. of tests in table	Tests with p=>0.05	No. of tests in table	Tests with p=>0.05	No. of tests in table
I1	-	4	*C at 3** P4 at 4 M1 at 3 M1 at 4	10	dc at 6 *dm1 at 6** dm1 at 7 dm2 at 6*	20	P3 at 6	10	-	5	I2 at 4 C at 3 C at 4 P4 at 4 M1 at 3 *M1 at 4**	9
I2	-	6	I1 at 5 *P4 at 1** *P4 at 4** M1 at 4	14	*di2 at 7** dc at 6 dm1 at 7 *dm2 at 6**	21	M1 at 7	7	-	6	*C at 4** *P3 at 3** *M1 at 4**	11
C	*I1 at 4** *I2 at 4** M1 at 3	8	*I1 at 5** P3 at 5 P4 at 1	17	*I2 at 4** di2 at 7 dc at 6 *dc at 7** *dm1 at 6** *dm1 at 7** dm2 at 5 *dm2 at 6**	25	I2 at 4 *P3 at 3** M1 at 5	18	I1 at 4*	18	*I2 at 4** *P3 at 3** P4 at 3 *M1 at 5** M2 at 3	22
P3	P4 at 4 *M1 at 4**	10	*I2 at 5** C at 3 C at 6 *M1 at 4** M2 at 6	14	I2 at 4 *M1 at 4**	11	*I1 at 4** I2 at 4 P4 at 4 *P4 at 5** *M1 at 6*	21	-	18	*I1 at 4** *I1 at 6** *I2 at 4** C at 4 C at 6 P4 at 5 *M1 at 6**	19
P4	-	6	*I1 at 5** I1 at 6 *I2 at 5** *I2 at 6** C at 6 *M1 at 6** *M2 at 4**	14	-	5	M1 at 6	13	-	13	*I2 at 6** C at 4 *C at 6** P3 at 4 P3 at 6 M1 at 6	17
M1	I1 at 4 di1 at 6 di2 at 5 di2 at 6 dc at 6 dm1 at 5 dm1 at 6 dm2 at 3	21	I1 at 5* I2 at 5*	11	I2 at 4 C at 5 P3 at 5 di2 at 7 *dc at 6** dc at 7 *dm1 at 6** *dm1 at 7** dm2 at 5 *dm2 at 6** dm2 at 7	28	I2 at 4	20	*C at 5** di1 at 6 dm2 at 4	31	I2 at 4 *C at 5** P3 at 5	19
M2	I1 at 6 P4 at 4	7	-	7	*P3 at 6**	6	I1 at 6 I1 at 7 I2 at 5 I2 at 7 P4 at 5 P4 at 7	21	*C at 6** P3 at 6 P4 at 4 *M1 at 7**	17	C at 4 *C at 6** *P3 at 6** M1 at 7	17

Table 4.7: P-values for Mann-Whitney tests on distribution of all permanent tooth formation relative to all formation stages of other teeth

** significant at table-wide level*

	Spitalfields & Poundbury		Spitalfields & Burlington		Spitalfields & Belleville		Poundbury & Burlington		Poundbury & Belleville		Belleville & Burlington	
	Tests with p=>0.05	No. of tests in table	Tests with p=>0.05	No. of tests in table	Tests with p=>0.05	No. of tests in table	Tests with p=>0.05	No. of tests in table	Tests with p=>0.05	No. of tests in table	Tests with p=>0.05	No. of tests in table
di1	-	15	-	0	M1 at 1	19	-	0	*Dc at 4**	13	-	0
di2	*dc at 4**	12	-	0	di1 at 6	18	-	0	M1 at 3	15	-	0
dc	*M1 at 3** di1 at 5 dm1 at 4 dm2 at 3	15	-	0	I1 at 3 I2 at 3 dm1 at 4 dm1 at 6	24	-	0	M1 at 3	14	-	0
dm1	C at 2 M1 at 3 di1 at 6 dc at 4	17	-	0	I1 at 3 I2 at 3 C at 3 M1 at 2 M1 at 3 di1 at 6 dc at 4	27	-	0	dm2 at 6	17	-	0
dm2	M1 at 3	16	-	0	I2 at 3 *C at 3** M1 at 3 dm1 at 4	25	-	0	di2 at 4 di2 at 6 dm1 at 4	15	-	0

Table 4.8: P-values for Mann-Whitney tests on distribution of all deciduous tooth formation relative to all formation stages of other teeth

** significant at table-wide level*

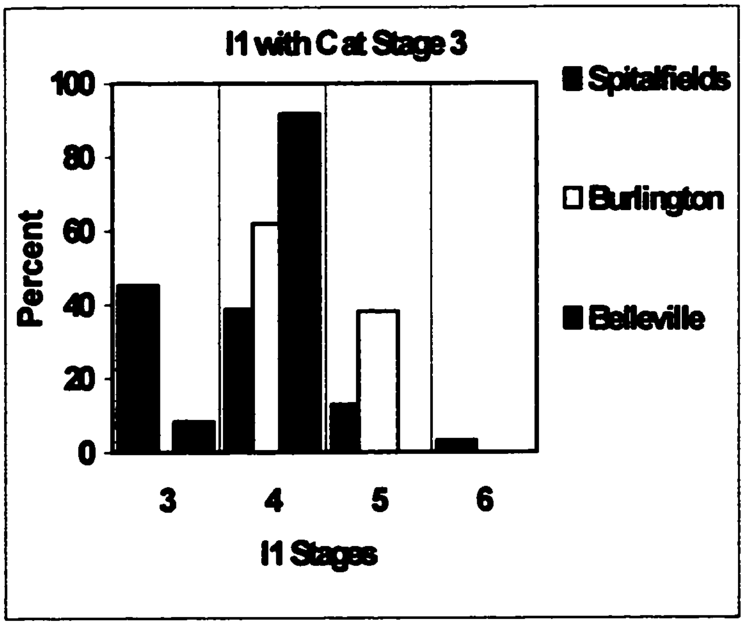


Figure 4.12: Distribution of I1 stages with C held constant as a reference tooth at stage 3

First Permanent Incisor (I1)

The sample size for I1 from Poundbury is small (see Appendix I). This may be responsible for the fact that there are no statistically significant results in the comparisons between Poundbury and all of the other samples for this particular tooth (see tables 4.7 and 4.9).

When the Spitalfields sample is compared to that of Burlington, statistically significant differences at the table-wide level can be observed in the distributions of I1 development, when C is held constant at stage 3 (for example, see figure 4.12).

Comparisons between Belleville and Burlington yield one table-wide significant result. There is a difference in the developmental distribution of I1 when M1 is held constant at stage 4. In both sets of comparisons described above, I1 development in the Burlington sample is advanced over that of both the Belleville and Spitalfields samples.

When comparisons are made between the Spitalfields and Belleville samples, however, a different pattern emerges. While no statistically significant differences are observable in the distribution of I1 formation during the developmental stages described above, differences are evident in comparisons with the deciduous dentition, namely the dm1 and dm2 held constant at stage 6. In these instances, the development of the I1 in the Spitalfields sample is behind that observed in the Belleville sample (figure 4.13).

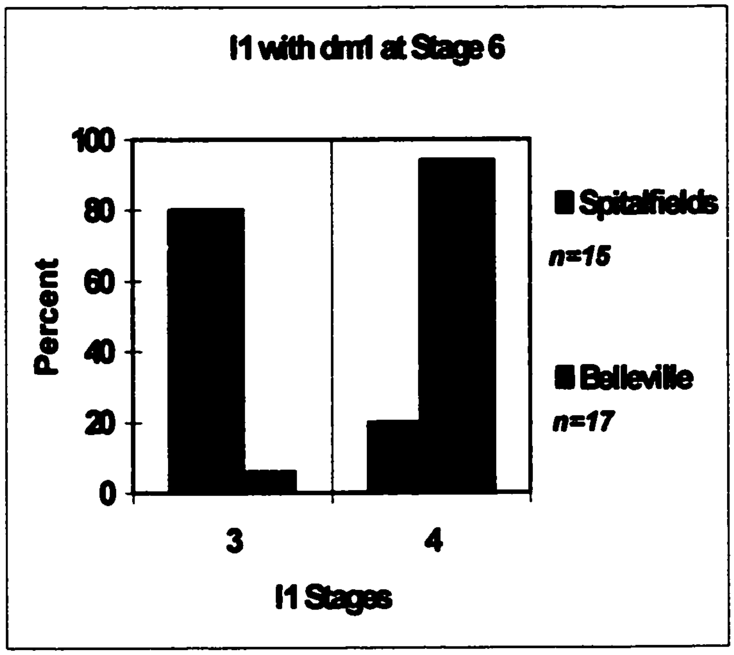


Figure 4.13: Distribution of I1 stages with dm1 held constant as a reference tooth at stage 6

In summary, no significant differences in I1 development are evident between Poundbury and the other samples. The Burlington sample, however, shows development which is advanced over that of the Spitalfields and Belleville samples during the last stages of crown development for the permanent dentition in all tooth classes. In contrast, differences in I1 development between the Belleville and Spitalfields samples occur during the latter stages of deciduous root formation.

Spitalfields and Poundbury: I1 Comparisons

Constants	Stage 1	Stage 2	Stage 3	Stage 4	Stage 5	Stage 6	Stage 7
I2				.820		.368	
C							
P3							
P4							
M1				.222			
M2				.690			
di1							
di2							
dc							
dm1							
dm2							

Spitalfields and Burlington: I1 Comparisons

Constants	Stage 1	Stage 2	Stage 3	Stage 4	Stage 5	Stage 6	Stage 7
I2				.551	.955	.677	
C			.001 *	.839	.959		
P3							
P4				.038			
M1			.024	.029			
M2				.645			
di1							
di2							
dc							
dm1							
dm2							

Spitalfields and Belleville: I1 Comparisons

Constants	Stage 1	Stage 2	Stage 3	Stage 4	Stage 5	Stage 6	Stage 7
I2			.312	.094		.931	
C			.277				
P3							
P4				1.000			
M1			.676	.160			
M2				.429			
di1						.776	.026
di2						1.000	
dc				.820		.008	
dm1				.876	.445	.001 *	.022
dm2				.823	.067	.000 *	

Poundbury and Burlington: I1 Comparisons

Constants	Stage 1	Stage 2	Stage 3	Stage 4	Stage 5	Stage 6	Stage 7
I2				.559		.080	
C			.081	.181	.782		
P3			.069			.016	
P4							
M1				.147		.235	
M2				.172			
di1							
di2							
dc							
dm1							
dm2							

Poundbury and Belleville: I1 Comparisons

Constants	Stage 1	Stage 2	Stage 3	Stage 4	Stage 5	Stage 6	Stage 7
I2				.565		.310	
C				.929			
P3			.958				
P4							
M1							
M2				.082			
di1							
di2							
dc							
dm1							
dm2							

Belleville and Burlington: I1 Comparisons

Constants	Stage 1	Stage 2	Stage 3	Stage 4	Stage 5	Stage 6	Stage 7
I2			.073	.025		.866	
C			.040	.039			
P3							
P4				.038			
M1			.043	.008*			
M2				.481			
di1							
di2							
dc							
dm1							
dm2							

Table 4.9:* P-*values for Mann-Whitney tests on distributions of I1 formation, relative to all formation stages of other teeth.

** significant at table-wide level*

Second Permanent Incisor (I2)

The results of this set of comparisons follow a similar pattern to those of the I1. Comparisons involving Poundbury yield no statistically significant results at the table-wide level. Comparisons between the other three samples produce significant differences, with table-wide level results occurring in higher numbers than for the I1. Again, Spitalfields and Belleville show later development of the I2 in comparison to Burlington. Comparisons between the Spitalfields and Burlington samples show differences occurring in I2 development when P4 is held constant at stages 1 and 4. In comparisons between the Belleville and Burlington samples, the differences are evident when C is held constant at stage 4, P3 at stage 3, and M1 at stage 4.

Perhaps the most striking differences occur in comparisons involving the deciduous dentition as reference teeth. Comparisons between distributions of I2 development in relation to di2 and dm2 reveal that Belleville shows a develop-

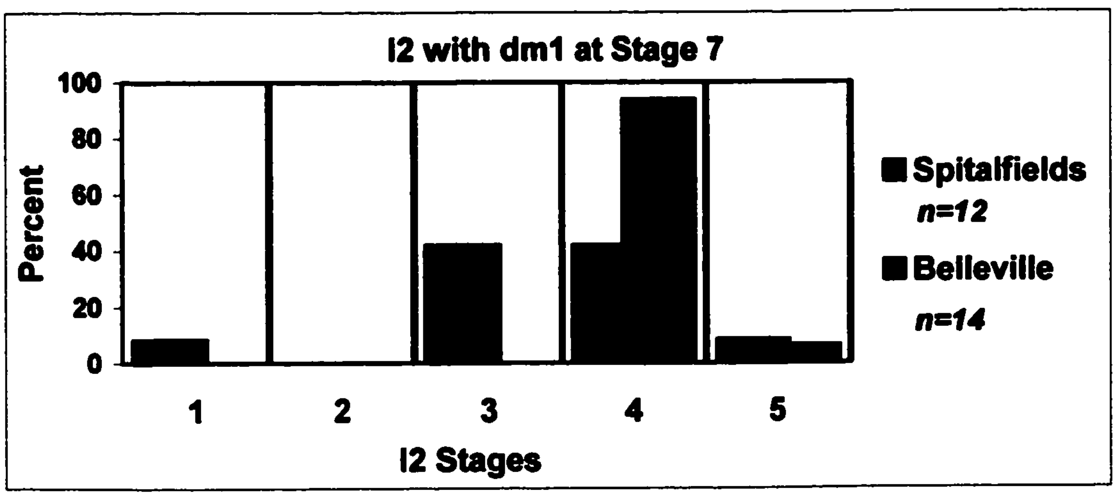


Figure 4.14: Distribution of I2 stages with dm1 held constant as a reference tooth at stage 7

Spitalfields and Poundbury: I2 Comparisons:

Constants	Stage 1	Stage 2	Stage 3	Stage 4	Stage 5	Stage 6	Stage 7
I1				.829			
C				.454			
P3		.368					
P4							
M1				.393		.864	
M2				1.000			
di1							
di2							
dc							
dm1							
dm2							

Spitalfields and Burlington: I2 Comparisons

Constants	Stage 1	Stage 2	Stage 3	Stage 4	Stage 5	Stage 6	Stage 7
I1				.352	***.014***	.316	
C			.341		.598	.353	
P3		.054			.401		
P4	***.002****			***.006****			
M1			.741	***.043***		.210	
M2			-	.483			
di1							
di2							
dc							
dm1							
dm2							

Spitalfields and Belleville: I2 Comparisons

Constants	Stage 1	Stage 2	Stage 3	Stage 4	Stage 5	Stage 6	Stage 7
I1			.235	.989	.792		
C			.620			.681	
P3							
P4							
M1			.103	.214		.699	
M2				.345			
di1							.076
di2						.549	***.000****
dc				.635		***.019***	
dm1					.699	.088	***.047***
dm2				.553	.259	***.000****	.072

Poundbury and Burlington: I2 Comparisons

Constants	Stage 1	Stage 2	Stage 3	Stage 4	Stage 5	Stage 6	Stage 7
I1				.336			
C							
P3				.291			
P4							
M1				.715	.514	.177	***.036***
M2				.503			
di1							
di2							
dc							
dm1							
dm2							

Poundbury and Belleville: I2 Comparisons

Constants	Stage 1	Stage 2	Stage 3	Stage 4	Stage 5	Stage 6	Stage 7
I1				.781			
C				.697			
P3				.731			
P4							
M1				.604		.776	
M2				.282			
di1							
di2							
dc							
dm1							
dm2							

Belleville and Burlington: I2 Comparisons

Constants	Stage 1	Stage 2	Stage 3	Stage 4	Stage 5	Stage 6	Stage 7
I1				.292		.753	
C				***.003****			
P3			***.001****	.142		.245	
P4				***.002****			
M1			.101	.077		.644	
M2				.647			
di1							
di2							
dc							
dm1							
dm2							

Table 4.10: P-values for Mann-Whitney tests on distributions of I2 formation, relative to all formation stages of other teeth.

** significant at table-wide level*

mental distribution advanced over that of Spitalfields (see table 4.10). In common with the I1 comparisons, these differences occur during the later stages of deciduous root formation. Again, no significant differences are evident in comparisons with other permanent teeth.

Permanent Canine (C)

In this set of comparisons, differences involving the Poundbury sample reach levels of table-wide significance. Comparisons between the Belleville and Poundbury samples reveal a difference in the distributions of C development when the I1 is held constant at stage 4 (figure 4.15). Differences of significance at the table-wide level also occur between Poundbury and Burlington when P3 is held constant at stage 3, and between Poundbury and Spitalfields when I1 and I2 are held constant at stage 4.

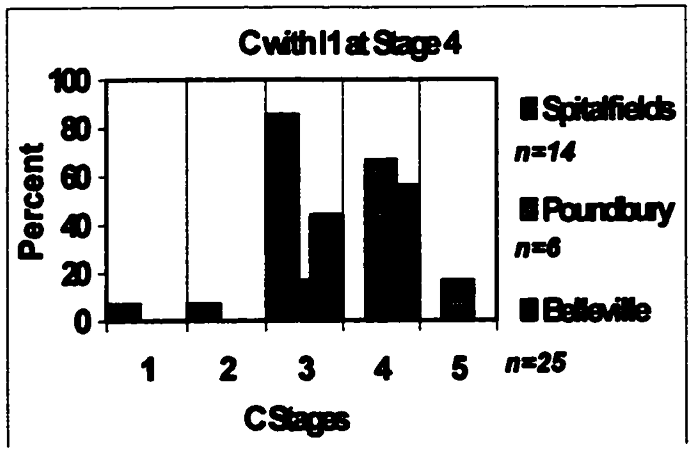


Figure 4.15: Distribution of C stages with I1 held constant as a reference tooth at stage 4

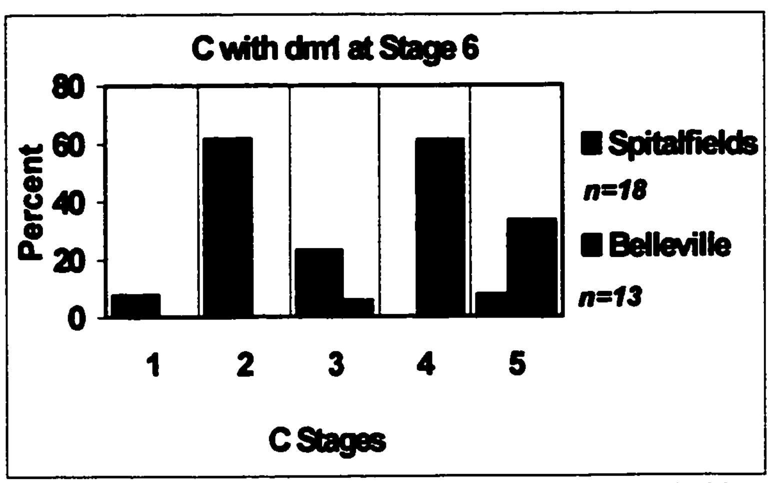


Figure 4.16: Distribution of C stages with dm1 held constant as a reference tooth at stage 6

Spitalfields and Poundbury: C Comparisons

Constants	Stage 1	Stage 2	Stage 3	Stage 4	Stage 5	Stage 6	Stage 7
I1				*.002* *		.833	
I2				*.004* *		.573	
P3							
P4						.247	
M1			*.011*	.132		.285	
M2							
di1							
di2							
dc							
dm1							
dm2							

Spitalfields and Burlington: C Comparisons

Constants	Stage 1	Stage 2	Stage 3	Stage 4	Stage 5	Stage 6	Stage 7
I1					*.001* *	.900	
I2			.246	.790		.858	
P3		.429			*.014*	.607	
P4	*.045*		.513	.104		.976	
M1			.135	.269		.454	
M2			.467	.256			
di1							
di2							
dc							
dm1							
dm2							

Spitalfields and Belleville: C Comparisons

Constants	Stage 1	Stage 2	Stage 3	Stage 4	Stage 5	Stage 6	Stage 7
I1				.376		.660	
I2			.925	*.000* *	.950	.864	
P3						1.000	
P4				.391		.222	
M1			.125	.391		.222	
M2				.745			
di1							.133
di2						.129	*.000*
dc						*.013*	*.002* *
dm1					.093	*.002* *	*.000* *
dm2				.296	*.007*	*.000* *	.154

Poundbury and Burlington: C Comparisons

Constants	Stage 1	Stage 2	Stage 3	Stage 4	Stage 5	Stage 6	Stage 7
I1						.549	
I2				*.006*		.610	.107
P3			*.001* *			.440	.577
P4			.531		.135	.074	
M1			.055	.560	*.038*	.721	.090
M2			.283		.915	.401	
di1							
di2							
dc							
dm1							
dm2							

Poundbury and Belleville: C Comparisons

Constants	Stage 1	Stage 2	Stage 3	Stage 4	Stage 5	Stage 6	Stage 7
I1				*.005* *		.950	
I2				.814		.734	
P3			.376	.711		.968	.383
P4			.210		1.000	.950	
M1			.118	.569	*.766*	.893	.555
M2			.492		.445		
di1							
di2							
dc							
dm1							
dm2							

Belleville and Burlington: C Comparisons

Constants	Stage 1	Stage 2	Stage 3	Stage 4	Stage 5	Stage 6	Stage 7
I1						.195	.548
I2				*.000* *		.978	
P3			*.000* *	.117	.090	.203	.081
P4			*.004*	.742	.135	.064	.085
M1			1.000	.922	*.002* *	.400	.563
M2			*.031*	.273	.345		
di1							
di2							
dc							
dm1							
dm2							

***Table 4.11:* P-values for Mann-Whitney tests on distributions of C formation, relative to all formation stages of other teeth.**

** significant at table-wide level*

Differences between Spitalfields and Burlington reach table-wide significance when I2 is held constant at stage 4, P3 at stage 3, and M1 at stage 1.

In all comparisons of C development there is a tendency for development in the Poundbury sample to show advancement over the other samples, and for Spitalfields to show retardation. The Belleville and Burlington samples fall somewhere between these two extremes. Comparisons of C development, when the deciduous dentition are used as reference teeth, continue the trend described for both permanent incisors (figure 4.16).

Third Permanent Premolar (P3)

The development of the P3 for the Poundbury sample proves significantly different at the table-wide level from that of the Spitalfields and Burlington samples, but not from that of the Belleville sample (see table 4.12). In comparisons between the Poundbury and Spitalfields samples, this difference is apparent when M1 is held constant at stage 4. Comparisons between the Poundbury and Burlington samples show significant differences occurring when I1 is held constant at stage 4, and P4 is held constant at stage 6.

Comparisons between the other three samples follow a similar pattern (see table 4.12). Table-wide significant differences occur between the Spitalfields and Burlington samples (I2 at stage 5 and M1 at stage 4), between the Spitalfields and Belleville samples (M1 at stage 4) and between the Burlington and Belleville samples (I1 at stages 4 and 6, I 2 at stage 4, and M1 at stage 6).

Spitalfields and Poundbury: P3 Comparisons:

Constants	Stage 1	Stage 2	Stage 3	Stage 4	Stage 5	Stage 6	Stage 7
I1						.222	
I2				.234		.950	
C				-		.118	
P4				_.032_		.121	
M1				_.000_ *		.482	
M2				.699		.648	
di1							
di2							
dc							
dm1							
dm2							

Spitalfields and Burlington: P3 Comparisons

Constants	Stage 1	Stage 2	Stage 3	Stage 4	Stage 5	Stage 6	Stage 7
I1					.327	.429	
I2				.710	_.000_ *	.203	
C			_.011_			_.016_	
P4	.482			.959		.545	
M1				_.000_ *		.168	
M2				.384		_.033_	
di1							
di2							
dc							
dm1							
dm2							

Spitalfields and Belleville: P3 Comparisons

Constants	Stage 1	Stage 2	Stage 3	Stage 4	Stage 5	Stage 6	Stage 7
I1				.121		.121	.669
I2				_.043_		.864	
C						.942	
P4				.289		.628	
M1				_.001_ *		.267	
M2				.378			
di1							
di2							
dc							
dm1							
dm2							

Poundbury and Burlington: P3 Comparisons

Constants	Stage 1	Stage 2	Stage 3	Stage 4	Stage 5	Stage 6	Stage 7
I1				_.000_ *		.052	.637
I2				_.045_		.529	.293
C				.487	.083	.950	
P4			.511	_.014_	_.000_ *	.156	
M1				.142	.523	_.011_	.058
M2			.560	.180	.876	.642	
di1							
di2							
dc							
dm1							
dm2							

Poundbury and Belleville: P3 Comparisons:

Constants	Stage 1	Stage 2	Stage 3	Stage 4	Stage 5	Stage 6	Stage 7
I1				.228		.374	.662
I2				.492		.888	
C				.409		.133	
P4			.739	.246	.371	.414	
M1				.414	1.000	.792	.107
M2			.971	.295	.918		
di1							
di2							
dc							
dm1							
dm2							

Belleville and Burlington: P3 Comparisons

Constants	Stage 1	Stage 2	Stage 3	Stage 4	Stage 5	Stage 6	Stage 7
I1				_.001_ *		_.001_ *	.915
I2				_.000_ *		.152	
C				_.040_		_.014_	
P4			.801	.168	_.021_	.898	
M1				.578	.543	_.001_ *	.850
M2			.687	.700	.793		.868
di1							
di2							
dc							
dm1							
dm2							

Table 4.12: P-values for Mann-Whitney tests on distributions of P3 formation, relative to all formation stages of other teeth.

* significant at table-wide level

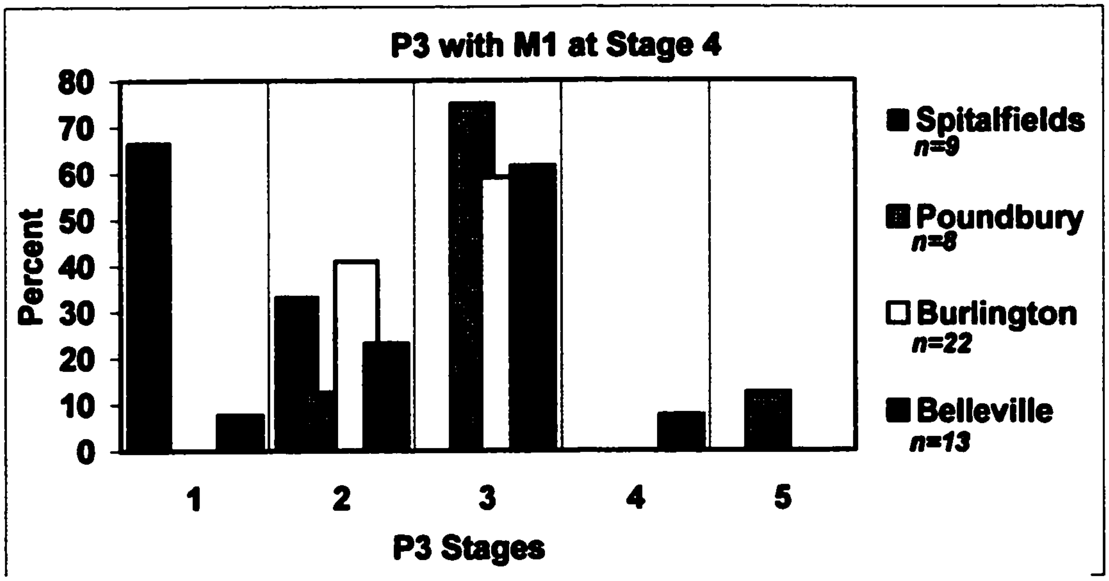


Figure 4.17: Distribution of P3 stages with M1 held constant as a reference tooth at stage 4

Figure 4.17 shows the development of the P3 in the Spitalfields sample falling behind that of the other three samples. This chart illustrates a trend that is typical for comparisons of third premolar development in this study.

Fourth Permanent Premolar (P4)

Comparisons of distributions of P4 formation follow a different pattern, with the only table-wide significant differences occurring between Burlington and the other samples. Differences between the archaeological samples are not statistically significant. In all comparisons involving Burlington, the other samples showed comparatively retarded development.

Comparisons between Burlington and Poundbury produce one significant result at the individual level; when the M1 is used as a reference tooth at

Spitalfields and Poundbury: P4 Comparisons

Constants	Stage 1	Stage 2	Stage 3	Stage 4	Stage 5	Stage 6	Stage 7
I1						.222	
I2						.177	
C						.603	
P3						.059	
M1						.930	
M2						.445	
di1							
di2							
dc							
dm1							
dm2							

Spitalfields and Burlington: P4 Comparisons

Constants	Stage 1	Stage 2	Stage 3	Stage 4	Stage 5	Stage 6	Stage 7
I1					.000 *	.028	
I2				.160	.000 *	.001 *	
C					.733	.012	
P3		.633	.755			.408	
M1				.208		.008 *	
M2				.008 *		.052	
di1							
di2							
dc							
dm1							
dm2							

Spitalfields and Belleville: P4 Comparisons

Constants	Stage 1	Stage 2	Stage 3	Stage 4	Stage 5	Stage 6	Stage 7
I1							
I2							
C						.687	
P3						.056	
M1						.699	
M2				.176		.412	
di1							
di2							
dc							
dm1							
dm2							

Poundbury and Burlington: P4 Comparisons

Constants	Stage 1	Stage 2	Stage 3	Stage 4	Stage 5	Stage 6	Stage 7
I1						.569	
I2						.130	
C				.529	.650	.116	
P3				.291			.569
M1					.253	.003	.578
M2			.498		.654	.441	
di1							
di2							
dc							
dm1							
dm2							

Poundbury and Belleville: P4 Comparisons

Constants	Stage 1	Stage 2	Stage 3	Stage 4	Stage 5	Stage 6	Stage 7
I1							
I2						.343	
C				.368	.710	.250	.216
P3				.231		.897	.497
M1					.536	.776	.303
M2			.791		.699		
di1							
di2							
dc							
dm1							
dm2							

Belleville and Burlington: P4 Comparisons

Constants	Stage 1	Stage 2	Stage 3	Stage 4	Stage 5	Stage 6	Stage 7
I1							.128
I2						.004 *	
C				.036	.938	.000 *	.925
P3			.457	.013	.133	.021	.702
M1					.714	.002 *	.319
M2			.825	.240	.356		
di1							
di2							
dc							
dm1							
dm2							

Table 4.13:* P*-values for Mann-Whitney tests on distributions of P4 formation, relative to all formation stages of other teeth.

** significant at table-wide level*

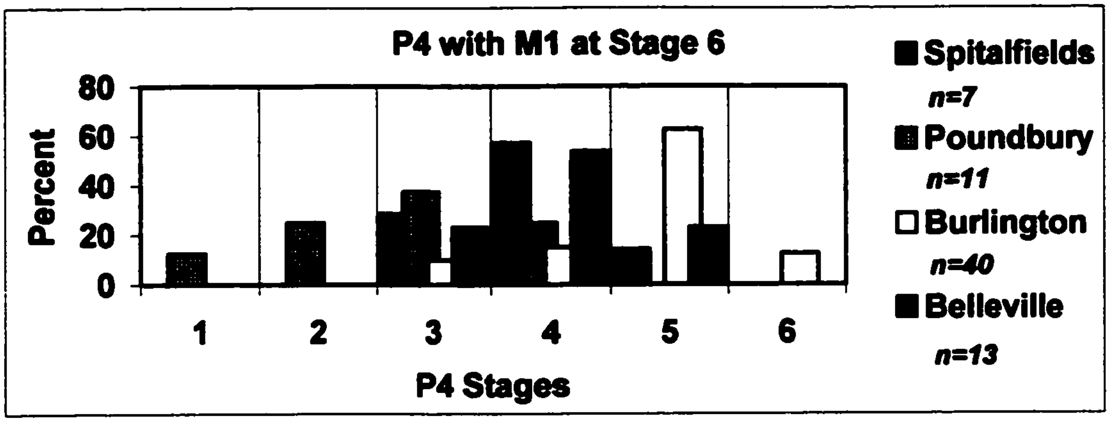


Figure 4.18: Distribution of P4 stages with M1 held constant as a reference tooth at stage 6

stage 6. This is not significant, however, at the table-wide level. The Burlington sample, does however, show significant advancement over all the other samples at the table-wide level (for example, see figure 4.18).

First Permanent Molar (M1)

Table-wide significant differences in the distributions of M1 development between Belleville and the other samples are evident in only two cases. There is a difference between the Poundbury and Belleville samples, and between the Burlington and Belleville samples when C is held constant at stage 5.

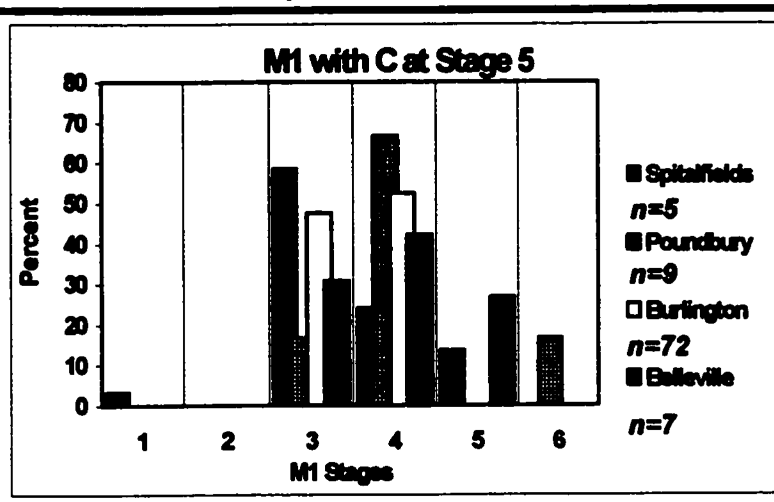


Figure 4.19: Distribution of M1 stages with C held constant as a reference tooth at stage 5

Spitalfields and Poundbury: M1 Comparisons

Constants	Stage 1	Stage 2	Stage 3	Stage 4	Stage 5	Stage 6	Stage 7
I1						.317	
I2			.116	*.020*		.827	
C			.102		.606	.250	
P3							
P4				.931			
M2			.296				
di1						*.010*	
di2					*.036*	*.038*	
dc					.394	*.013*	
dm1					*.008*	*.006*	
dm2			.488	*.011*	.966	*.038*	.127

Spitalfields and Burlington: M1 Comparisons

Constants	Stage 1	Stage 2	Stage 3	Stage 4	Stage 5	Stage 6	Stage 7
I1					*.001* *		
I2				.305	*.000* *	.553	
C			.552	.188	.674	.169	
P3		.836	.617		.944		
P4							
M2							
di1							
di2							
dc							
dm1							
dm2							

Spitalfields and Belleville: M1 Comparisons

Constants	Stage 1	Stage 2	Stage 3	Stage 4	Stage 5	Stage 6	Stage 7
I1						.931	
I2			.856	*.003*		.755	
C			.385		*.011*	.381	
P3					*.035*		
P4				.234			
M2			.461				
di1						.870	
di2				.628	.112	.206	*.010*
dc				.338	.792	*.001* *	*.028*
dm1				.579	.133	*.000* *	*.000* *
dm2			.911	.339	*.008*	*.002* *	*.040*

Poundbury and Burlington: M1 Comparisons

Constants	Stage 1	Stage 2	Stage 3	Stage 4	Stage 5	Stage 6	Stage 7
I1					.466	.761	.858
I2				*.036*		.281	
C			.157	.336	.574	.815	
P3			.961	.123		.928	.591
P4			.773	.964	.586	.445	
M2			.803	.209	.862		
di1							
di2							
dc							
dm1							
dm2							

Poundbury and Belleville: M1 Comparisons:

Constants	Stage 1	Stage 2	Stage 3	Stage 4	Stage 5	Stage 6	Stage 7
I1						.441	1.000
I2			.268	.524		.479	
C			.221	.191	*.002* *	.614	
P3			.544	.125		1.000	
P4			.750	.199	.829		
M2			.750	.199	.529		
di1						*.005*	.328
di2					.537	.207	
dc				.525	.662	.562	
dm1					.318	.528	
dm2				*.040*	.050	.872	.649

Belleville and Burlington: M1 Comparisons

Constants	Stage 1	Stage 2	Stage 3	Stage 4	Stage 5	Stage 6	Stage 7
I1						.371	.755
I2				*.008*		.945	
C			.768	.988	*.000* *	.531	
P3			.451	.082	*.008*	.873	
P4			.511	.060	.244	.483	
M2			.621	.645	.616		
di1							
di2							
dc							
dm1							
dm2							

Table 4.14:* P-*values for Mann-Whitney tests on distributions of M1 formation, relative to all formation stages of other teeth.

** significant at table-wide level*

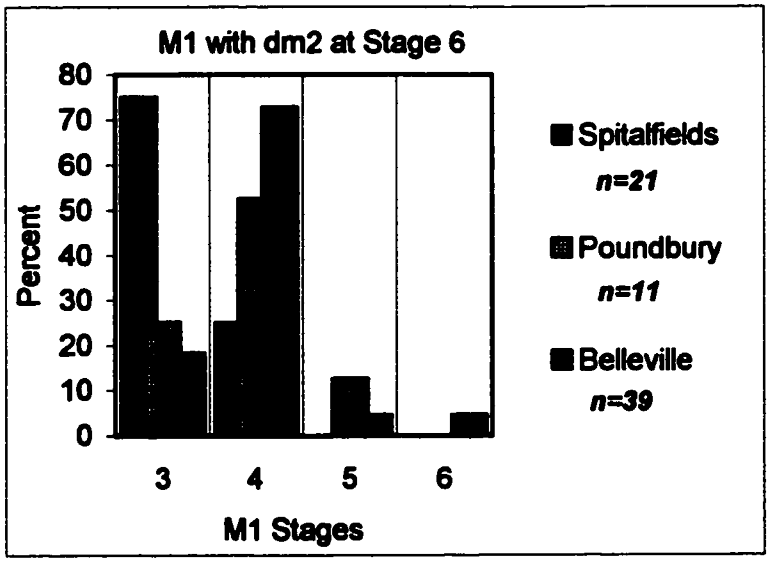


Figure 4.20: Distribution of M1 stages with dm2 held constant as a reference tooth at stage 6

(See figure 4.19). In this set of comparisons, Poundbury is slightly advanced, while Burlington is slightly retarded. The Belleville sample lies somewhere between these two extremes. (Differences involving the Spitalfields sample are not statistically significant in this case).

This trend is also evident in comparisons between the other samples, but with differences involving Spitalfields also reaching levels of table-wide significance. M1 development in the Spitalfields sample appears to be falling behind that of the other samples, with the Belleville and Poundbury samples displaying slight advancement. When M1 development is examined using the deciduous dentition as reference teeth, table-wide significant differences are also apparent. The differences between the Spitalfields and Belleville samples are most striking, with the latter showing consistent advancement over the former. M1 development in the Poundbury sample appears to fall somewhere between these two extremes (figure 4.20).

Second Permanent Molar (M2)

Table-wide significant differences in the distributions of M2 development are evident in comparisons between the Poundbury and Belleville samples; when C is held constant at stage 6 (figure 4.21) and when M1 is held constant at stage 7.

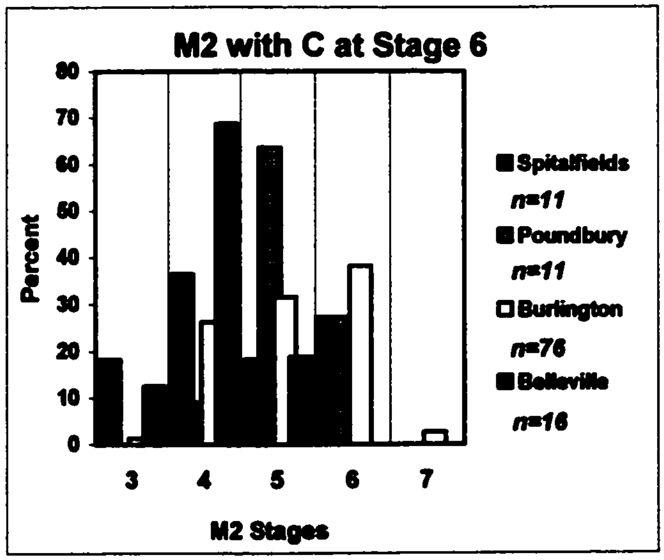


Figure 4.21: Distribution of M2 stages with C held constant as a reference tooth at stage 6

Differences between the Poundbury and Burlington samples and between the Poundbury and Spitalfields samples, do not reach table-wide significance. Furthermore, none of the observable differences between the Spitalfields and Burlington samples are statistically significant at the table-wide level.

In contrast, comparisons between the Spitalfields and Belleville samples produce one significant result; Spitalfields is advanced over Belleville when P3 is held constant at stage 6. Differences between the Belleville and Burlington samples are significant when P3 and P4 are held constant at stage 6 (see figure 4.22) and M1 at stage 7. This set of comparisons of M2 development shows the Burlington sample advanced over that of Belleville.

Spitalfields and Poundbury: M2 Comparisons

Constants	Stage 1	Stage 2	Stage 3	Stage 4	Stage 5	Stage 6	Stage 7
I1						*.048*	
I2						.541	
C						.331	
P3						.098	
P4				*.026*		.541	
M1						.717	
di1							
di2							
dc							
dm1							
dm2							

Spitalfields and Burlington: M2 Comparisons

Constants	Stage 1	Stage 2	Stage 3	Stage 4	Stage 5	Stage 6	Stage 7
I1						.205	
I2							
C					.557	.084	
P3						.071	
P4				.067		.138	
M1						.959	
di1							
di2							
dc							
dm1							
dm2							

Spitalfields and Belleville: M2 Comparisons

Constants	Stage 1	Stage 2	Stage 3	Stage 4	Stage 5	Stage 6	Stage 7
I1							
C					.273		
C						.318	
P3						*.000* *	
P4				.733		.094	
M1						.630	
di1							
di2							
dc							
dm1							
dm2							

Poundbury and Burlington: M2 Comparisons

Constants	Stage 1	Stage 2	Stage 3	Stage 4	Stage 5	Stage 6	Stage 7
I1						*.020*	*.045*
I2				.417	*.036*	.512	*.047*
C				.126	.132	.540	.199
P3			.452	.755	.875	.098	.299
P4			.305	.616	*.036*	.512	.047
M1						.185	
di1							
di2							
dc							
dm1							
dm2							

Poundbury and Belleville: M2 Comparisons

Constants	Stage 1	Stage 2	Stage 3	Stage 4	Stage 5	Stage 6	Stage 7
I1							
I2						.174	.429
C				1.000	.937	*.002* *	.142
P3				.376		*.019*	.408
P4			.530	*.048*	.147	.174	.429
M1					.613	.065	*.000* *
di1							
di2							
dc							
dm1							
dm2							

Belleville and Burlington: M2 Comparisons

Constants	Stage 1	Stage 2	Stage 3	Stage 4	Stage 5	Stage 6	Stage 7
I1							.201
I2							.604
C				*.045*	.056	*.000* *	.328
P3					.427	*.000* *	.814
P4			.305	.069	.601	.138	.604
M1					.199	.151	*.014*
di1							
di2							
dc							
dm1							
dm2							

Table 4.15: P-values for Mann-Whitney tests on distributions of M2 formation, relative to all formation stages of other teeth.

* *significant at table-wide level*

In summary, the patterning of differences is less clear in comparisons of distribution of M2 development. The obvious retardation of development in the Spitalfields sample, readily observable in examinations of the other permanent teeth, is no longer clearly evident. What is evident, however, is the consistent advancement of M_2 development in the Burlington sample.

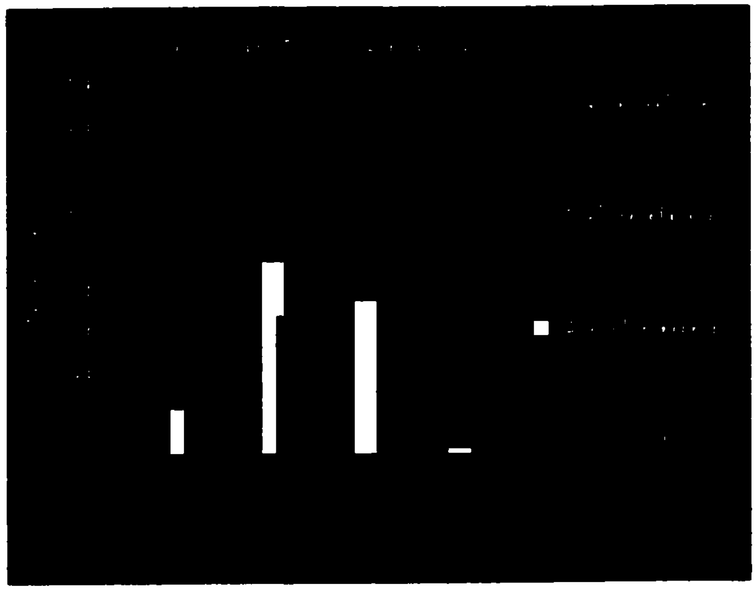


Figure 4.22: Distribution of M2 stages with P3 held constant as a reference tooth at stage 6

First Deciduous Incisor (di1)*

The number of significantly different results for comparisons of relative di1 development is low. Comparisons between the Spitalfields and Poundbury samples show no significant differences. There is, however, one significant difference between Belleville and the other two archaeological sam-

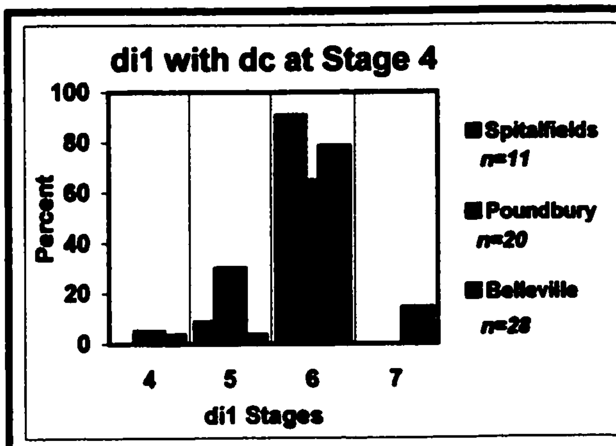


Figure 4.23: Distribution of di1 stages with dc held constant as a reference tooth at stage 4

* There are no data available for deciduous teeth in the Burlington sample. See Chapter 3.5.3

Spitalfields and Poundbury: di1 Comparisons

Constants	Stage 1	Stage 2	Stage 3	Stage 4	Stage 5	Stage 6	Stage 7
I1							
I2							
C							
P3							
P4							
M1			.219				
M2							
di2			.898	.832	.066	.744	
dc			.697	.244	.171	.662	
dm1			.600	.062	1.000	.831	
dm2			.935	.766			

Spitalfields and Belleville: di1 Comparisons

Constants	Stage 1	Stage 2	Stage 3	Stage 4	Stage 5	Stage 6	Stage 7
I1			.403				
I2		.429	1.000				
C							
P3							
P4							
M1	.046	1.000	.719				
M2							
di2			.210	.623	.051	.381	
dc			.381	.488		.996	
dm1			.511	.312	.181	.254	
dm2	.202			.685			

Poundbury and Belleville: di1 Comparisons

Constants	Stage 1	Stage 2	Stage 3	Stage 4	Stage 5	Stage 6	Stage 7
I1							
I2							
C							
P3							
P4							
M1			.067				
M2							
di2			.432	.809	.604	.263	
dc			.881	.007 *		.683	
dm1			.175	.443	.114	.519	
dm2				.264			

Table 4.16:* P*-values for Mann-Whitney tests on distributions of di1 formation, relative to all formation stages of other teeth.

** significant at table-wide level*

ples at the table-wide level. This occurs between the Belleville and Poundbury samples when dc is held constant at stage 4. (See figure 4.23) In this case the Belleville sample is advanced.

Second deciduous Incisor (di2)

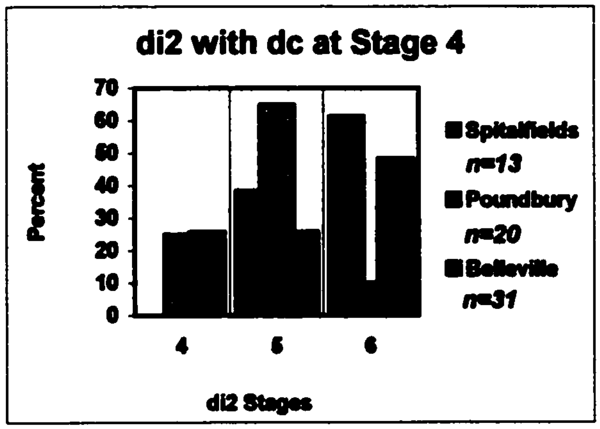


Figure 4.24: Distribution of di2 stages with dc held constant as a reference tooth at stage 4

The number of significant results for comparisons of di2 development is also low (table 4.17). Differences between the Poundbury and Spitalfields samples only reach table-wide significance when dc is held constant at stage 4 (see figure 4.24). In this instance, the Spitalfields sample is advanced over than of Poundbury. This is in contrast to the pattern evident for the permanent dentition. No other results reach table-wide significance.

Spitalfields and Poundbury: di2 Comparisons

Constants	Stage 1	Stage 2	Stage 3	Stage 4	Stage 5	Stage 6	Stage 7
I1							
I2							
C							
P3							
P4							
M1			.076				
M2							
di1							
dc			.839	.002 *			
dm1			.093	.921	.251	.886	
dm2		.662	.988	.853			

Spitalfields and Belleville: di2 Comparisons

Constants	Stage 1	Stage 2	Stage 3	Stage 4	Stage 5	Stage 6	Stage 7
I1			.073				
I2			.197				
C							
P3							
P4							
M1	.392	.314	.567				
M2							
di1				.420		.832	.885
dc			.832	.188			
dm1			.374	.373	.877	.445	
dm2	.091	1.000	.302	.427			

Poundbury and Belleville: di2 Comparisons

Constants	Stage 1	Stage 2	Stage 3	Stage 4	Stage 5	Stage 6	Stage 7
I1							
I2							
C							
P3							
P4							
M1			.043				
M2							
di1				.818		.634	.581
dc			.660	.073	.768	.374	
dm1			.294	.881	.166	.539	
dm2		.662	.219	.364			

***Table 4.17:* P-values for Mann-Whitney tests on distributions of di2 formation, relative to all formation stages of other teeth.**

** significant at table-wide level*

Spitalfields and Poundbury: dc Comparisons

Constants	Stage 1	Stage 2	Stage 3	Stage 4	Stage 5	Stage 6	Stage 7
I1							
I2							
C							
P3							
P4							
M1			.037				
M2							
di1				.867	.038	.650	.864
di2				.236	.141	.403	
dm1			.260	.011	.074	.697	
dm2			.046	.983		.364	

Spitalfields and Belleville: dc Comparisons:

Constants	Stage 1	Stage 2	Stage 3	Stage 4	Stage 5	Stage 6	Stage 7
I1			.006	.407			
I2			.012	.539			
C		.154	.118				
P3							
P4							
M1	.145	.470	.713				
M2							
di1						.879	.562
di2				.157	.139	.604	.470
dm1			.930	.023	.309	.011	.121
dm2			.540	.461	.087	.597	

Poundbury and Belleville: dc Comparisons

Constants	Stage 1	Stage 2	Stage 3	Stage 4	Stage 5	Stage 6	Stage 7
I1							
I2							
C							
P3							
P4							
M1			.026				
M2							
di1				.636		.252	.524
di2				.939	.815	.631	
dm1			.384	.405	.421	.151	
dm2			.119	.290		.120	

***Table 4.18:* P-values for Mann-Whitney tests on distributions of dc formation, relative to all formation stages of other teeth.**

Deciduous Canine (dc)

No results reached table-wide significance in this set of tests.

First Deciduous Molar (dm1)

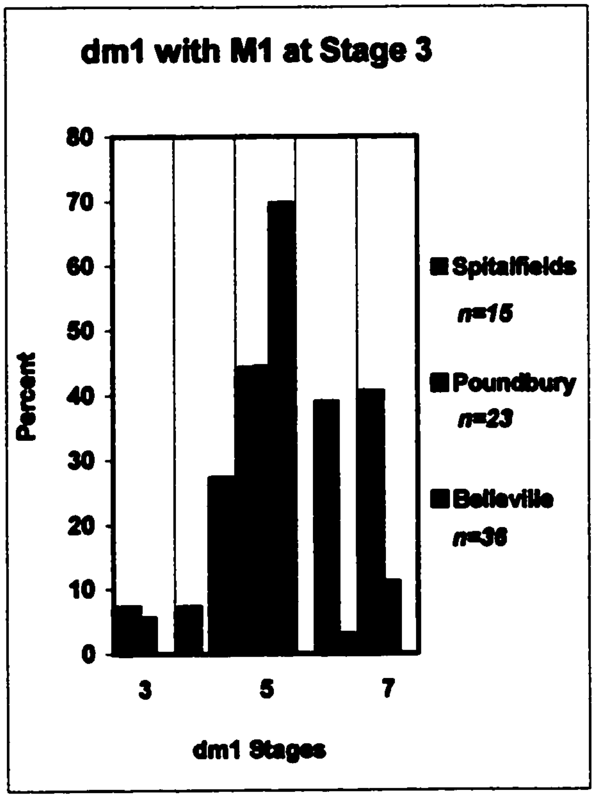


Figure 4.25: Distribution of dm1 stages with M1 held constant as a reference tooth at stage 3

Differences in the distribution of dm1 formation are small for all sets of comparisons. Only one result reaches significance at the table-wide level. This occurs between the Spitalfields and Poundbury samples, when M1 is held constant at stage 3.

Spitalfields and Poundbury: dm1 Comparisons

Constants	Stage 1	Stage 2	Stage 3	Stage 4	Stage 5	Stage 6	Stage 7
I1							
I2							
C		*.044*					
P3							
P4							
M1			*.005* *				
M2							
di1				.101	.694	*.039*	.860
di2				.379	.892	.477	
dc				*.020*	.075	.896	
dm2	.950	.755	.148	.539	.916		

Spitalfields and Belleville: dm1 Comparisons

Constants	Stage 1	Stage 2	Stage 3	Stage 4	Stage 5	Stage 6	Stage 7
I1			*.003*	.269			
I2		.429	*.012*	.590			
C			*.014*				
P3							
P4							
M1	.960	*.027*	*.012*				
M2							
di1				.323	.724	*.006*	.250
di2			.806	.730	.645	.313	.856
dc			.404	*.014*	.530	.058	
dm2	.189	.662	.148	.171	.073		

Poundbury and Belleville: dm1 Comparisons

Constants	Stage 1	Stage 2	Stage 3	Stage 4	Stage 5	Stage 6	Stage 7
I1							
I2							
C							
P3							
P4							
M1			.101				
M2							
di1				.223	.432	.325	
di2				.412	.644	.872	
dc			.231	.508	.133	.404	
dm2	.234	1.000	.855	.223	.447	*.031*	

Table 4.19: P-values for Mann-Whitney tests on distributions of dm1 formation, relative to all formation stages of other teeth.
** significant at table-wide level*

Second Deciduous Molar (dm₂)

Differences in distributions of dm2 development are significant at the table-wide level in only two instances. Both occur in comparisons between the Spitalfields and Belleville samples; when C and M1 are held constant at stage 3. In both cases, the Spitalfields sample is advanced over that of Belleville.

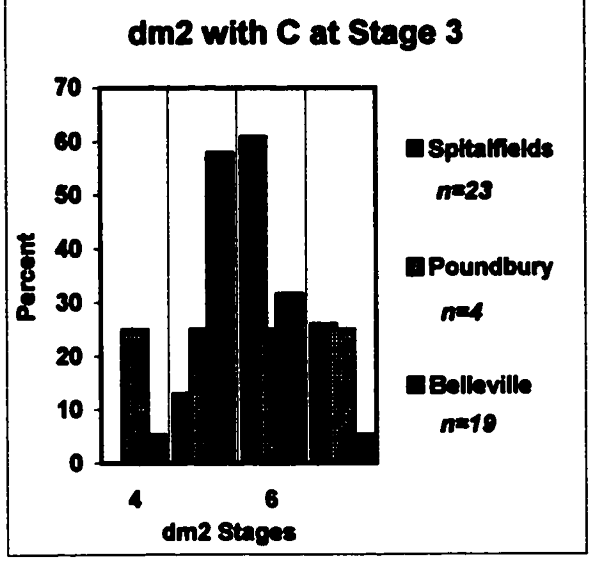


Figure 4.26: Distribution of dm2 stages with C held constant as a reference tooth at stage 3

Spitalfields and Poundbury: dm2 Comparisons:

Constants	Stage 1	Stage 2	Stage 3	Stage 4	Stage 5	Stage 6	Stage 7
I1							
I2							
C							
P3							
P4							
M1			*.006*	.138			
M2							
dI1				.321	.721	.886	.142
dI2				.525	.751	.981	
dc			.123	.267	.694	.127	
dm1			.960	.983	.489		

Spitalfields and Belleville: dm2 Comparisons

Constants	Stage 1	Stage 2	Stage 3	Stage 4	Stage 5	Stage 6	Stage 7
I1							
I2		.177	*.004*	.698			
C			*.001* *				
P3							
P4							
M1	.114	.443	*.002* *	.104			
M2							
dI1				.202		.917	.893
dI2				.885	.321	.800	.383
dc			.657	.753	.318	.444	.664
dm1			.586	*.048*	.211	.277	.122

Poundbury and Belleville: dm2 Comparisons

Constants	Stage 1	Stage 2	Stage 3	Stage 4	Stage 5	Stage 6	Stage 7
I1							
I2							
C							
P3							
P4							
M1			.314	.793			
M2							
dc				.881		.635	.091
dI1				.350	.406	.704	
dI2			.217	*.035*	.121	*.015*	
dm1			.496	*.036*	.462		

Table 4.20: P-values for Mann-Whitney tests on distributions of dm2 formation, relative to all formation stages of other teeth.
** significant at table-wide level*

4.3.3: Summary

The results presented in the two preceding sections illustrate the complex nature of studies of comparative dental development. This section summarizes these results, emphasizing the prevailing trends in order to present a clear, interpretable picture.

A: Extent of inter-population difference

Figure 4.27 illustrates a comparison of the percentages of table-wide significant results of Mann-Whitney tests for all pairs of inter-population comparisons. The most striking feature illustrated by this figure is the low percentage of

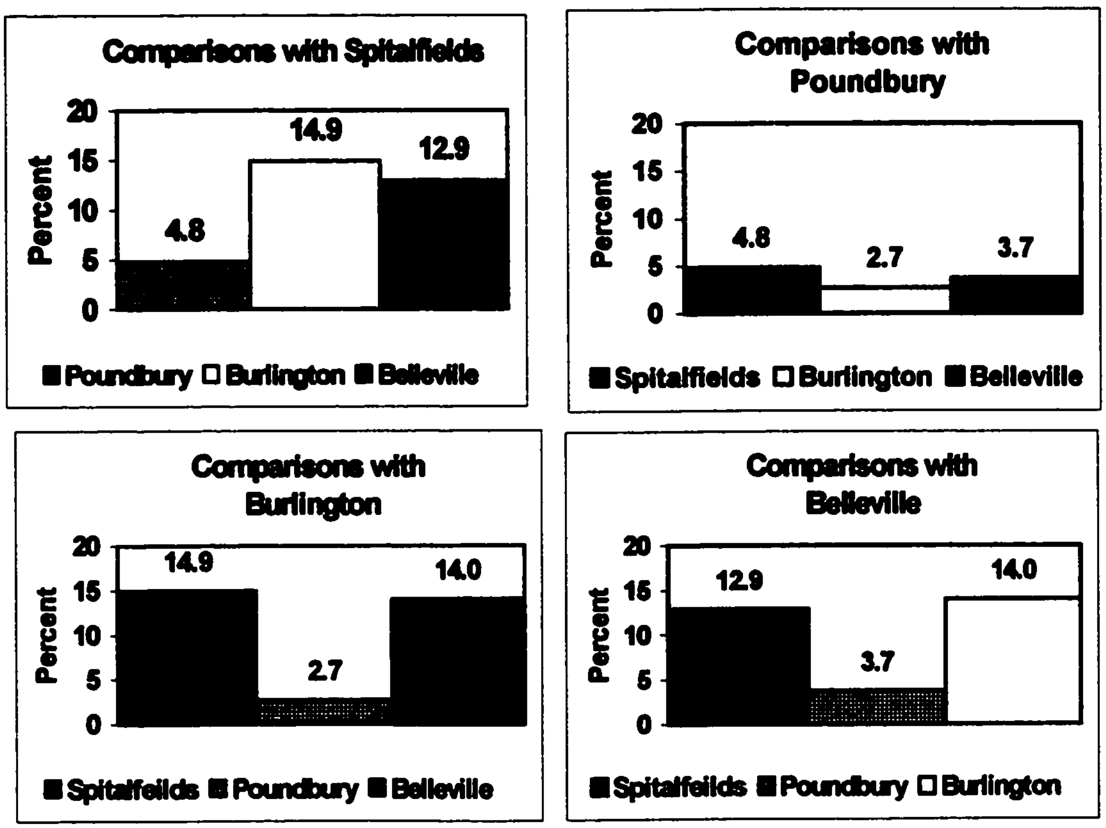


Figure 4.27: Total percentages of statistically significant results at the table-wide level of Mann-Whitney tests on paired comparisons of dental development.

cally significant results at the table-wide level of all sets of comparisons involving Poundbury. The highest percentage of significant results occur for comparisons between Burlington and Spitalfields, closely followed by those between Burlington and Belleville, and Spitalfields and Belleville.

B: Extent of inter-sample difference by tooth

Table 4.21 presents a comparison of the percentages of statistically significant results at the table-wide level for Mann-Whitney tests on all pairs of inter-sample comparisons broken down by tooth. It is interesting to note that differences in comparisons involving the deciduous dentition reach table-wide significance in only a small number of cases.

Spitalfields

	Poundbury	Burlington	Belleville
H	0.0	10.0	10.0
I2	0.0	14.3	9.5
C	25.0	5.9	20.0
P3	10.0	14.3	9.1
P4	0.00	35.7	0.0
M1	0.00	18.2	15.4
M2	0.00	0.0	16.7
di1	0.0		0.0
di2	8.3		0.0
dc	0.0		0.0
dm1	0.0		0.0
dm2	0.0		8.0

Poundbury

	Spitalfields	Burlington	Belleville
H	0.0	0.0	0.0
I2	0.0	0.0	0.0
C	25.0	5.6	5.6
P3	10.0	9.5	0.0
P4	0.00	0.0	0.0
M1	0.00	0.0	3.2
M2	0.00	0.0	11.8
di1	0.0		7.7
di2	8.3		0.0
dc	0.0		0.0
dm1	0.0		0.0
dm2	0.0		0.0

Burlington

	Spitalfields	Poundbury	Belleville
H	10.0	0.0	11.1
I2	14.3	0.0	27.3
C	5.9	5.6	13.6
P3	14.3	9.5	15.7
P4	35.7	0.0	11.8
M1	18.2	0.0	5.3
M2	0.0	0.0	11.8
di1			
di2			
dc			
dm1			
dm2			

Belleville

	Spitalfields	Poundbury	Burlington
H	10.0	0.0	11.1
I2	9.5	0.0	27.3
C	20.0	5.6	13.6
P3	9.1	0.0	15.7
P4	0.0	0.0	11.8
M1	15.4	3.2	5.3
M2	16.7	11.8	11.8
di1	0.0	7.7	
di2	0.0	0.0	
dc	0.0	0.0	
dm1	0.0	0.0	
dm2	8.0	0.0	

Table 4.21: Percentage of statistically significant results at the table-wide for each set of comparisons of developmental distributions, for each tooth.

The major differences between the Belleville and Spitalfields samples are evident when deciduous teeth are held constant at the later stages of root formation (see figure 4.7), while differences between Poundbury and the other two samples are small and follow no distinct pattern.

The only table-wide, significant difference in CEI for individuals in the M1 at stages 6-7 occurs between the Burlington and Belleville samples. The CEI for Belleville is more expanded than that of Burlington, indicating relative retardation.

An examination of the results for the paired comparisons, however, presents a more complex picture. In many instances, there are statistically significant differences between the Belleville and Spitalfields samples, with the former showing consistent advancement over the latter (see table 4.7). This scenario is probably a more accurate reflection of the true situation, since the paired comparisons include all combinations of teeth. Furthermore, the use of paired comparisons is not hampered by the exclusion of large number of individuals due to incomplete dentitions, as is the situation with the CEI (see section 4.2.1). Both sets of results, however, concur in demonstrating consistent retardation in the Spitalfields sample.

The CEI for M2 at stages 6-7 (the oldest individuals in this study) reveals a different picture. The development of the Belleville sample is shown as condensed, while the other three samples are expanded. These differences are not significant at the table-wide level. Conversely, an examination of the paired comparison results contradicts this to some extent, (see figure 4.23). In most cases, however, the only statistically significant differences between individuals in this older, developmental category are between Burlington and the other samples. What is clear, is that the obvious retardation of the Spitalfields sample is no longer evident in the dentition of these older children.

4.4: Discussion

4.4.1: Applicability of the term "close biological affinity?"

Has the preceding section demonstrated a significant difference in patterns of tooth formation between groups of close biological affinity? Before this question can be answered it is necessary to discuss the notion of biological affinity.

It could be argued that considerable genetic differences probably exist between the four populations included in this study, and that "close biological affinity" is not an appropriate term in this instance. However, the term must be examined in a broader context. On a global scale, the four groups can be considered relatively homogenous, since they are considerably different from the indigenous peoples of North America, Africa and Asia. It is these three broad population groups that have been the subject of studies into comparative dental development (see Crossner and Mansfield, 1983; Owsley and Jantz, 1983; Tompkins, 1996) and observed to be different from modern Europeans*

For the purpose of this study, therefore, the four samples can be considered of close biological affinity in a global sense, since they are all not only of European origin, but are all either British or of largely British ancestry (see chapter 3.4).

* The term "European" is frequently applied to not only indigenous Europeans, but also to all people of European ancestry, wherever their place of birth.

4.4.3: Significance of the difference in the patterns of dental development

If it is accepted that the four populations are of close biological affinity, can it also be accepted that significant differences in dental development exist between them? Although statistically significant differences are evident in both the CEI and paired comparisons, there are many stages of development where there are no significant differences. It could, therefore, be argued that when differences do occur it is by chance, or merely an artifact of normal human variation. If this were the case, then the percentage of results with p-values of less than .05 would be far lower (5% or less). Furthermore, the sequential Bonferoni procedure minimizes this possibility, since it provides a very conservative estimate of significance (Rice 1989).

If the results had occurred by chance, one would expect statistically significant results to be randomly distributed, both throughout inter-population comparisons, and throughout all teeth and developmental stages. It is clear that this is not the case. Figure 4.27 illustrates variation in the results of inter-population comparisons, while table 4.21 provides a clear picture of the pattern of results evident in comparisons involving different teeth. A re-examination of table 4.14 (page 104) provides a striking example of the patterning of results according to tooth type , population, and stage of development. All of these results indicate that significant differences in the duration and patterning of development do exist between the populations under study.

4.4.3: Causal Factors

If we accept that the four groups are of close biological affinity, and that significant differences exist between them, what factors are responsible? Although there are differences between all the groups under study at some stages of development, the most striking observation to be made is that the development of the Spitalfields sample is retarded in comparison to the other three samples.* In order to determine the reasons for this, it is necessary to examine when this retardation occurs.

A: The youngest developmental category

Between sample differences are not statistically significant for the youngest individuals in this study. This makes biological sense if environmental influences are dominant causal factors. Since the development of the dentition of these youngest individuals occurred mainly during the pre-natal period, it can be taken as a reflection of the uterine environment. Since this environment is relatively buffered from stress, differences in developmental rates due to environmental causes would not be expected to occur until later in life. Furthermore, immunity provided by breastfeeding would offer the youngest infants some degree of protection from infectious diseases, and provide a reliable source of nourishment.

* This concurs with the findings of Stringer and colleagues (1990) who found the dental development of the Spitalfields named sample to be retarded in comparison to other groups of modern Europeans.

B: The intermediate developmental categories

The most striking difference in the results of both the paired comparisons of developmental distributions and the CEI occur in children sampled during the growth period after crown completion of the anterior deciduous teeth and before crown completion of the second permanent molar. As discussed previously, in the absence of known chronological age, the length and timing of this period is difficult to assess. It is probable, however, that this period begins in the latter half of infancy (in the cases of Spitalfields and Belleville)* and ends prior to the onset of adolescence. Throughout this whole period of development, the Spitalfields sample shows consistent retardation when compared to the other three samples.

As discussed in the preceding section, the most striking developmental differences in the paired comparisons exist between the Spitalfields and Belleville samples, when deciduous teeth are held constant at the latter stages of root development. The high mortality rate for children at this stage of development in both Belleville (Herring et al. 1998) and Spitalfields (Molleson and Cox, 1993) suggests a high proportion of deaths due to weanling diarrhea syndrome and food allergies. Herring and colleagues (1998) describe this condition as follows:

> *The infants' immature digestive system and immune systems are forced to cope with food-borne allergies and other pathogens while the infant simulatneously risks malnutrition as maternal milk production falls off in association with fewer and less intense nursing bouts."*

* Using the biometric method and isotopic analysis, Herring and colleagues (1998) provide evidence suggesting that the weaning process began as early as 12 months in the Belleville sample, and state that this was also the fashion in European society during the 19th century.

Is it possible that the children of Spitalfields suffered from this syndrome to a greater extent than those of Belleville? The striking retardation in dental development evident in the Spitalfields sample during this phase suggests that this was the case. Conversely, it could be argued that the Belleville children succumbed to this syndrome in greater numbers, dying before retardation in dental development became apparent. The retardation in the Spitalfields sample could be an indication that these children suffered, yet recovered from the weanling diarrhea syndrome in greater numbers. An examination of other lines of evidence provides some clues to the relative likelihood of these two scenarios.

As discussed in section 4.3, documentary evidence exists which indicates that the majority of childhood deaths in the Belleville sample were attributable to acute infection (Saunders et al. 1995b). Although this information is unavailable for the other two archaeological populations, the possibility that a similar situation existed in Poundbury but not Spitalfields must be discussed.

How likely is this scenario? Little is known about the disease load in Roman Dorchester, making it difficult to interpret this regarding the Poundbury sample. However, archaeological evidence suggests the existence of a fresh water supply and an effective sewage system (Collingwood and Richmond, 1971) Houses were large and well spaced (Frend, 1968), providing an environment that protected against the spread of infectious diseases. Furthermore, the houses had spacious gardens containing a variety of fruit trees and vegetables. In contrast, Spitalfields in the 18th and 19th centuries is known to have suffered from overcrowding and lack of an effective sewage system. Infected water supplies were common, as were outbreaks of water-borne infection. The 19th-century population of Belleville, however, also experienced a problem with contaminated water, particularly towards the end of the period. However, it is likely that this was only a problem towards the end of the cemetery period in Belleville, since it accompanied the trend towards increasing urbanization. In contrast, the Spitalfields sample represents an urban population for the whole period of cemetery use. It can therefore be argued that the lack of hygiene associated with urban living is seen more strongly in the Spitalfields sample.

Furthermore, although the children of Belleville undoubtedly suffered from diarrheal diseases, the mortality records show that this was not a frequent cause of death, other than for infants due to weanling diarrhea. It could be argued that the children in Belleville enjoyed better health due to good nutrition, and so were able to cope with bouts of illness in a way the Spitalfields children were not. Nutritional deficiency is known to retard somatic growth and also to increase susceptibility to infectious disease (Largo, 1993; Freeman et al. 1995). The diets of the populations in this study must therefore be discussed as a possible causal factor with regard to retardation of dental development.

Is it likely that the relatively wealthy children buried in the vaults of Christ Church, Spitalfields suffered from nutritional deficiencies?

Harris writes:

> *Preferred foods generally pack more energy, protein, vitamins or minerals per serving than avoided foods.*
>
> *(Harris 1986:15)*

while the poor existed on grains, vegetables and fish. It is interesting to note that this is reversed in modern British society where foods which are high in fat and sugar are highly prized amongst the lower socio-economic groups, while the middle class place an emphasis on what it now seen as "healthy eating", and prefer a diet which is high in grains, vegetables, and fish (Taylor, 1979; Tollinson and Wagner, 1992).

How different was the diet of Belleville? It could be argued that British immigrants enjoyed a higher standard of living upon their arrival in Upper Canada, and had the means to replicate a "prestigious" British diet. Abonyi (1993) examined changes in eating habits which accompany migration, and discovered that while people tend to cling to familiar tastes, adaptation to new local surroundings involves the incorporation of new foods into the diet.

Saunders and colleagues (1997) demonstrated that caries rates for the Belleville were high, suggesting a link to sugar consumption. While the Belleville caries rates are not as high as those of post-1850 century British samples (Saunders *et al.*, 1997), they are slightly higher that those of Spitalfields (Whittaker, 1993).

Using historic sources, Abonyi (1993) indicated that the Belleville diet comprised mainly meat, vegetables and bread. She also stated that the inhabitants of Belleville also incorporated local foodstuffs into their recipes, such as maize, pumpkins and wild fruits. It would appear, therefore, that while some elements bear a similarity to the diet of Spitalfields, the occupants of 19th-century Belleville did not replicate a British diet entirely. For example, it is likely that they did not view grains and vegetables in the same way. The fact that 19th century Belleville included rural populations could mean that people felt closer ties to the land, and therefore would not have viewed fresh vegetables and grains as undesirable.

In summary, although the environments of Belleville and Spitalfields were similar in some ways, a strong case can be made in favour of biologically poorer living conditions for the latter, and it can be argued that poorer diet and lower standards of hygiene had a detrimental effect on even the more affluent inhabitants of 18th – 19th century London. The evidence suggests that poor nutrition and disease were sufficiently severe to retard the dental development of some of the Spitalfields children, who then died.

C:The oldest developmental category

The statistically significant results apparent for the CEI for the oldest group of individuals shows that all samples are similar with the exception of Belleville, which shows a condensed developmental pattern. As discussed in section 4.3, this is in contrast to the paired comparison results which shows that the greatest difference in development of individuals in the oldest age group occurs between Burlington and the three archaeological samples. Since the paired comparison results provide a more detailed picture of development and allow the inclusion of partial dentitions, this set of results is probably a closer reflection of reality.

Both sets of results, however, show that the Spitalfields sample is no longer retarded in comparison to the other archaeological samples. The most obvious interpretation of this phenomenon is that the mechanism of "catch up growth", as documented for somatic growth (Freeman et al. 1995; Largo, 1995) is in operation. Put simply, detrimental environmental influences which operated earlier in life have been removed for children surviving to this age category, and conditions are now good enough for individuals to experience a period of rapid growth, in order to "catch up" and fulfill their growth potential. Before this interpretation can be accepted, alternatives must be discussed.

Firstly, the data analyzed in this chapter are not derived from longitudinal growth studies (see chapter 3). The differences and similarities evident in the analysis of these data, therefore, are not the result of "tracking" the developmental profiles of a group of individuals, but are merely providing developmental snapshots of a number of children at different ages. Consequently, there is no way of knowing if the children observed in the oldest age group ever experienced the same patterns of growth as those observed in the earlier.

Perhaps more seriously, mortality bias may be in operation in the archaeological samples, making them unrepresentative of the living populations. This could account for the relative advancement of the Burlington sample in this age category, since this is the only sample of living children. Furthermore, it is possible that children in the preceding age categories (prior to M2 crown completion) died of chronic disease which affected their dental development, while the older children died of either traumatic causes or acute diseases which did not.

4.4.4: Summary

In summary, the data analyzed in this chapter suggest that:

1 *There are differences in the dental development of the four groups under study.*

This supports the hypothesis that environment has an influence on the duration of dental development, since the four groups are of relatively close biological affinity

2 *The most striking differences occur between Spitalfields and the other samples for children who died in later infancy.*

This may be accounted for by gastrointestinal disease, since weanling diarrhea was probably common for children in this age group.

3 *Historical records show that the Spitalfields environment was characterized by poor sanitary conditions and inadequate diet.*

This supports the hypothesis that environmental influences caused retardation in dental development in the Spitalfields sample.

4 *The retardation in Spitalfields is no longer evident in children after crown completion of the second permanent molar.*

This implies that either: a) catch up growth was in operation and that children who experienced dental retardation during later infancy had then experienced a period of accelerated development, allowing them to achieve normal standards by late childhood, or b) differences were due to age related differences in causes of death.

Chapter 5

Correlates of Human Dental Development:

What is the Relationship Between Dental Development, Tooth Size, and Mandibular Size?

5.1: Introduction

This chapter examines the relationship between dental development, tooth size, and mandibular size. This investigation serves two purposes.

Firstly, by exploring these relationships, factors involved in the mechanisms of dental retardation may come to light. For example, an association between dental development and overall mandibular size could suggest that individuals who are more advanced in dental development, are also more advanced skeletally. Furthermore, an association between dental development and size of the dental arcade could indicate that crowding is playing a role in the retardation of dental development.

Secondly, evidence of a correlation between tooth size and dental development will support the hypothesis that the retardation discussed in the preceding chapter is due to environmental influences. This is because some researchers have found a link between low birth weight, socio-economic status and tooth size (see Keiser, 1990). Furthermore, a secular trend in tooth size over two generations was discovered by Garn and colleagues (1968). Since the results of these studies suggest an environmental role in the determination of tooth size, a significant correlation between *dental development* and tooth size will strengthen the argument that a poor environment caused retardation in the Spitalfields sample.

In addition to an examination of the correlation between dental development, tooth size and mandibular size, this chapter investigates inter-group differences in tooth size and mandibular size. This investigation is undertaken for both children and adults. Furthermore, an investigation of mortality bias is undertaken by intra-population comparisons of these dimensions in adults and subadults.

source of error to this study, so were avoided. The sample sizes in this chapter are smaller than those in chapter 4 (See table 5.1) due to post mortem damage and pathological damage due to caries and excessive tooth wear.

In order to assess the degree of mortality bias present in the three samples investigated in this chapter, mandibular and dental metric data were required. In the case of Burlington, a random sample of 30 males and 30 females over the age of 18 years was selected. For the two skeletal samples, Spitalfields and Poundbury, 30 individuals of each sex were selected from both samples. Individual skeletons were selected on the basis of completeness; only individuals with all teeth present and undamaged were analyzed. In the Spitalfields sample, advanced caries and ante-mortem tooth loss rendered the majority of individuals unmeasurable. In the Poundbury sample, the number of measurable individuals was limited by the amount of excessive tooth wear. This reduction of measurable teeth dictated that the sample was limited to only 60 individuals from each sample, mainly from the young adult age categories.

5.2.2: Measurements

A: Tooth Size

Maximum mesiodistal crown diameter was used as an indicator of tooth size, and was measured according to the method outlined by Mayhall (1992). Some researchers have criticized this method (see Hillson, 1996), since rotated or displaced teeth no longer retain their spatial mesiodistal integrity (Hillson, 1996). For this reason, the use of contact points is frequently the preferred measurement.

In this study however, the large number of loose teeth made the maximum mesiodistal measurement the most appropriate.

Measurements were limited to those of the left mandibular teeth. In instances were the left tooth was missing or damaged, the right tooth was substituted. Measurements were taken to the nearest 0.01mm using Vernier calipers and then rounded to the nearest 0.1mm. As mentioned in section 5.1, only unworn, undamaged teeth were included in this study.

Figure 5.1: Illustration of maximum mesiodistal diameter and contact point diameter (adapted from Mayhall, 1992)

B: Mandibular Size

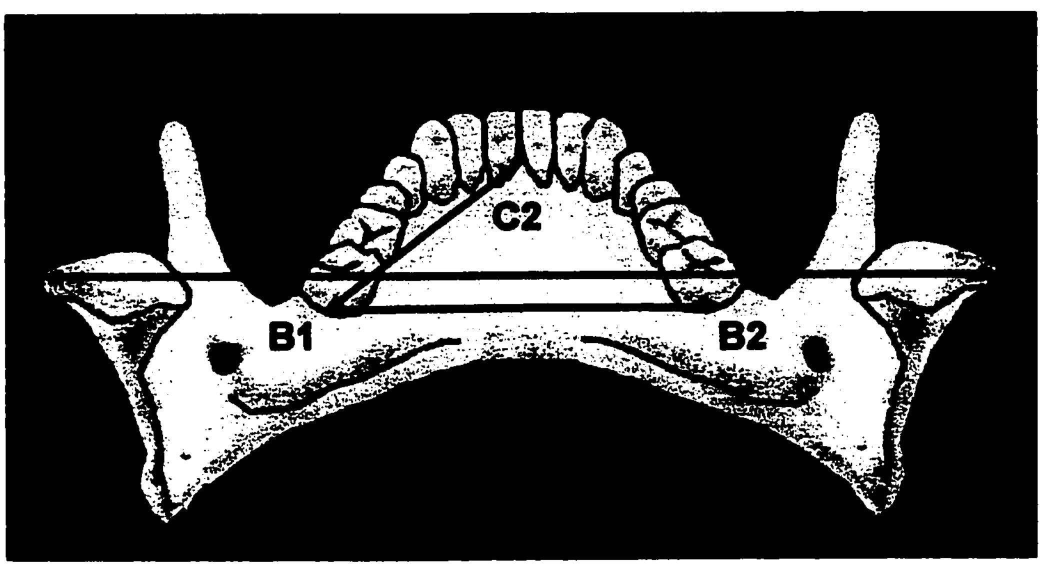


Figure 5.2: Mandibular measurements

In order to assess mandibular size, the following five measurements were taken (see figure 5.2).

A1-A2 Bi-condylar width

B1-B2 Dental arcade width

D1-D2 Bi-gonial width

D1-C1 Gonial angle-inferior limit of mandibular symphysis

B1-C2 Distal limit of dental arcade-superior limit of mandibular symphysis

Measurements were taken with Vernier calipers and then rounded to the nearest 1.0mm. Again, only undamaged mandibles were included in this study.

C: Long Bone Lengths

Adult stature was estimated for the Spitalfields and Poundbury population in order to obtain an attained skeletal size standard for comparison with mandibular measurements. This necessitated the measurement of the femur and tibia of 155 skeletons from the Spitalfields collection, and 266 from the Poundbury collection. These numbers represent all adult skeletons with these elements complete and undamaged. Maximum lengths to the nearest 1.0mm were obtained by the use of an osteometric board.

D: Sex Determination

Due to sexual dimorphism, male and female adult metric data needed to be analyzed separately. This was straightforward for the Burlington sample, since the sex of all individuals was known. In the case of Poundbury and for the "unnamed" Spitalfields skeletons, sex was estimated. This involved the macroscopic observation of sexually dimorphic traits of the pelvis (see table 5.2).

Sciatic notch	Subpubic angle	Subpubic concavity	Ischio-pubic ramus
Ventral arc	Preauricular sulcus	Obturator foramen	Pelvic brim
acetabulum	Sacral segments	Auricular surface	

Table 5.2: Pelvic traits used to determine sex

5.2.3: Recording

The metric data described above were noted on a recording sheet. This information was later entered into an Excel spreadsheet and double checked for data entry errors. The data were then imported into an Access database in order to facilitate cross referencing with the developmental data described in the preceding chapter.

5.2.4: Intra observer error

A: Mesiodistal Diameter

The entire dentitions of 40 randomly chosen specimens were re-measured three months after initial data collection. After rounding the measurements to the nearest 0.1mm (as recommended by Hillson, 1992), less than 1.25% of measurements lay outside the accepted range of precision (table 5.3)

Tooth Type	*% of Measurements >.01mm different*
Uniradicular teeth	1.1%
Molars	1.3%
Total	1.2%

Table 5.3: Intra-observer error (mesiodistal diameter)

B: Mandibular measurements

Measurement	% of Measurements >.01mm different
A1-A2	0%
B1-B2	1.3%
D1-C1	0%
B1-C2	0%
D1-D2	2%
Total	0.8%

Table 5.3: Intra-observer error (mandibular measurements)

The five mandibular measurements described above were repeated on 50 randomly chosen mandibles four months after initial data collection. After rounding to the nearest 1.0mm, less than 1% of measurements lay outside the acceptable range of precision (table 5.4).

5.2.5: Analysis

A: Inter sample comparison of adult tooth size

In order to investigate the relationship between mandibular size and tooth size, an assessment of both these parameters in adulthood was necessary. In order to compare adult tooth size between populations, mesiodistal diameters of permanent teeth were compared separately for males and females. The data were assessed for normality using the Kolmogorov-Smirnov one-sample test. The results confirmed a normal distribution for all three sets of data. One-way ANOVA tests were then conducted to compare means. Tables of results were then subjected to the sequential Bonferroni procedure as described in the preceding chapter.

B: Inter sample comparison of sub-adult tooth size

Sub-adults were defined as individuals with dental developmental prior to M2 root completion. Due to lack of available sex information for the skeletal samples, it was not possible to analyze the mesiodistal diameters of males and females separately. Instead, the data from all individuals from each population were assessed for normalcy using the Kolmogorov-Smirnov one-sample test. After confirming that the each data set showed a normal distribution, one way ANOVA tests were performed. Statistical significance was considered at a table-wide level after the sequential Bonferroni procedure had been performed.

C: Investigation of mortality bias: comparison of adult and subadult tooth size

Within sample comparisons were made between adult and sub-adult mesiodistal diameters. This was undertaken in order to assess if there was mortality bias present for tooth size. Put simply, this procedure can be used to check if individuals with small teeth are more or less likely to survive to adulthood. For the purpose of comparability, the data from adult males and females were pooled. Again, one way ANOVA tests were used to compare means, after confirming that each distribution was normal using the Kolmogorov-Smirnov one-sample test. Statistical significance was considered at a table-wide level after the sequential Bonferroni procedure had been performed.

D: Correlation of mandibular measurements

The five mandibular measurements represent different portions of the mandible. For example measurement D1-D2 (bigonial breadth), represents the width of the entire mandible, while B1-B2 represents the breadth of the dental arcade. In order to investigate the inter-relationships of these variables, it was necessary to determine measurement inter-correlations. Pearson's correlation coefficient was used to measure the linear association between all permutations of mandibular measurements. Statistical significance was considered at a table-wide level after the sequential Bonferroni procedure had been performed.

E: Inter population comparisons of adult mandibular size

One way ANOVAs were used to compare differences in adult mandibular size between populations. Males and females were compared separately for Spitalfields and Poundbury (as mentioned in section 5.2.1, this information was not available for the Burlington sample).

F: Correlation between adult mandibular and tooth size

In order to investigate the inter-relationships between mandibular dimensions and tooth size in adults, each tooth was, in turn, examined in relation to each mandibular measurement. Pearson's correlation coefficient was used to assess the strength of each individual relationship. Statistical significance was considered at a table-wide level after the sequential Bonferroni procedure had been performed.

G: Correlation between tooth size and dental development

The relationship between tooth size and dental development was investigated using the results of the paired comparisons from the preceding chapter. For each developing tooth, the paired comparison with the largest sample size was selected, and the tooth sizes for that tooth compared. For example, to investigate the relationship between I1 development and mesiodistal diameter, all first permanent incisors with C at stage 3 were measured. The correlation between this measurement and developmental stage was then examined using Kendall's non-parametric, tau-b correlation coefficient. Statistical significance was considered at a table-wide level after the sequential Bonferroni procedure had been performed.

H: Correlation between mandibular size and dental development

The relationship between mandibular size and dental development was investigated using the selected paired comparisons as described above. In this instance, the developmental stages were compared, in turn, to each mandibular measurement.

5.3: Results

5.3.1: Inter-sample comparisons of adult tooth size

Inter-sample comparisons of female adult tooth size, as indicated by maximum mesiodistal diameter, show that the Burlington women generally have the largest teeth. This is true for I1, I2, P3, P4, and M1. The only statistically significant difference at the table-wide level, however, occurs in the comparison of P3 size.

An investigation of the inter-sample differences in male tooth size, yielded different results, with Burlington having significantly larger teeth in all cases except I2, C and M2. In these cases, although the mean tooth size of the Burlington sample is still largest, the one way ANOVA results are not statistically significant at the table-wide level.

Comparisons involving only Poundbury and Spitalfields (table 5.2) show no significant difference in tooth size at the table-wide level.

In summary, there are minor differences in female tooth size, with the Burlington females having significantly larger teeth in only one set of comparisons. In the case of the males, the differences are larger, with Burlington males having significantly larger teeth in cases except for I2, C and M2.

Adult Females

	N	Mean	SD	*f*	*p*
I1					
Spitalfields	30	5.00	.48		
Poundbury	30	5.12	.36		
Burlington	30	5.34	.44	4.630	0.12
I2					
Spitalfields	30	5.70	.53		
Poundbury	30	5.77	.29		
Burlington	30	5.82	.42	.629	.535
C					
Spitalfields	30	6.80	.46		
Poundbury	30	6.68	.40		
Burlington	30	6.56	.55	1.937	.150
P3					
Spitalfields	30	6.76	.39		
Poundbury	30	6.50	.44		
Burlington	30	6.92	.41	*7.439*	*.001 **
P4					
Spitalfields	30	6.93	.43		
Poundbury	30	6.79	.47		
Burlington	30	7.04	.46	2.232	.113
M1					
Spitalfields	30	11.05	.74		
Poundbury	30	11.14	.56		
Burlington	30	11.25	.63	.730	.485
M2					
Spitalfields	30	10.55	.69		
Poundbury	30	10.56	.53		
Burlington	30	10.52	.59	.047	.954

Adult Males

	N	Mean	SD	*f*	*p*
I1					
Spitalfields	30	4.96	.46		
Poundbury	30	5.02	.39		
Burlington	30	5.36	.45	*7.200*	*.001 **
I2					
Spitalfields	30	5.71	.56		
Poundbury	30	5.74	.37		
Burlington	30	5.99	.41	3.526	.034
C					
Spitalfields	30	6.71	.57		
Poundbury	30	6.59	.38		
Burlington	30	6.92	.40	.3955	.023
P3					
Spitalfields	30	6.71	.46		
Poundbury	30	6.51	.56		
Burlington	30	7.01	.41	*9.082*	*.000 **
P4					
Spitalfields	30	6.93	.46		
Poundbury	30	6.67	.52		
Burlington	30	7.25	.48	*10.466*	*.000 **
M1					
Spitalfields	30	11.04	.73		
Poundbury	30	11.07	.61		
Burlington	30	11.54	.56	*5.5657*	*.005 **
M2					
Spitalfields	30	10.49	.65		
Poundbury	30	10.46	.57		
Burlington	30	10.57	.52	.272	.762

Table 5.5: Comparison of tooth size: all adults

** Significant at table-wide level*

Adult Females

	N	Mean	SD	f	p
I1					
Spitalfields	30	5.00	.48		
Poundbury	30	5.12	.36	1.187	.281
I2					
Spitalfields	30	5.70	.53		
Poundbury	30	5.77	.29	.397	.531
C					
Spitalfields	30	6.80	.46		
Poundbury	30	6.68	.40	1.207	.276
P3					
Spitalfields	30	6.76	.39		
Poundbury	30	6.50	.44	5.517	.022
P4					
Spitalfields	30	6.93	.43		
Poundbury	30	6.79	.47	2.232	.113
M1					
Spitalfields	30	11.05	.74		
Poundbury	30	11.14	.56	.279	.599
M2					
Spitalfields	30	10.55	.69		
Poundbury	30	10.56	.53	.011	.918

Adult Males

	N	Mean	SD	f	p
I1					
Spitalfields	30	4.96	.46		
Poundbury	30	5.02	.39	.323	.572
I2					
Spitalfields	30	5.71	.56		
Poundbury	30	5.74	.37	.073	.788
C					
Spitalfields	30	6.71	.57		
Poundbury	30	6.59	.38	.853	.360
P3					
Spitalfields	30	6.71	.46		
Poundbury	30	6.51	.56	2.645	.109
P4					
Spitalfields	30	6.93	.46		
Poundbury	30	6.67	.52	4.281	.043
M1					
Spitalfields	30	11.04	.73		
Poundbury	30	11.07	.61	.037	.849
M2					
Spitalfields	30	10.49	.65		
Poundbury	30	10.46	.57	.053	.819

Table 5.6: Comparison of adult tooth size: Spitalfields and Poundbury

5.3.2: Inter-sample comparison of sub-adult tooth size

Inter-sample comparisons of sub-adult tooth size follow a similar pattern to that of the male adults, with the highest mean tooth size for Burlington. The teeth of the Spitalfields children tend to be the smallest, with Poundbury lying somewhere between these two extremes. This is true for all deciduous teeth, and all permanent teeth with the exception of M2 and P4 where the differences are not statistically significant (Table 5.7).

5.3.3: Investigation of mortality bias: comparison between adult and sub-adult tooth size

Evidence was found to support the hypothesis that individuals who survive to adulthood have larger teeth than those who die in childhood. In the Spitalfields sample, mean permanent tooth size is significantly larger in adults than children for all teeth with the exception of the P4 and M2 (table 5.8). For Poundbury, mean adult tooth size is not significantly larger. The only significant result at the table-wide level for Poundbury indicates that *child* tooth size is larger for P3.

In summary, there is a clear pattern of difference in the Spitalfields sample, with individuals surviving to adulthood having larger teeth than those who died in childhood. This pattern is not evident in the Poundbury sample.

All Sub-adults

	N	Mean	SD	*f*	*p*		N	Mean	SD	*f*	*p*
I1						**M2**					
Spitalfields	65	4.96	.39			Spitalfields	21	10.70	.87		
Poundbury	35	5.20	.46			Poundbury	75	10.70	.71		
Burlington	164	5.36	.43	*20.754*	*.000 **	Burlington	93	10.60	56	.550	.578
I2						**di1**					
Spitalfields	66	5.51	4.56			Spitalfields	55	3.75	.34		
Poundbury	47	5.58	5.47			Poundbury	46	4.05	.39		
Burlington	146	5.90	4.22	*19.996*	*.000 **	Burlington	86	4.14	.37	*19.228*	*.000 **
C						**di2**					
Spitalfields	54	5.94	.59			Spitalfields	65	4.32	.4287		
Poundbury	59	6.76	.56			Poundbury	52	4.57	.3886		
Burlington	118	6.84	.56	*48.845*	*.000 **	Burlington	113	4.69	.4107	*16.926*	*.000 **
P3						**dc**					
Spitalfields	24	6.45	.60			Spitalfields	65	5.41	.54		
Poundbury	61	6.79	.56			Poundbury	45	5.73	.62		
Burlington	106	6.97	.43	*11.202*	*.000 **	Burlington	145	5.95	.54	*27.153*	*.000 **
P4						**dm1**					
Spitalfields	20	7.02	.65			Spitalfields	93	7.47	.53		
Poundbury	52	7.00	.69			Poundbury	95	7.99	.72		
Burlington	100	7.07	.51	.282	.754	Burlington	152	8.01	.51	*26.930*	*.000 **
M1						**dm2**					
Spitalfields	79	10.53	.90			Spitalfields	99	9.47	.80		
Poundbury	120	11.02	.81			Poundbury	99	9.69	.75		
Burlington	157	11.31	.61	*27.944*	*.000 **	Burlington	152	10.63	.57	*20.407*	*.000 **

Table 5.7: Comparison of tooth size: all sub-adults

** Significant at table-wide level*

5.3.4: Correlation between mandibular measurements

The strength and pattern of correlation between mandibular measurements show differences when broken down by sex and sample (figure 5.3). An analysis of adult females from Spitalfields shows a table-wide significant correlation between one pair of measurements: D1-D2 with D1-C1. The association between all other pairs of measurements is weak (table 5.9). These results show that, while some measurements relating to overall mandibular size are significantly correlated, there is no correlation between this set of measurements and those relating to the size of the dental arcade.

In contrast, an analysis of the Spitalfields males reveals that two pairs of mandibular measurements are correlated to a table-wide level of statistical significance. These results show a strong association between the width of the dental arcade (B1-B2) and overall mandibular width (A1-A2). In addition length and breadth of the mandible are also strongly associated (D1-C1 and D1-D2).

Analysis of data obtained from the Poundbury collection shows a weaker association between mandibular measurements. In the case of the females, none of the measurements are correlated to a table-wide, statistically significant level. In contrast, two pairs of measurements show statistically significant correlations in males: A1-A2 with B1-B2 and D1-D2 (see table 5.9). Unlike both sets of female measurements, these results reveal a strong association between overall mandibular size (A1-A2), and size of the dental arcade (B1-B2). Furthermore, they reveal that while there is a significant correlation between both measurements relating to mandibular breadth (A1-A2 and D1-D2), this set of measurements is only weakly correlated with those relating to mandibular length.

5.3.5: Inter-sample comparisons of adult mandibular size

A comparison of adult mandibular size presents conflicting results. Spitalfields females are significantly larger than Poundbury females in width of the dental arcade (B1-B2). In contrast, measurements relating to the overall size of the mandible show that Poundbury females are larger (table 5.10). This latter set of measurements, however, is not statistically significant.

This pattern is repeated for males; the dental arcades from the Spitalfields collection are larger, while the overall size of mandibles from the Poundbury collection is larger. The differences are statistically significant at the table-wide level for all measurements relating to the size of the dental arcade, and for D1-C1 (table 5.11).

Adult Females

	N	Mean	SD	*f*	*p*
A1-A2					
Spitalfields	22	115.5	6.87		
Poundbury	24	118.3	5.64	2.290	.137
B1-B2					
Spitalfields	22	57.0	3.10		
Poundbury	24	51.9	4.48	***19.436***	***.000****
D1-C1					
Spitalfields	21	109.7	6.95		
Poundbury	18	113.5	6.42	3.056	.089
B1-C2					
Spitalfields	21	64.3	11.66		
Poundbury	19	57.3	4.69	5.940	.020
D1-D2					
Spitalfields	21	90.1	5.76		
Poundbury	19	92.2	8.79	.830	.368

Adult Males

	N	Mean	SD	*f*	*p*
A1-A2					
Spitalfields	24	122.9	5.59		
Poundbury	24	126.1	6.64	3.226	.079
B1-B2					
Spitalfields	25	58.3	2.59		
Poundbury	24	53.8	3.68	***25.378***	***.000****
D1-C1					
Spitalfields	22	114.7	9.38		
Poundbury	18	127.0	13.43	***11.665***	***.002****
B1-C2					
Spitalfields	25	64.1	4.01		
Poundbury	21	61.0	4.11	***6.457***	***.015****
D1-D2					
Spitalfields	23	98.0	8.89		
Poundbury	19	103.4	9.30	3.740	.060

Table 5.11: Comparison of mandibular size: Spitalfields and Poundbury

** Significant at table-wide level*

5.3.6: Correlation between adult mandibular size and tooth size

Strikingly different patterns of correlation are evident when broken down by sex and sample. In the Spitalfields sample (tables 5.12-13 and figure 5.4), there is a statistically significant correlation between B1-B2 (width of dental arcade) and several teeth. For the females this occurs in all teeth with the exception of the I1 and I2. In males significant correlations between B1-B2 and C and P4 are evident. In contrast, there are no significant correlations between the size of the dental arcade and any teeth in the Poundbury collection (tables 5.14-15 and figure 5.4).

When the association between overall mandibular size and tooth size is examined, however, this pattern is reversed to some extent. There are no statistically significant correlations between overall mandibular size and tooth size in females of either sample.

In the case of Poundbury males, however, overall mandibular size as indicated by bicondylar width is significantly correlated with all teeth in males with the exception of I1 and M2. In contrast, an examination of the Spitalfields males reveals only one significant correlation between overall mandibular size and tooth size. This occurs between A1-A2 and I2.

These results suggest that more dental crowding is present in Poundbury males, since there is a low correlation between tooth size and measurements reflecting the size of the dental arcade. This is in spite of the fact that overall mandibular size is larger in Poundbury (this will be discussed in further detail in section 5.4).

	N	Pearson Correlation	p
A1-A2			
I1	21	-.140	.545
I2	21	.131	.571
C	21	.225	.326
P3	21	.224	.343
P4	21	.245	.298
M1	21	.304	.180
M2	21	*.470*	*.027*
B1-B2			
I1	21	.338	.135
I2	21	.298	.189
C	21	*.619*	*.003* *
P3	21	*.612*	*.004* *
P4	21	*.721*	*.000* *
M1	21	*.595*	*.004* *
M2	21	*.727*	*.000* *

	N	Pearson Correlation	p
D1-C1			
I1	20	-.136	.567
I2	20	-.046	.846
C	20	-.244	.300
P3	20	.152	.535
P4	20	-.116	.638
M1	20	.257	.274
M2	20	.261	.252
B1-C2			
I1	20	.334	.150
I2	20	.378	.100
C	20	.288	.219
P3	20	.359	.131
P4	20	.225	.355
M1	20	.470	.037
M2	20	.541	.011

	N	Pearson Correlation	p
D1-D2			
I1	20	.082	.624
I2	20	.754	.075
C	20	.502	.024
P3	20	.266	.352
P4	20	.020	.936
M1	20	.310	.183
M2	20	.375	.094

Table 5.12:

Correlation between tooth size (mesiodistal diameter) and mandibular size in Spitalfields adult females.

** Significant at table-wide level*

	N	Pearson Correlation	p
A1-A2			
I1	20	.245	.297
I2	22	***.574***	***.005****
C	24	.396	.057
P3	21	.315	.164
P4	24	.321	.126
M1	22	.205	.361
M2	24	.238	.263
B1-B2			
I1	21	.387	.083
I2	23	***.571***	***.004****
C	25	.456	.022
P3	22	***.670***	***.001****
P4	25	.337	.099
M1	23	.259	.234
M2	25	.330	.107

	N	Pearson Correlation	p
D1-C1			
I1	18	.062	.807
I2	20	.031	.895
C	22	.164	.467
P3	19	.090	.714
P4	22	.404	.187
M1	20	.179	.449
M2	22	.096	.671
B1-C2			
I1	21	.239	.297
I2	23	***.531***	***.009***(
C	25	.466	.019
P3	22	.317	.151
P4	25	.259	.212
M1	23	.442	.035
M2	25	.379	.062

	N	Pearson Correlation	p
D1-D2			
I1	19	.052	.833
I2	21	0264	.248
C	23	.159	.468
P3	20	.283	.227
P4	23	.036	.869
M1	21	.014	.950
M2	23	.099	.653

Table 5.13:

Correlation between tooth size (mesiodistal diameter) and mandibular size in Spitalfields adult males.

** Significant at table-wide level*

Table 5.14:

Correlation between tooth size (mesiodistal diameter) and mandibular size in Poundbury adult females.

	N	Pearson Correlation	p
A1-A2			
I1	24	-.109	.612
I2	24	.048	.825
C	24	-.115	.592
P3	24	.352	.091
P4	24	.472	.020
M1	24	.239	.260
M2	24	.343	.101
B1-B2			
I1	24	-.081	.707
I2	24	.104	.630
C	24	-.042	.845
P3	24	.425	.039
P4	24	.040	.854
M1	24	-.017	.936
M2	24	.663	.768

	N	Pearson Correlation	p
D1-C1			
I1	18	-.091	.721
I2	18	.218	3.384
C	18	-.451	.060
P3	18	-.167	.509
P4	18	-.117	.643
M1	18	-.163	.519
M2	18	.006	.983
B1-C2			
I1	19	-.031	.899
I2	19	.220	.364
C	19	-.016	.950
P3	19	-.146	.550
P4	19	-.268	.268
M1	19	-.122	.620
M2	19	-.125	.611

	N	Pearson Correlation	p
D1-D2			
I1	19	-.335	.161
I2	19	.209	.390
C	19	.027	.912
P3	19	.062	.802
P4	19	-.045	.833
M1	19	.234	.336
M2	19	.157	.520

* Significant at table-wide level

Table 5.15:

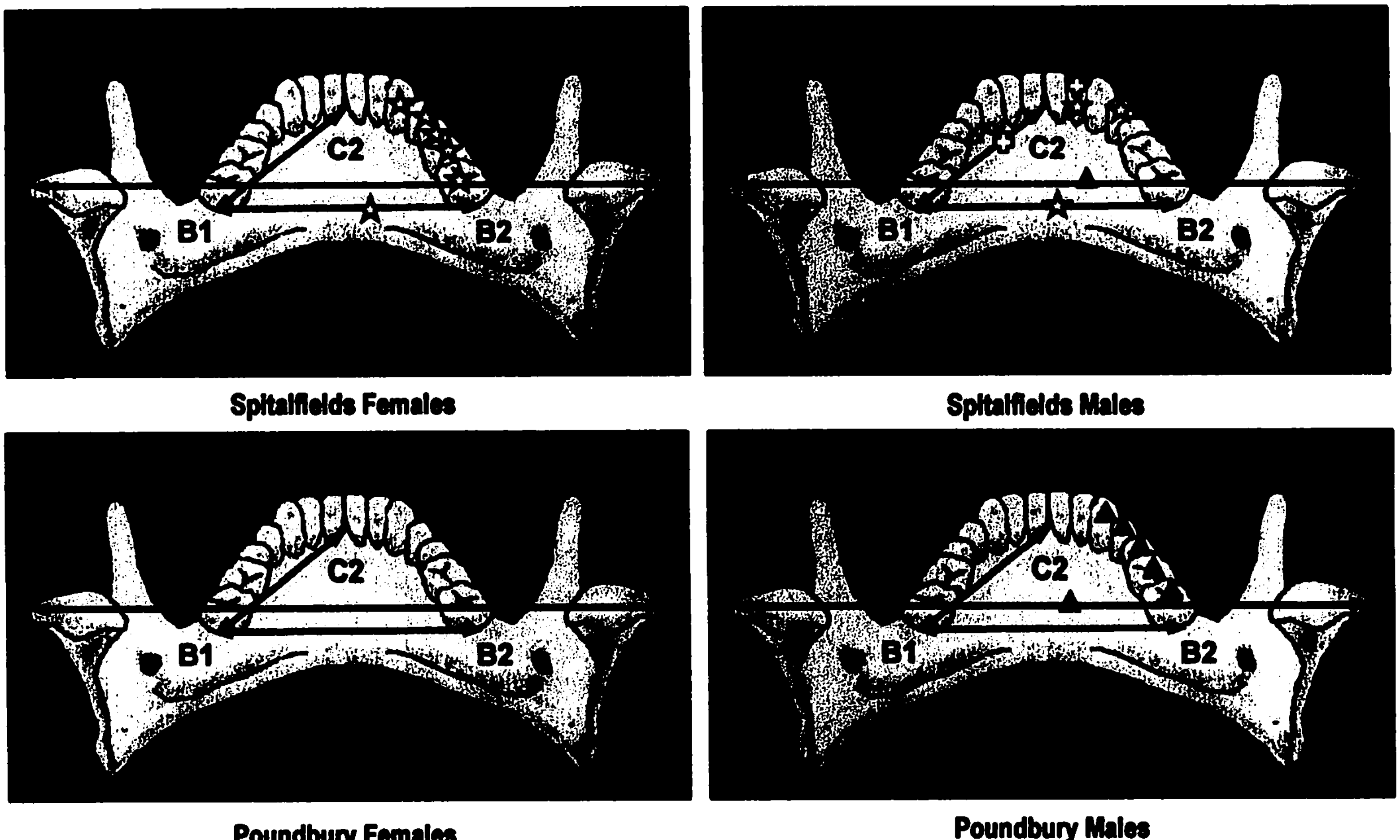
Correlation between tooth size (mesiodistal diameter) and mandibular size in Poundbury adult males.

	N	Pearson Correlation	p
A1-A2			
I1	24	.150	.318
I2	24	.324	.122
C	24	*.547*	*.006* *
P3	24	*.542*	*.006* *
P4	24	*.565*	*.004* *
M1	24	*.544*	*.006* *
M2	24	*.535*	*.007* *
B1-B2			
I1	24	.141	.513
I2	24	.260	.219
C	24	-.041	.850
P3	24	.211	.322
P4	24	.260	.219
M1	24	.258	.223
M2	24	.453	.026

	N	Pearson Correlation	p
D1-C1			
I1	24	-.134	.595
I2	24	.037	.883
C	24	.206	.412
P3	24	.356	.147
P4	24	.404	.097
M1	24	.298	.231
M2	24	.395	.105
B1-C2			
I1	24	.050	.830
I2	24	.084	.718
C	24	.150	.517
P3	24	.041	.859
P4	24	.158	.495
M1	24	.120	.603
M2	24	.255	.265

	N	Pearson Correlation	p
D1-D2			
I1	19	-.022	.928
I2	19	.331	.166
C	19	.396	.093
P3	19	.140	.566
P4	19	.256	.290
M1	19	.278	.250
M2	19	.184	.451

* Significant at table-wide level



(Paired symbols indicate statistically significant correlation between tooth and mandibular measurement)

Figure 5.4: Correlation between tooth size and mandibular size by population and sex

5.3.7: Adult Stature

Comparison of adult stature shows a striking similarity between Spitalfields and Poundbury (table 5.16). Mean female stature of the Spitalfields collection is only 9mm taller than that of Poundbury. In males there is only a 1mm difference. In this case, Poundbury males are slightly taller than Spitalfields males. These differences are not statistically significant in either case.

The stature estimates of both skeletal populations, however, are considerably smaller than the adult stature of the Burlington sample. The mean stature of all children included in the growth study, measured at age 20, is 163.3cm for females and 177.2cm for males (T. Garlie, 1999, *pers. comm.*).

Females

	N	Mean (cm)	SD	*f*	*p*
Spitalfields*	87	156.95	6.32		
Poundbury*	110	156.02	5.68	1.159	.283

Males

	N	Mean (cm)	SD	*f*	*p*
Spitalfields*	68	173.81	10.32		
Poundbury*	156	173.89	7.035	.001	.973

Table 5.16: Inter-sample comparison of stature
calculated after Trotter, 1970

5.3.8: Correlation between tooth size and dental development

An examination of the Spitalfields, Poundbury and Burlington samples revealed no statistically significant associations between tooth size and stage of dental development at the table-wide level (see tables 5.17-5.20).

		n	Mean Mesiodistal diameter	SD	n	P-value (Kendall's tau_b
I1 with M1 at stage 6	I1 stages					
	5	4	5.4	.53	29	.511
	6	16	5.3	.57		
	7	9	5.2	.34		
I2 with I1 at stage 5	I2 Stages					
	4	1	5.9	-	19	.713
	5	18	5.9	.40		
C with M1 at Stage 6	C stages					
	5	4	7.1	.37	14	.357
	6	9	6.7	.39		
	7	1	7.1	-		
P3 with P4 at stage 6	P3 stages					
	5	1	6.6	-	42	.091
	6	27	6.7	.48		
	7	74	7.1	.48		
P4 with P3 at stage 5	P4 stages					
	4	2	7.0	.14	15	.942
	6	12	7.0	.19		
	5	1	7.0	-		
M1 with C at stage 5	M1 stages					
	4	2	11.6	.21	36	.268
	5	10	11.2	.77		
	6	22	11.3	.64		
	7	2	12.2	.71		
M2 with M1 at stage 6	M2 stages					
	3	2	11.4	.28	12	.015
	4	5	11.0	.51		
	5	1	10.7	-		
	6	4	10.0	.67		

Table 5.17: Correlation between permanent tooth size and dental development in the Burlington sample

		n	Mean Mesiodistal diameter	SD	n	P-value (Kendall's tau_b
I1 with M1 at stage 6	I1 stages					
	4	1	5.5	-	10	.897
	5	1	5.0	-		
	6	8	5.3	.50		
I2 with M1 at stage 4	I2 Stages					
	4	2	6.1	.28	10	.364
	5	7	5.6	.45		
	6	1	5.9	-		
C with M1 at Stage 4	C stages					
	3	4	6.6	.55	10	.129
	4	5	7.2	.26		
	5	1	7.2	-		
P3 with P4 at stage 3	P3 stages					
	3	4	6.7	.54	10	.517
	4	6	7.0	.82		
P4 with P3 at stage 4	P4 stages					
	3	6	7.1	.91	11	.855
	4	5	7.1	.91		
M1 with C at stage 4	M1 stages					
	3	4	10.8	.50	14	.163
	4	5	11.3	.60		
	5	4	11.4	.28		
	6	1	11.3	-		
M2 with M1 at stage 7	M2 stages					
	4	1	10.8	-	19	.968
	5	7	10.8	.68		
	6	9	10.7	.56		
	7	2	11.2	.99		

Table 5.18: Correlation between permanent tooth size and dental development in the Poundbury sample

		n	Mean Mesiodistal diameter	SD	n	P-value (Kendall's tau_b
di1 ***with di2 at stage 4***	di1 stages					
	4	5	3.9	.61	11	.043
	5	5	4.2	.15		
	6	1	4.6	-		
di2 ***with di1 at stage 6***	di2 Stages					
	4	1	5.2	-	17	.842
	5	11	4.7	.39		
	6	5	5.1	.95		
dc ***with dm1 at Stage 4***	dc stages					
	3	1	6.5	-	12	.110
	4	11	5.6	.38		
dm1 ***with dm2 at stage 4***	dm1 stages					
	3	4	7.3	.38	18	.263
	4	10	7.8	.37		
	5	4	7.4	.29		
	6	2	7.4	-		
dm2 ***with dm1 at stage 6***	dm2 stages					
	4	3	9.8	.32	15	.914
	5	7	9.6	.56		
	6	5	9.8	.42		

Table 5.19: Correlation between deciduous tooth size and dental development in the Poundbury sample

		n	Mean Mesiodistal diameter	SD	n	P-value (Kendall's tau_b
I1 with C at stage 3	I1 stages					
	3	12	4.9	.29	27	.453
	4	10	5.1	.48		
	5	4	5.1	.48		
	6	1	5.2	-		
I2 with M1 at stage 3	I2 Stages					
	3	14	5.4	-	29	.907
	4	10	5.2	.37		
	5	5	5.3	.48		
C with M1 at Stage 3	C stages					
	3	16	5.8	.68	18	.174
	4	1	6.2	-		
	5	1	5.5	-		
M1 with C at stage 3	M1 stages					
	3	16	10.8	.75	25	.174
	4	6	11.2	.73		
	5	3	11.5	.67		

Table 5.20: Correlation between permanent tooth size and dental development in the Spitalfields sample

		n	Mean Mesiodistal diameter	SD	n	P-value (Kendall's tau_b
di1 *with dc at stage 3*	di1 stages					
	3	1	3.9	-	13	.460
	4	5	3.8	.12		
	5	6	3.9	.13		
	6	1	4.0	-		
di2 *with dc at stage 3*	di2 Stages					
	3	5	4.5	.46	17	.505
	4	10	4.4	.35		
	5	2	4.3	.28		
dc *with dm1 at Stage 4*	dc stages					
	3	1	5.4	-	14	.072
	4	3	5.8	.42		
	5	3	5.5	..52		
	6	7	5.3	.32		
dm1 *with dm2 at stage 3*	dm1 stages					
	3	4	7.3	.38	20	.697
	4	10	7.8	.37		
	5	4	7.4	.29		
	6	2	7.3			
dm2 *with dm1 at stage 6*	dm2 stages					
	3	2	10.1	.99	20	.599
	4	6	9.8	.72		
	5	9	9.9	.49		
	6	8	9.4	.55		

Table 5.21: Correlation between deciduous tooth size and dental development in the Spitalfields sample

5.3.9: Correlation between mandibular size and dental development

The same paired comparisons as those chosen to evaluate the correlation between tooth size and dental development were used in this set of tests. Unfortunately, post mortem damage to some mandibles reduced sample size. This resulted in some teeth (for example both premolars in the Spitalfields sample) being excluded from this analysis.*

	Mandibular Measurements	n	Correlation Coefficient	P (Kendall's Tau-b)
I1 with C at stage 3	A1-A2	19	.080	.672
	B1-B2	19	.206	.281
	D1-C1	18	.328	.183
	B1-C2	18	.145	.456
	D1-D2	18	.339	.082
I2 with M1 at stage 3	A1-A2	19	-.020	.911
	B1-B2	19	.239	.193
	D1-C1	19	-.014	.941
	B1-C2	19	.245	.181
	D1-D2	19	.339	.082
C with M1 at Stage 3	A1-A2	18	.262	.174
	B1-B2	18	.295	.126
	D1-C1	18	.065	.734
	B1-C2	18	.065	.734
	D1-D2	18	-.041	.832
M1 with C at stage 3	A1-A2	18	.304	.116
	B1-B2	18	**.572**	**.003**
	D1-C1	18	.479	.018
	B1-C2	17	.479	.018
	D1-D2	17	.444	.029

Table 5.22: Correlation between mandibular size and development of the permanent dentition in the Spitalfields sample
** Significant at table-wide level*

* As discussed in section, 5.2.1 the entire Burlington sample was excluded due to lack of data for the development of deciduous teeth

	Mandibular Measurements	n	Correlation Coefficient	*P* (Kendall's Tau-b)
di1 with dc at stage 3	A1-A2	9	.149	.624
	B1-B2	9	.075	.806
	D1-C1	9	.000	1.000
	B1-C2	9	-.075	.806
	D1-D2	9	.075	.806
di2 with dc at stage 3	A1-A2	10	.470	.092
	B1-B2	10	.108	.689
	D1-C1	10	.325	.244
	B1-C2	10	.108	.698
	D1-D2	10	.325	.244
dc with dm1 at Stage 4	A1-A2	6	.258	.513
	B1-B2	6	.667	.148
	D1-C1	6	.602	.127
	B1-C2	6	.430	.275
	D1-D2	6	.602	.127
dm1 with dm2 at stage 3	A1-A2	10	.666	.014
	B1-B2	10	.187	.492
	D1-C1	10	.580	.032
	B1-C2	10	.053	.845
	D1-D2	10	.527	.051
dm2 with dm1 at stage 6	A1-A2	14	.445	.045
	B1-B2	14	.366	.098
	D1-C1	14	.403	.068
	B1-C2	14	-.224	.315
	D1-D2	14	.250	.261

Table 5.23: Correlation between mandibular size and development of the deciduous dentition in the Spitalfields sample

	Mandibular Measurements	n	Correlation Coefficient	P (Kendall's Tau-b)
I1 with M1 at stage 6	A1-A2	10	.415	.133
	B1-B2	10	.526	.057
	D1-C1	10	.368	.035
	B1-C2	10	.526	.057
	D1-D2	10	.692	.012
I2 with M1 at stage 4	A1-A2	7	.583	.089
	B1-B2	7	.350	.308
	D1-C1			
	B1-C2			
	D1-D2			
C with M1 at Stage 4	A1-A2	7	***.900***	***.007****
	B1-B2	7	.370	.266
	D1-C1			
	B1-C2			
	D1-D2			
P3 with P4 at stage 3	A1-A2	6	.000	1.000
	B1-B2	6	.365	.355
	D1-C1	6	-.365	.355
	B1-C2	6	-.183	.643
	D1-D2	6	-.365	.355
P4 with P3 at stage 4	A1-A2	6	.000	1.000
	B1-B2	6	.389	.304
	D1-C1	6	.234	.537
	B1-C2	6	.545	.150
	D1-D2	6	.545	.150
M1 with C at stage 4	A1-A2	7	***.839***	***.015****
	B1-B2	7	.436	.196
	D1-C1			
	B1-C2			
	D1-D2			
M2 with M1 at stage 7	A1-A2	13	.446	.061
	B1-B2	13	.216	.366
	D1-C1	7	.056	.869
	B1-C2	7	.056	.869
	D1-D2	7	.169	.672

Table 5.24: Correlation between Mandibular size and development of the permanent dentition in the Poundbury sample

** Significant at table-wide level*

	Mandibular Measurements	n	Correlation Coefficient	P (Kendall's Tau-b)
dm1 *with dm2 at stage 4*	A1-A2	5	-.816	.076
	B1-B2	5	.775	.083
	D1-C1			
	B1-C2			
	D1-D2			
dm2 *with dm1 at stage 6*	A1-A2	7	.597	.078
	B1-B2	7	.159	.634
	D1-C1			
	B1-C2			
	D1-D2			

Table 5.25: Correlation between mandibular size and development of the deciduous dentition in the Poundbury sample

There is no significant correlation between mandibular size and development of the deciduous dentition in Spitalfields (table 5.21). There is however, a correlation between development of M1 and B1-B2 (width of the dental arcade). This occurs when C is held constant at stage 6. There is no association between dental development and any other mandibular measurement in the Spitalfields sample (5.20).

Small sample sizes for the Poundbury sample resulted in the exclusion of di1, di2 and dc from this analysis. Furthermore, post mortem damage to the mandibles of younger individuals limited measurements to A1-A2 and B1-B2. None of the correlations involving deciduous teeth reach levels of significance (table 5.25). There are two significant correlations between mandibular size and development of the permanent dentition in the Poundbury sample, both involving A1-A2 (bi-condylar width). One is with C when M1 is held constant at stage 4, the other with M1 when C is held constant at stage 4.

5.3.10: Summary

A: Differences between adult samples

Comparisons of tooth size show that the adults from the Burlington sample have larger teeth than those from the Spitalfields and Poundbury samples. This difference is more pronounced in males than females. Although differences exist between Spitalfields and Poundbury, they are not statistically significant at the table-wide level.

Mandibular measurements reveal that while mean, overall mandibular size of males and females from Poundbury is larger, the dental arcade size of males and females from *Spitalfields* is larger.

Analysis of correlation between overall mandibular size and measurements reflecting the size of the dental arcade shows an association between overall mandibular size and size of the dental arcade in males from both samples. This association is not evident for females from either sample.

There is a significant correlation between tooth size and measurements associated with the size of the dental arcade in the Spitalfields sample, but not in the Poundbury sample (figure 5.4). This suggests that dental crowding occurs more frequently in the Poundbury sample than in the Spitalfields sample, in spite of the larger overall mandibular size of the former.

There is no significant difference in adult stature between Poundbury and Spitalfields. Burlington adults, however, have a higher mean adult stature. This is evident in both males and females.

B: Differences between sub-adult samples

There are significant differences in sub-adult tooth size between the Spitalfields, Poundbury and Burlington samples. As with the differences in adult tooth size, the Burlington sample has the largest mean sub-adult tooth size. Unlike the differences in adult tooth size, however, there is a difference between the archaeological sub-adult samples, with Poundbury sub-adults showing a larger mean tooth size. The only teeth which are not significantly different are P4 and M2.

C: Differences between adult and sub-adult tooth size

Comparison of adult and sub-adult tooth size, shows that individuals who survive to adulthood have larger teeth than those who die during childhood. This pattern is evident only in Spitalfields suggesting that, while mortality bias may exist in both samples, it is more severe in Spitalfields.

D: Correlation between dental development and tooth size

There is a striking lack of association between tooth size and developmental stage (see tables 5.17-5.21). This is true for Spitalfields, Poundbury and Burlington.

E: Correlation between mandibular size and dental development.

An examination of the association between mandibular size and dental development shows some correlation between overall mandibular size and dental development. This is to be expected since individuals with more advanced dental development are more likely to be more advanced skeletally.

There is no evidence, however of a correlation between measurements reflecting the size of the dental arcade and dental development. This implies that crowding is not a significant factor in the delaying of dental development in either Poundbury or Spitalfields.

5.4: Discussion

5.4.1: Significance of inter-sample differences in adults

Using mesiodistal diameters from 870 seven – eight year old children, Garn and colleagues (1979) found that gestational variables have an effect on mesiodistal crown diameter. He found that children whose mothers had suffered from diabetes and hypothyroidism during pregnancy had high birth weights and went on to develop large teeth, while children whose mothers had suffered from hypertension had low birth weights and went on to develop small teeth. Even crowns which developed post-natally followed this trend.

Furthermore, the secular trend in tooth size discovered by Garn and colleagues (1968) suggests that the increase in somatic growth, due to improved living conditions in western societies has also been accompanied by an increase in tooth size. This notion means that more individuals are now reaching their growth potential in both dental and skeletal growth than was evident in previous generations.

Evidence from adult tooth size found in this study concurs with this hypothesis, since mean adult tooth size of the Burlington sample is larger than that of the Spitalfields and Poundbury samples. Furthermore, the fact that the difference is more pronounced in males than females, provides further supporting evidence, since females are more buffered from environmental stress (DeVito and Saunders, 1990). It is therefore reasonable to expect a greater increase in the tooth size of males as a response to improving environmental conditions.

Differences in adult stature also concur with this hypothesis, since the Burlington sample has a higher mean adult stature than that of either the Spitalfields or Poundbury sample. The results of this study therefore, support the hypothesis that the well documented increase in stature has been accompanied by an increase in tooth size.

Initially, this notion would appear to be at odds with theories concerning human dental reduction over time. Garn and colleagues (1979) and Keiser (1990) have suggested the apparent trend in human dental reduction may be due to the increasing survival of low birth weight babies. This is unlikely, since survival of small neonates is part of an overall reduction in infant mortality, and due largely to advances in western medicine; a relatively recent phenomenon (McVey and Kalbach, 1995). Furthermore, the effects of improved survival of low birth weight babies is probably more than counteracted by an increase in overall birth weight due to improvements in maternal health, and the improved survival of the *larger* than average babies of diabetic mothers.

It must also be stressed that secular trends should not be confused with evolutionary change. As discussed in chapter 2, various ideas have been suggested to account for the decrease in tooth size since the emergence of modern humans. The increase in tooth size discussed in this study, however, is not evidence which can be used to support or refute any of these hypotheses since secular trends increase the *fulfilment* of growth potential, rather than *changes* in growth potential.

The differences in mandibular size evident in this study, however, are of relevance to the debate concerning the mechanisms of dental reduction. As discussed in section 2.3.9, many researchers argue that hard diets result in large, robust jaws due to mechanical stresses caused by chewing, and that conversely, soft diets result in small jaws due to bone resorption. Put simply, this will result in selection in favour of smaller teeth, since these will not be crowded in the smaller mandibles.

Evidence from Poundbury and Spitalfields, however, illustrates that the relationship between overall mandibular size and size of the dental arcade is complex. While mean overall mandibular size is larger for the Poundbury sample, the mean dental arcade size for the *Spitalfields* sample is larger. Since dimensions relating to the dental arcade are the most important mandibular measurements for the study of dental crowding these findings have obvious implications for the debate surrounding human dental reduction. Put simply, the large, robust mandibles of the Poundbury sample are a consequence of a rough diet, while the small, gracile mandibles of the Spitalfields sample are a consequence of a soft, processed diet. The dental arcades of the two samples, however, show a reversal of this, with the sample with the roughest diet exhibiting a small, crowded dental arcade. In this instance, the rough diet and consequently large robust jaw, has not alleviated dental crowding, since the dental arcades have remained small. This suggests that the assumption that dental crowding occurs as a consequence of a soft diet may not always be correct.

Furthermore, the association between tooth size and the dimensions relating to the size of the dental arcade is closer in Spitalfields than Poundbury. This also implies that dental crowding is more evident in the Poundbury sample.

Using data from 16 year olds, Anderson and colleagues (1975) found an association between tooth size, and both overall mandibular size and size of the dental arcade. A further study by Anderson and colleagues (1977) showed an association between tooth size and chin size, when controlled for body weight. The results of these two studies could be used to argue, that under conditions of optimum health and nutrition, these dimensions are closely related. When, however, these environmental conditions are not met, as in the archaeological samples, differential effects of stressors on bone and teeth can result in a lack of association.

5.4.2: Significance of inter-sample differences in sub-adults.

The differences in sub-adult tooth size show striking differences between all three samples. Burlington children have the largest teeth, while Spitalfields children have the smallest. The children in the Poundbury sample lie somewhere between these two extremes. This concurs with the findings of the previous chapter which suggest that the environment conditions of Spitalfields were the poorest, while those of Burlington were the most healthy. The fact that the two archaeological samples show a significant difference in sub-adult tooth size which is not evident in adult tooth size suggests that the sub-adults with the smallest teeth in the Spitalfields sample tended not to survive to adulthood. This is explored in more detail below.

5.4.3: Significance of differences between adults and sub-adults

The evidence from tooth size of adults and sub-adults suggests mortality bias is in operation in both archaeological populations, but is stronger in Spitalfields. Put simply, the mean tooth size of those who survived to adulthood is larger than that of those who died in infancy. Duray (1996) investigated mortality bias in a Prehistoric Native American sample and found that individuals with enamel defects died, on average, five years earlier than those who did not. Several reasons are suggested for this:

A) The same culturally determined environmental stressors result in enamel defects and early mortality

B) Individuals exposed to stress pre-natally or in early childhood are biologically damaged, and are therefore unable to survive later insults

C) Individuals with genetic susceptibility to certain stressors show a pattern of illness which results in enamel defects and early death.

Although Duray's study involved the relationship between enamel defects and early adult mortality, his suggestions can be also be viewed in the context of the relationship between small tooth size and infant mortality

As stated by Garn (1979), small teeth are highly correlated with small birth weight. Although a variety of conditions can result in low birth weight, in Garn's study of affluent, modern American children, maternal hypertension during pregnancy was the most significant factor. Since the vault burials at Spitalfields comprised wealthy individuals, the high cholesterol diet and high alcohol consumption enjoyed by the more affluent portion of 18th-19th century society could well have resulted in high rates of maternal hypertension. Furthermore, the data relating to adult tooth size also suggest that these low birth weight infants did not survive to maturity. These factors support Duray's second hypothesis: that pre-natal stress caused biological damage to some infants, which resulted in small teeth and an inability to cope with stressors in early childhood.

Does this mean that the population of Spitalfields was actually healthier than that of Poundbury, and that these results indicate that only frail children died in the former, whereas healthy *and* frail children died in the latter? This is probably not the case. The two adult samples are similar in both tooth size and stature, which would imply that the factors influencing dental development target infants at specific ages. When other lines of evidence are taken into account, it appears that these factors (poor infrastructure, high pathogen load and poor diet) were more prevalent in Spitalfields than Poundbury. It can therefore be argued that the Spitalfields population produced a high number of low birth weight babies who did not survive to adulthood.

What is apparent, however, is that the sub-adult sample from Spitalfields is not representative of their peer group, the majority of whom had normal size teeth and survived to adulthood.

5.4.4: Correlates of dental development

There is little correlation between mandibular size and developmental stage at death. The only significant correlation between mandibular size and dental development involves development of the M1, and is present in both the Spitalfields and Poundbury samples. This could indicate that developmental retardation of the first molar is related to slow mandibular growth.

There is a distinct lack of correlation between the tooth size and dental development, as assessed by the paired comparisons. At first, this may appear surprising; if small tooth size and dental development are consequences of a detrimental environment, why is there no correlation between these two variables? Garn and colleagues (1965) discovered a slight correlation between dental development, tooth size and nutritional status. In this study of American schoolchildren of European origin, children with a large caloric surplus had larger teeth which developed more quickly. However, Garn and colleagues emphasize that the diet of the children in their study was relatively homogenous and characterized by good nutritional qualities. The children in the two archaeological samples analyzed in this chapter are different in this regard. It is therefore necessary to discuss the severity and type of environmental insult from which the Spitalfields children may have suffered.

The answer may lie in the type of environmental insult. As suggested above, small tooth size may be due to low birth weight, occurring as a consequence of a poor, pre-natal environment. It is possible that dental retardation is occurring as a response to a poor, *post-natal* environment. As discussed in the preceding chapter, the dental retardation evident in the Spitalfields sample appears in individuals living to the latter part of infancy and beyond, and is possibly a consequence of weaning since gastrointestinal disease often proved fatal to older infants during this period of London's history (Mercer, 1990). The evidence therefore suggests that small tooth size occurs as a consequence of poor maternal health, and dental retardation occurs as a consequence of poor health during infancy. Furthermore, there appears to be only a weak correlation between these two factors.

This evidence can be used to argue that the low birth weight children were from the wealthier families, since the diet of the affluent predisposed mothers to hypertension. It can also be argued that the children with growth retardation came from less well off families, and experienced poor conditions in terms of diet and disease during infancy.

5.4.5: Alternative Interpretations

A: Reverse secular trend

An argument can also be made for a hidden, reverse, secular trend in tooth size occurring within the cemetery sample. If this is true, then it could be argued that two separate mechanisms are in operation: one affecting the tooth size of children born during the second half of the cemetery period, and another operating on the *duration* of dental development of children born during the first.

The life spans of individuals buried at Spitalfields occurred over a 200 year period. As discussed in chapter 3, London became increasingly industrialized during this period. This resulted in a growing population, and escalating pressure on the infrastructure of the city (Jordan, 1993). This may well have led to an increasing pathogen load over the period of cemetery use. If this were the case, it can be argued that a reverse secular trend in tooth size occurred during this time, and that the children with small teeth were those who died during the latter part of this period.

While this may be true, the question of what caused dental retardation remains unanswered. Furthermore, it can be argued that a reverse secular trend in tooth size *accompanied* increasing dental retardation, rather than replaced it.

B: Over-representation of male sub-adults.

The teeth of male children develop at a slower rate than those of females (Moorrees *et. al.* 1963a, 1963b). Furthermore, male teeth tend to be larger than female teeth (Devito and Saunders, 1990). Therefore an over-representation of male children in either cemetery could cause considerable bias. For example, a disproportionately large number of male skeletons in the Spitalfields sample could be accounting for the apparent retardation in dental development. Moreover, over representation of males in the Poundbury cemetery could be accounting for the apparently larger tooth size of sub-adults.

Since infant mortality has been found to be higher in males than females, the argument in favour of male over-representation is feasible, and worthy of further discussion. Male bias in Spitalfields can be investigated to some degree by examining the ratio of males to females in the named sample. The male to female ratio in the named sample of 59 sub-adults is 61% males and 39% females. Although this is indicative of male over-representation, it is not sufficient to bias results (Hoppa, pers, comm). Furthermore, when the named sample is broken down into the development categories used for the CEI in chapter 4, it becomes apparent that male bias is moving in the wrong direction. The youngest developmental category (individuals with di1 at stages 3-5) has a sex ratio of 63% males and 37% females, while the following developmental category (individuals with M1 at stages 5-7) has a sex ratio of 40% males and 60% females. Since the dental retardation in the Spitalfields sample does not occur until this latter developmental category, it is unlikely that male bias is the cause. Put simply, if over representation of males was accounting for the retardation, it would be more pronounced in the earliest age category, since male bias is more severe in this instance. The reverse of this situation is true, since it is the second developmental category which contains comparatively more females which shows greatest developmental retardation.

The issue of male over-representation and tooth size can be discounted in the light of these findings. Since the Poundbury sample has the larger mean sub-adult tooth size, but is more advanced in dental development, it is unlikely that difference in sex ratios is the cause. If small tooth size was accompanied by advanced dental development (characteristics of female dentition), in either cemetery, there may be cause for concern, since these two factors are both characteristic of the female dentition. Since this is not the case, the validity of the argument in favour of the results of this study being an artifact of sex bias can be disregarded.

C: Differential responses to environmental stress

Since there is no correlation between dental development and tooth size, it could be argued that the individual can respond to environmental stress in one of two ways; by a reduction in tooth size, or by a slowing in the rate of development. Put simply, normal tooth size may be achieved by an individual developing within an inadequate environment, by a retardation in the rate of development. Alternately, other individuals may respond to environmental stress by continuing to develop their dentitions at a normal rate, but by forming smaller teeth. The evidence from this study suggests this is not the case, since such an "either/or" situation would produce a negative correlation between tooth size and dental retardation. This is not the case, since some small toothed individuals are developmentally retarded while others are not.

5.4.6: Summary

The results of the data analyzed in this chapter can be summarized as follows:

1. *While there is no difference in adult stature or tooth size between the Spitalfields and Poundbury samples, both are smaller than the Burlington sample in both parameters.*

This is evidence supporting the hypothesis that the well documented secular trend in height has been accompanied by a secular trend in tooth size.

2. *There are significant differences in sub-adult tooth size between the Spitalfields, Poundbury and Burlington samples. Burlington children have the largest teeth, while Spitalfields have the smallest. Poundbury lies somewhere between these two extremes.*

Since these results concur with those for dental retardation, this can be used as supporting evidence in favour of the hypothesis that poor environmental conditions had a detrimental effect on the children buried in the vault at Christ church, Spitalfields.

3 *Adult tooth size is larger than sub-adult tooth size in the Spitalfields sample.*

This suggests that there is a mortality bias in favour of small toothed individuals in the Spitalfields sample. It can, therefore, be argued that small tooth size is associated with poor maternal health and consequently poor infant health, since these individuals did not survive to adulthood.

4: *There is no correlation between dental development and tooth size.*

This evidence supports the hypothesis that the mortality samples of subadults comprise two subgroups: one group who had suffered from a poor *pre-natal* environment (possibly as a consequence of maternal hypertension), and a second group who had suffered from a poor *post natal* environment (possibly in the form of high disease load and poor diet).

5: *While there is some correlation between dental development and overall size of the mandible, there is no correlation between dental development and measurements reflecting the size of the dental arcade.*

This suggests that dental development is keeping pace with skeletal development. Furthermore it can be argued that crowding is causing retardation of the first molar.

6: *There is a correlation between tooth size and size of the dental arcade in the Spitalfields sample, but not in the Poundbury sample.*

This supports the hypothesis that, in spite of larger overall mandibular size, the Poundbury sample experienced a greater degree of dental crowding.

Chapter 6
Conclusion

6.1: Introduction

This thesis has established that significant differences in the duration of dental development and in tooth size can exist between population groups of close biological affinity. Furthermore, there is evidence suggesting that environmental stress in the form of pathogen load and poor nutrition is a causal factor.

The implications of these findings are far reaching, and have both biological and anthropological implications. In order to highlight the potential contributions of this research, this chapter places the results of this thesis in the context of different fields of study. Suggestions for further research are also outlined.

6.2: Contributions

6.2.1: Palaeoanthropology

As discussed in chapter 1, there is a great deal of interest surrounding the timing of dental development in extinct hominid species. In order to conduct research in this area, a reasonable standard for modern humans is required for purposes of comparison. Frequently, the developmental standards used are those derived from living children of European origin. More recently, Tompkins (1996), and Harris and McKee (1990) have assessed variability in the timing of human tooth formation between groups of different biological affinity.

The results of this thesis, however, highlight the range of developmental patterns that can exist between groups of *close* biological affinity. The effects of environmental stress on the sub-adult groups of Spitalfields, for example, provide a cautionary tale, highlighting the dangers of making uniformitarian assumptions regarding patterns of dental development. Researchers need to be aware that environmental pressures may also have affected the dental development of the dead, sub-adult, extinct hominid specimens they are examining. Put simply, the sub-adult remains of extinct hominids may have suffered extreme physiological stress, and may, consequently, be displaying patterns of dental development atypical of their species.

The mandibular data from Poundbury and Spitalfields can contribute to the debate surrounding human dental reduction. As discussed in the preceding chapter, the robust mandibles of the Poundbury males are probably a consequence of excessive chewing necessary for the processing of a rough diet. However, the large masticatory muscle attachment sites appear to artificially inflate mandibular measurements, obscuring the correlation between mandibular size and size of the dental arcade. Furthermore, although the overall mandibular size of the Poundbury sample is larger than that of the Spitalfields sample, measurements reflecting the size of the dental arcade. Since tooth size is similar for the two samples, dental crowding is greater in the Poundbury sample in spite of larger overall mandibular size. Some researchers (for example, Calcagno and Gibson, 1988) argue that human dental reduction occurred due to an increasingly soft diet which resulted in smaller jaws with insufficient room for large teeth. The results of this thesis cast doubt on this hypothesis by illustrating that overall mandibular size and size of the dental arcade are not necessarily correlated

6.2.2: The age estimation of subadult skeletons

It is generally accepted that the use of tooth formation stages is the most reliable method of assessing the age of death of subadult skeletons (Moorrees *et al.*, 1963a; Saunders, 1992; Smith, 1991; Ubelaker, 1989). Recently, however, Tompkins (1996) and Owsley and Janz, (1983) have argued that assumptions regarding uniformity in the timing of dental development are unfounded, since significant differences have been discovered between children of European origin, Indigenous African children, and archaeological samples of Native American children.

The results of this thesis show that standards derived from modern Europeans may be inappropriate, even for some *European*, archaeological collections, since the patterns and duration of dental development in the collections investigated in this thesis show significant differences; the differences between Spitalfields and Burlington being most pronounced.

This has important implications for research involving subadult skeletons from archaeological contexts, since palaeodemography, palaeopathology, and studies of human skeletal growth in past populations, all require accurate estimates of age at death. In the absence of information relating to chronological age, tooth formation stages are relied upon to provide this information.

6.2.3: The assessment of health and environment in past populations

The information provided in this thesis can also be of use in research involving investigations into the health and nutrition of past populations. The expanded pattern of dental development in the Spitalfields sample can be used as an indication of environmental stress, while the condensed pattern, as seen in the Burlington sample, can be used as an indication of good nutrition and high standards of health. The analysis of skeletal collections from prehistoric sites can be assessed in the same way in order to glean knowledge about past lifestyles. Tooth size can also be used in a similar way, since diachronic studies of tooth size can be used to detect secular trends.

Furthermore, if adult and subadult tooth sizes from prehistoric collections are compared, the presence of mortality bias can be detected. It has been argued that the growth and development of individuals who die during childhood may be different from those who survive to adulthood. This is because the smallest, frailest children in a population have less chance of reaching adulthood (Wood *et al.* 1992). This appears to be the case in the Spitalfields sample, since those who died during childhood have smaller teeth than those who survived to adulthood.

6.3: Suggestions for future research

6.3.1: Correlation of dental development with long bone lengths and dietary information

Further research is needed into correlates of dental development. As Garn and colleagues (1965) suggest, there is a lack of such studies involving groups of close biological affinity, but from different environments. Archaeological samples may provide suitable material for such studies. Research involving the correlation between dental parameters, long bone lengths, and non-specific stress indicators would provide useful information in this regard. Furthermore, the comparison of the results from this study with information gleaned from dietary reconstruction in the form of chemical analysis, would add a further dimension to this field of research.

6.3.2: Investigation of environmental indicators: Modern studies

In order to understand the relationship between environment and dental development, specific contributing factors need to be established. An investigation of which diseases and nutritional deficiencies have an impact on tooth formation needs to be undertaken. Examination of the timing of dental development in modern clinical samples, for whom medical records exist, would make it possible to explore the associations between developmental patterns, specific diseases, and nutritional deficiencies.

6.3.3: Microscopic study of incremental lines in enamel.

The observation and counting of cross striations, which represent daily increments of enamel formation, can be used to determine the duration of crown development (FitzGerald, 1995). Since this method can be used to determine chronological age, it has great potential for the study of variability in the timing of human tooth formation.

In common with other studies (Tompkins, 1996; Owsley and Jantz, 1983) the research into dental development undertaken in this thesis involved statistical analysis of data derived from radiographs. Although this method has the advantage of speed, some researchers have argued that the interpretation of radiographs is subjective and problematic (see Macho and Wood, 1995). More research needs to be undertaken to assess the reliability of these observations. Comparison of microscopic and radiographic results will test the reliability of this present research. Furthermore, although this study has highlighted different duration and patterns of tooth formation, the exact timing of tooth formation remains unknown. Microscopic analysis could be used to determine the exact timing of developmental events.

Bibliography

Abonyi, S.
1993 *The Effects of Processing on Stable Isotope Levels and Mineral Concentration in Foods: Implications for Paleodietary Reconstruction.* Unpiblished M.A. Thesis: University of Alberta

Anderson, D.L., Thompson, G.W. and Popovich, F.
1977 Tooth, chin, bone and body size correlation *American Journal of Physical Anthropology. 46:* 7-12

Anderson, D.L., Thompson, G.W. and Popovich, F.
1975 Evolutionary dental changes *American Journal of Physical Anthropology. 43:* 95-102

Anderson, T.W. and Sclove, S.L.
1986 *The Statistical Analysis of Data* Palo Alto: The Scientific Press

Armelagos, G.J., Van Gerven, D.P., Goodman, A.H. and Calcagno, J.M.
1989 Post-Pleistocene facial reduction, biomechanics and selection against morphologically complex teeth: A rejoinder to Macchiarelli and Bondioli *Human Evolution. 4:* 1-7

Berkovitz, B.K.B. and Moxham, B.
1981 Early stages in tooth development. In J.W. Osborn (ed) *Dental Anatomy and Emryology:* 246-255 Oxford: Blackwell Scientific Publications

Berry, A. C.
1976 The anthropological value of minor variations of the dental crown. *American Journal of Physical Anthropology. 45:* 257-268

Benyon, A. A. and Wood, B. A.
1987 Patterns and rates of enamel growth in molar teeth in early hominids.
Nature. 328: 493-496

Brace, C. L.
Environment, tooth form and size in the Pleistocene.
Journal of Dental Research. 46: 809-816

Brace, C. L. and Mahler, P. E.
1971 Post-Pleisocene changes in the human dentition.
American Journal of Physical Anthropology. 34: 191-204

Bromage, T. and Dean, M. C.
1986 Re-evaluation of age at death of Plio-Pleistocene fossil hominids.
Nature. 317: 981-983

Butler, P. M.
1971 Growth of human tooth germs.
In A. A. Dahlberg (ed)
Dental Morphology and Human Evolution: 1-155
Chicago University of Chicago Press

Calcagno, J. M.
1986 Dental reduction in Post Pleistocene Nubia.
American Journal of Physical Anthropology. 70: 349-363

Calcagno, J. M. and Gibson, K. R.
1988 Human dental reduction: Natural selection or the probable mutation effect.
American Journal of Physical Anthropology. 77: 505-517

Cavalli-Sforza, L. L. and Bodmer, W. F.
1971 *The Genetics of Human Populations*
San Francisco: W.H. Frieman

C.C.C. (Canadian Council of Churches)
1967 *Satellite City by the Skyway*
Hamilton: Special Publications, Hamilton Public Library

Chavez-Lomeli, E., Mansilla Lory, J. M., Pompa, J. A. and Kjaer, I.
1996 The human mandibular canal arises from three separate canals innervating different tooth groups.
Journal of Dental Research. 75(8): 1540-1544

Christensen, A. F.
1998 Odontometric microevolution in the Valley of Oaxaca, Mexico
Journal of Human Evolution. 34: 333-360

Collingwood, R.G. and Myers, J.N.L.
1949 ***Roman Britain and the English Settlements***
Oxford: Clarendon Press

Collingwood, R.G. and Richmond, I.
1971 ***The Archaeology of Roman Britain***
London: Methuen and Co

Colton, T.
1974 ***Statistics in Medicine***
Boston: Little, Brown and Company

Corruccini, R. S. , Potter, R. H. Y. and Dahlberg, A. A.
1983 Changing occlusal variation in Pima Amerinds
American Journal of Physical Anthropology. 62: 317-324

Crossner C. G. and Mansfield, L.
1983 Determination of dental age in adopted non-European children.
Swedish Dental Journal. 7: 1-10

Dahlberg, A. A.
1971 Penetrance and expressivity of dental traits.
In A.A. Dahlberg (ed)
Dental Morphology and Human Evolution: 257-262
Chicago: University of Chicago press

Daegling, D. J. and Hylander, W. L.
1994 Profiles of strain in the human mandible
Journal of Dental Research. 73: 195

Daegling, D.J. and Hylander, W.L.
1997 Occlusal forces and mandibular bone strain: Is the primate jaw "overdesigned"?
Journal of Human Evolution. 33: 705-717

de Castro, J. M. B., and Nicolas, M.E.
1996 Changes in the lower pre-molar sequence during hominid evolution.
Human Evolution. 11 (3-4): 205-215

Demirjian, A., Goldstein, H. and Tanner, J.M.
1973 ***A new system of dental age assessment***
Human Biology. 45: 211-227

De Vito, C., and Saunders, S.R.
1990 A discriminant function analysis of deciduous teeth to determine sex.
Journal of Forensic Sciences. 35 (4): 845-858

Douglas, M
1966 ***Purity and Danger: an Analysis of the Concepts of Pollution and Taboo.***
London: ARK

Drummond, J.C. and Wilbraham, A.
1991 ***The Englishman's Food: Five Centuries of English Diet.***
London: Pimlico.

Duray, S.M.
1996 Dental indicators of stress and reduced age at death in prehistoric Native Americans.
American Journal of Physical Anthropology. 99: 275-286

Fanning, E.A. and Moorrees, C.F.A.
1969 A comparison of permanent mandibular molar formation in Australian Aborigines and Caucasoids
Archives of Oral Biology. 14: 999-1006

Farrell, D.E. and Molleson, T.I.
1993 ***Poundbury Volume 2: The Cemeteries***
Dorset Natural History and Archaeology Society

FitzGerald, C.M
1995 Tooth Crown Formation and the Variation of Enamel Microstructural Growth Markers in Modern Humans.
Unpublished Ph.D. thesis.
Department of Biological Anthropology, University of Cambridge

Floud, R. and McClosky D.
The Economic History of Great Britain Since 1700
Cambridge: University Press

Freeman, J.V., Cole, T.J., Chinn, S., Jones, P.R.M., White, E.M. and Preece
1995 Cross sectional stature and weight reference curves for the U.K., 1990
Archives of Diseases of Childhood. 73: 17-24

Frend, W.H.C.
1968 The Christianization of Roman Britain
In M.W. Barley and R.P.C. Harrison (eds)
Christianity in Britain, 300-700
Leicester University Press

Garn, S. M. and Kerewesky, R. S.
1968a Relationship between buccolingual and mesiodista; tooth diameters.
Journal of Dental Research. 47: 503

Garn, S.M., Lewis, A.B. and Kerewesky, R.S.
1965 Genetic, nutritional and maturational correlates of dental development
Journal of Dental Research. 37: 561-567

Garn, S.M., Lewis, A.B., and Walenga, A.
1968b Evidence of a secular trend in tooth size over two generations
Journal of Dental Research. 47: 503

Garn, S.M., Lewis, A. B., Swindler, D. R. and Kerewesky, R. S.
1967 Genetic control of sexual dimorphism in tooth size.
Journal of Dental Research. 46: 963-972

Goose, D. H.
1967 Preliminary study of tooth size in families.
Journal of Dental Research. 46: 959-962

Gould, S.J.
1977 *Ontogeny and Phylogeny.*
Cambridge: Harvard University Press

Green, L.J.
1961 The inter-relationship among height, weight and chronological, dental and skeletal ages.
Angle Orthodontist. 31(3): 189-192

Greene, D.L.
1972 Dental anthropology of early Egypt and Nubia.
Journal of Human Evolution. 1: 315-324

Harris, E.F. and McKee, J.H.
1990 Tooth mineralization standards for blacks and whites from the middle southern United States
Journal of Forensic Science. 35: 859-872

Harris, M.
1986 *Good to Eat: Riddles of Food and Culture*
New York: Simon and Schuster

Hanihara, K.
1967 Racial Characteristics in Dentition.
Journal of Dental Research. 46: 923-926

Herring, D.A., Saunders, S. And Boyce, G.
1991 Bones and burial registers: infant mortality in a 19th-century cemetery from Upper Canada
Northeast Historical Archaeology 91:54-67

Hillson, S.
1986 *Teeth.*
Cambridge: Cambridge University Press

Hillson, S.
1996 Dental Anthropology
Cambridge: Cambridge University Press

Holm, S.
1979 A simple sequentially rejective multiple test procedure
Scandinavian Journal of Statistics. 6: 65-70

Holz, R.
1959 The relation of calcification to chronological and skeletal age.
European Orthodontic Society. 1959: 140-149

Hoppa, R.D.
1996 ***Representativeness and Bias in Cemetery Samples: Implications for Palaeodemographic Reconstructions of Past Populations***
Unpublished Ph.D. Thesis, McMaster University

Hunter, W.S. & Priest, W.R.
1960 Errors and discrepencies in measurement of tooth size
Journal of Dental Research. 39: 405-408

Hylander, W. L. and Johnson, K.R.
1997 *In vivo* bone strain patterns in the zygomatic arch of macaques and the significance of these patterns for functional interpretations of craniofacial form
American Journal of Physical Anthropology. 24: 134

Jakobsen, J., Jorgensen, J. B. and Kjaer, I.
1991 Tooth and bone development in a Danish medieval mandible with unilateral absence of the mandibular canal
American Journal of Physical Anthropology. 85: 15-23

Jordan, T.E.
1993 Social change, height, and body mass in Victorian youth, 1805-1914
Annals of Human Biology. 20: 155-181

Kaifu, Y.

1997 Changes in mandibular morphology from Jomon to modern periods in Eastern Japan
American Journal of Physical Anthropology. *104*: 227-243

Keiser. A. K.

1990 *Human Adult Orthodontics: The Study of Variation in Tooth Size.*
Cambridge: Cambridge University Press

Kieser, J.A. and Groeneveld, H.T.

1987 Tooth size and arcadal length correlates in man
International Journal of Anthropology. *2*: 37-46

Knoell, A. C.

1977 A mathematical model of an in vitro human mandible
Journal of Biomechanics. *10*: 159-166

Koski, K.

1996 Mandibular Ramus Flexure-Indicator of Sexual Dimorphism?
American Journal of Physical Anthropology. *101*: 545-546

Largo, R.H.

1993 Catch-Up Growth During Adolescence
Journal of Hormone Research. *39*(3): 41-48

Lampl, M., Monge, J. M. and Mann, A. E.

1993 Further observations on a method of estimating Hominoid dental development patterns.
American Journal of Physical Anthropology. *90*: 113-127

Levi-Strauss, C.

1969 *The Raw and the Cooked*
Transl. J. and D. Weightman
New York: Harper and Row

Lumsden, A. G. S.

1982 The developing innervation of the lower jaw and its relation to the formation of tooth germs in mice embryos.
in B. Kurten (ed)
Teeth: Form, Function and Evolution: 32-43
New York: Columbia University Press

Lundstrom, A.

1948 *Tooth Size and Occlusion in Twins*
New York: S. Krager

Macchiarelli, R. and Bondioli, L.

1986 Post-Pleistocene reductions in human dental structure: A reappraisal in terms of increasing population density
Human Evolution. 1: 405-417

Mann, A., Lampl, M. and Monge, J.

1990 Patterns of ontogeny in human evolution: evidence from dental development.
Yearbook of Physical Anthropology. 33: 111-150

Mann, A. E., Monge, J. M. and Lampl, M.

1991 Investigation into the relationship between perikymata and crown formation times.
American Journal of Physical Anthropology. 86: 175-191

Mayhall, J.T.

1992 Techniques for the study of dental morphology
In Saunders, S.R. and Katzenberg, M.A. (eds)
Skeletal Biology of Past Peoples: Research methods. 59-58

Mayhall, J. T. and Kanazawa, E.

1989 Three-dimensional analysis of the maxillary first molar crowns of Canadian Inuit.
American Journal of Physical Anthropology. 78: 73-78

McKee, J. K.
1984 A genetic model of dental reduction through he probable mutation effect.
American Journal of Physical Anthropology. 65: 231-241

McMinn, R.M.H., Hutchings, R.T., Pegington, J. and Abrahams, P.
1992 *A Colour Atlas of Human Anatomy*
London: Mosby-Yearbook Europe Ltd.

McVey, W.W. and Kalbach, W.E.
1995 *Canadian Population*
Toronto: Nelson Canada

Mercer, A.
1990 *Disease, Mortality and Population in Transition*
Leicester: Leicester university Press

Mincer, H. H., Harris, E. F and Berryman, H. E.
1994 The ABFO study of third molar development as an indicator of chronological age.
Journal of Forensic Sciences. 38: 379-390

Molleson, T., Cox, M., Waldron, A.H. and Whittaker, D.K.
1993 *The Spitalfields Project Volume 2*
York: Council for British Archaeology

Moorrees, C.F.A., Fanning, E.A. and Hunt, E.E.
1963aAge variation of formation stages of ten permanent teeth
Journal of Dental Research 42: 1490-1502

Moorrees, C.F.A., Fanning, E.A. and Hunt, E.E.
1963b Formation and resorption of three deciduous teeth in children
American Journal of Physical Anthropology: 21: 205-213

Moorrees, C.F.A., Thomsen, S.O., Jensen, E. and Yen, P.K.J.
1957 Mesiodistal crown diameters of deciduous and permanent teeth.
Journal of Dental Research. 36: 39-47

Niswander, J. D.
1963 Effects of heredity and environment on the development of dentition.
Journal of Dental Research. 42: 1288-1296

Osborn, J. W.
1981 Developmental Controls.
In J.W. Osborn (ed)
Dental Anatomy and Embryology: 255-260
Oxford: Blackwell

Osborn, J. W.
1975 Tooth replacement: Efficiency, patterns and evolution.
Evolution. 29: 180-186

Osborn, J. W.
1973 The evolution of dentitions.
American Scientist. 61: 548-599

Osborn, J. W.
1970 New approach to Zahnreihen.
Nature. 255: 343-346

Osborne, R. H.
1967 Some genetic problems in interpreting the evolution of human dentition.
Journal of Dental Research. 46: 945-948

Owsley, D. and Jantz, R.
1983 Formation of the permanent dentition in Arikara Indians: timing differences that affect dental age assessments.
American Journal of Physical Anthropology. 61: 467-471

Pelsmaekers, B., Loos, R., Carels, C. , Dermon, C., and Vlietinck, R.
1997 The genetic contribution of dental maturation
Journal of Dental Research. 76: 1337-1340

Perzigian, A. J.
1977 Fluctuating dental asymmetry: variation among skeletal populations. ***American Journal of Physical Anthropology. 47:*** 81-88

Rice, W.R.
1989 Analyzing tables of statistical tests
Evolution. 43: 223-225

Sakashita, R., Inoue, N., Pan, Q. and Zhu, H.
1997 Diet and discrepancy between tooth and jaw size in the Yin-Shang period of China.
American Journal of Physical Anthropology. 103: 497-505

Saunders, S.R., Herring, D.A., Sawchuk, L.A. and Boyce, G.
1995a The nineteenth-century cemetery at St. Thomas' Anglican Church Belleville: skeletal remains, parish records and censuses.
In S.R. Saunders and D.A. Herring (eds)
Grave reflections. Portraying the Past through Cemetery Studies: 93-118
Toronto: Canadian Scholars Press

Saunders, S.R., Herring, D.A. and Boyce, G.
1995b Can skeletal samples actually represent the living populations they come from? The St. Thomas' Cemetery Site, Belleville, Ontario.
In A.L. Grauer
Bodies of Evidence: 69-89
New York: Wiley and Sons

Saunders, S.R.
1992 Subadult Skeletons and Growth Related Studies
In S.R. Saunders and M.A. Katzenberg (eds)
Skeletal Biology of Past Peoples: Research Methods: 1-20
New York: Wiley-Liss Inc.

Saunders, S.R.
1978 The Development and Distribution of Discontinuous Morphological Variation of the Human Infracranial Skeleton.
Archaeological Survey of Canada: Paper 81
Ottawa: National Museums of Canada

Saunders, S.R., Devito, C. and Katzenberg, M.A.
1997 Dental caries in nineteenth century Upper Canada
American Journal of Physical Anthropology. 104: 71-87

Schwartz, J.H. and Langdon, H.L.
1991 Innervation of the human upper primary dentition: Implications for understanding tooth initiation and rethinking growth and eruption patterns.
American Journal of Physical Anthropology. 86: 273-286

Shapiro, H. L.
1939 *Migration and Environment.*
New York: Oxford University press

Smith, B. H.
1994 Patterns of dental development in Homo, Australopithecus, Pan and Gorilla.
American Journal of Physical Anthropology. 94: 307-325

Smith, B.H.
1991 Standards of human tooth formation and dental age assessment
In C. Larsen (ed)
Advances in Dental Anthropology: 143-168
Wiley-Liss Inc

Smith, B.H., Garn, S.M. and Cole, P.E.
1982 Problems of sampling and inference in the study of fluctuating dental asymmetry.
American Journal of Physical Anthropology. 58: 281-289

Smith, B.H. and Tompkins, R.L.
1995 Towards a life history of hominidae
Annual Review of Anthropology. 24: 257-279

Sperber, G.H.
1981 *Craniofacial Embryology.*
Bristol: Wright PSG

Steele, D.G. and Bramblett, C.A.
1988 *The Anatomy and Biology of the Human Skeleton*
Texas: Texas A&M University Press

Stringer, C.B., Dean, M.C. and Martin, R.D.
1990 A comparative study of cranial and dental development within a recent British sample and among Neanderthals
Primate Life History and Evolution: 115-152
New York: Wiley-Liss, Inc

Suarez, B.K.
1974 Neanderthal dental asymmetry and the probable mutation effect.
American journal of Physical Anthropology. 41: 411-416

Taylor, D.T.
1979 Absent workers and lost work hours.
Monthly Labour Review 102: 49-52

Thomas, C.
1981 *Christianity in Roman Britain to AD 500*
London: Batsford

Tollinson, T.D. & Wagner, R.E.
1992 *The Economics of Smoking*
Boston: Kluwer Academic Publisher

Tompkins, R. L.
1996 Human variability in relative dental development.
American Journal of Physical Anthropology. 99: 79-102

Trinkaus, E.
1983 *The Shanidar Neandertals*
New York: Academic Press

Trotter, M.
1970 Estimation of stature from intact limb bones
In Stewart, T.D. (ed)
Personal Identification in Mass Disasters
Washington: Smithsonian Institute

Turner II, C.G.
1967 Dental genetics and microevolution in prehistoric and living Koniag Eskimo.
Journal of Dental Research. 46: 911-917

Ubelaker, D.H.
1989 Human Skeletal Remains: Excavation, Analysis and Interpretation
Washington: Taraxum

Watts, D.
1991 *Christians and Pagans in Roman Britain*
London: Routledge

Ward, S.C.
1991 Taxonomy, palaeobiology and adaptations of the "robust" australopithecines
Journal of Human Evolution. 21: 469-483

Welsch, R.
1981 An interdependence of foodways and architecture: a foodways contrast in the American plains.
In A. Fenton and T. Owen (eds)
Food in Perspective: Proceedings of the Third International Conference on Ethnological Food Research, Cardiff. Wales: 263-376
Edinburgh: John Donald

Wittaker, D.K.
1993 Oral Healthin
T. Molleson and M.Cox (eds)
The Spitalfields Project, volume 2: The Anthgropology, The Middling Sort: 49-61
CBA Research Report 86, Council for British Archaeology.

Wood, B.
1992 Early hominid species and speciation
Journal of Human Evolution. *22:* 351-365

Wood, J. W., Milner, G. R., Harpending, H. C. and Weiss, K. M.
1992 The osteological paradox: problems of inferring health from skeletal samples.
Current Anthropology. *33(4):* 343-370

Wright, S.
1964 Pleiotrophy in the evolution of structural reduction and of dominance.
American Naturalist. *98:* 65-69

Appendix

Distributions of all permanent and deciduous tooth formation presented as percentages.

(Spitalfields, Poundbury, Burlington and Belleville).

Spitalfields

I2 Stages × **I1 Stages**

I2 \ I1	1	2	3	4	5	6	7	*n*
1								
2		40.0	60.0					5
3			96.7	3.3				30
4				72.2	27.8			18
5					75.0	25.0		8
6				10.0		66.7	33.3	6
7								

C Stages × **I1 Stages**

C \ I1	1	2	3	4	5	6	7	*n*
1		20.0	60.0	20.2				5
2	7.7		76.9	7.7	7.7			13
3			45.2	38.7	12.9	3.2		31
4					100			4
5			20.0		20.0	60.0		5
6								
7								

P3 Stages × **I1 Stages**

P3 \ I1	1	2	3	4	5	6	7	*n*
1			76.9		23.1			13
2			12.5	12.5	62.5	12.5		8
3								
4								
5								
6								
7								

Poundbury

I2 Stages × **I1 Stages**

I2 \ I1	1	2	3	4	5	6	7	*n*
1								
2								
3								
4				77.8	22.2			9
5								
6				10.0		80.0	10.0	10
7								

C Stages × **I1 Stages**

C \ I1	1	2	3	4	5	6	7	*n*
1								
2								
3								
4				66.7	16.7	16.7		6
5				20.0	20.0	60.0		5
6						40.0	60.0	5
7								

P3 Stages × **I1 Stages**

P3 \ I1	1	2	3	4	5	6	7	*n*
1								
2								
3				66.7	33.3			6
4								
5								
6						60.0	40.0	5
7								

Burlington

I2 Stages × **I1 Stages**

I2 \ I1	1	2	3	4	5	6	7	*n*
1								
2								
3								
4				92.5	37.5			16
5					75.9	24.1		54
6						53.3	13.7	15
7							100	23

C Stages × **I1 Stages**

C \ I1	1	2	3	4	5	6	7	*n*
1								
2								
3				61.9	38.1			21
4				10.0	90.0			10
5					54.5	36.4	9.1	55
6						3.8	96.2	26
7								

P3 Stages × **I1 Stages**

P3 \ I1	1	2	3	4	5	6	7	*n*
1								
2				66.7	33.3			18
3				3.8	92.3	3.8		26
4					83.3	16.7		12
5					15.0	45.0	40.0	40
6							100	8
7							100	5

Belleville

I2 Stages × **I1 Stages**

I2 \ I1	1	2	3	4	5	6	7	*n*
1								
2								
3			71.4	28.6				7
4				92.3	3.8	3.8		26
5								
6						60.	40.0	5
7								

C Stages × **I1 Stages**

C \ I1	1	2	3	4	5	6	7	*n*
1								
2								
3			8.3	91.7				12
4				70.0	15.0	15.0		20
5								
6						14.3	85.7	7
7								

P3 Stages × **I1 Stages**

P3 \ I1	1	2	3	4	5	6	7	*n*
1								
2								
3				70.0	10.0	20.0		10
4				20.0		80.0		5
5								
6								
7								

Spitalfields

P4 Stages × I1 Stages

	1	2	3	4	5	6	7	n
1			12.5	12.5	62.8	12.5		8
2								
3								
4						60.0	40.0	5
5								
6								
7								

M1 Stages × I1 Stages

	1	2	3	4	5	6	7	n
1		55.6	33.3		11.1			9
2			83.3	16.7				6
3	3.0		48.5	27.3	15.2	6.1		33
4			50.0	12.5	37.5			8
5								
6								
7								

M2 Stages × I1 Stages

	1	2	3	4	5	6	7	n
1								
2								
3								
4					20.0	40.0	40.0	5
5								
6								
7								

Poundbury

P4 Stages × I1 Stages

	1	2	3	4	5	6	7	n
1								
2								
3								
4								
5								
6								
7								

M1 Stages × I1 Stages

	1	2	3	4	5	6	7	n
1								
2								
3								
4				77.8	11.1		11.1	9
5								
6				10.0	10.0	80.0		10
7								

M2 Stages × I1 Stages

	1	2	3	4	5	6	7	n
1								
2								
3				60.0	40.0			5
4						100		5
5								
6								
7								

Burlington

P4 Stages × I1 Stages

	1	2	3	4	5	6	7	n
1				66.7	33.3			12
2				16.7	83.3			12
3					88.9	11.1		27
4					45.5	54.5		11
5					6.1	36.4	57.6	33
6							100	9
7								

M1 Stages × I1 Stages

	1	2	3	4	5	6	7	n
1								
2								
3				60.0	40.0			10
4				36.4	63.6			22
5					84.6	15.4		26
6					19.4	51.6	29.0	31
7							100	12

M2 Stages × I1 Stages

	1	2	3	4	5	6	7	n
1				50.0	50.0			8
2					100			9
3					67.7	29.0	3.2	31
4					6.5	38.7	54.8	31
5							100	6
6								
7								

Belleville

P4 Stages × I1 Stages

	1	2	3	4	5	6	7	n
1								
2								
3								
4						60.0	40.0	5
5								
6								
7								

M1 Stages × I1 Stages

	1	2	3	4	5	6	7	n
1								
2								
3			27.3	63.6	9.1			11
4				81.8	9.1	9.1		22
5								
6						60.0	40.0	5
7								

M2 Stages × I1 Stages

	1	2	3	4	5	6	7	n
1								
2								
3								
4						33.3	66.7	6
5								
6								
7								

Spitalfields

di1 Stages (rows) × I1 Stages (columns)

	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6	16.7	16.7	50.0	16.7				6
7			81.8	9.1	9.1			11

di2 Stages (rows) × I1 Stages (columns)

	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5		60.0	40.0					5
6	11.14		66.7	22.2				9
7			100					7

dc Stages (rows) × I1 Stages (columns)

	1	2	3	4	5	6	7	*n*
1								
2								
3		57.1	28.6			14.3		7
4		16.7	66.7	16.7				6
5	20.0		80.0					5
6			58.3	41.7				12
7								

Poundbury

di1 Stages (rows) × I1 Stages (columns)

	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6								
7								

di2 Stages (rows) × I1 Stages (columns)

	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6								
7								

dc Stages (rows) × I1 Stages (columns)

	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6								
7								

Burlington

di1 Stages (rows) × I1 Stages (columns)

	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6								
7								

di2 Stages (rows) × I1 Stages (columns)

	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6								
7								

dc Stages (rows) × I1 Stages (columns)

	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6								
7								

Belleville

di1 Stages (rows) × I1 Stages (columns)

	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6		22.2	66.7	11.1				9
7			32.1	69.2	7.7			13

di2 Stages (rows) × I1 Stages (columns)

	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6		8.3	75.0	16.7				12
7				92.9	7.1			14

dc Stages (rows) × I1 Stages (columns)

	1	2	3	4	5	6	7	*n*
1								
2								
3								
4		16.7	75.0	8.3				12
5								
6				94.1	5.9			17
7				71.4	14.3	14.3		14

Spitalfields

dm1 Stages (rows) × I1 Stages (columns)

	1	2	3	4	5	6	7	*n*
1								
2								
3								
4		57.1	28.6			14.3		7
5		16.7	83.3					6
6			80.0	20.0				15
7	8.3		41.7	41.7	8.3			12

dm2 Stages (rows) × I1 Stages (columns)

	1	2	3	4	5	6	7	*n*
1								
2								
3		62.5	37.5					8
4			100					6
5	7.1		50.0	28.6	7.1	7.1		14
6			68.8	25.0	6.3			16
7			57.1	14.3	28.6			7

I1 Stages (rows) × I2 Stages (columns)

	1	2	3	4	5	6	7	*n*
1								
2								
3		9.4	90.6					32
4			7.1	92.9				14
5				45.5	54.5			11
6					33.3	66.7		6
7								

Poundbury

dm1 Stages (rows) × I1 Stages (columns)

	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6								
7								

dm2 Stages (rows) × I1 Stages (columns)

	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6								
7								

I1 Stages (rows) × I2 Stages (columns)

	1	2	3	4	5	6	7	*n*
1								
2								
3								
4			11.1	77.8		11.1		9
5								
6						100		8
7						20.0	80.0	5

Burlington

dm1 Stages (rows) × I1 Stages (columns)

	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6								
7								

dm2 Stages (rows) × I1 Stages (columns)

	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6								
7								

I1 Stages (rows) × I2 Stages (columns)

	1	2	3	4	5	6	7	*n*
1								
2								
3								
4			28.6	71.4				14
5				12.8	87.2			47
6					61.9	38.1		21
7						23.3	76.7	30

Belleville

dm1 Stages (rows) × I1 Stages (columns)

	1	2	3	4	5	6	7	*n*
1								
2								
3								
4		40.0	60.0					5
5			83.3	16.7				6
6			5.9	94.1				17
7				76.9	15.4	7.7		13

I2 Stages (rows) × I1 Stages (columns)

	1	2	3	4	5	6	7	*n*
1								
2								
3								
4		7.7	69.2	23.1				13
5				100				12
6				75.0	18.8	6.3		16
7				83.3		16.7		6

I1 Stages (rows) × I2 Stages (columns)

	1	2	3	4	5	6	7	*n*
1								
2								
3		37.5	62.5					8
4			7.7	92.3				26
5								
6				20.0	20.0	30.0		5
7								

Spitalfields

C Stages × I2 Stages

C Stages	1	2	3	4	5	6	7	n
1		50.0	50.0					6
2	7.78	7.7	69.2	7.7	7.7			13
3			48.5	45.5	6.1			33
4								
5			20.0		20.2	60.0		5
6						52.9	57.1	7
7								

P3 Stages × I2 Stages

P3 Stages	1	2	3	4	5	6	7	n
1			76.9	15.4	7.7			13
2			12.5	25.0	62.5			8
3								
4								
5						66.7	33.3	6
6								
7								

P4 Stages × I2 Stages

P4 Stages	1	2	3	4	5	6	7	n
1			12.5	25.0	62.5			8
2								
3								
4						100		5
5								
6								
7								

Poundbury

C Stages × I2 Stages

C Stages	1	2	3	4	5	6	7	n
1								
2								
3								
4				60.0	30.0	10.0		10
5								
6								
7								

P3 Stages × I2 Stages

P3 Stages	1	2	3	4	5	6	7	n
1								
2								
3								
4				16.7	33.3	50.0		6
5								
6						42.9	57.1	7
7								

P4 Stages × I2 Stages

P4 Stages	1	2	3	4	5	6	7	n
1								
2								
3								
4								
5								
6								
7								

Burlington

C Stages × I2 Stages

C Stages	1	2	3	4	5	6	7	n
1								
2								
3			20.0	70.0	10.0			20
4				10.0	90.0			10
5					75.4	15.8	8.8	57
6						20.0	80.0	35
7								

P3 Stages × I2 Stages

P3 Stages	1	2	3	4	5	6	7	n
1								
2			22.2	66.7	11.1			18
3					100			24
4					100			12
5					34.0	31.9	34.0	47
6						7.7	92.3	13
7							100	5

P4 Stages × I2 Stages

P4 Stages	1	2	3	4	5	6	7	n
1			25.0	75.0				12
2				25.0	75.0			12
3					100			27
4					83.3	16.7		12
5					16.7	33.3	50.0	42
6							100	11
7								

Belleville

C Stages × I2 Stages

C Stages	1	2	3	4	5	6	7	n
1								
2								
3			25.0	75.0				12
4				80.0	12.0	8.0		25
5								
6						55.6	44.0	9
7								

P3 Stages × I2 Stages

P3 Stages	1	2	3	4	5	6	7	n
1								
2								
3				77.8	22.2			9
4				28.6		71.4		7
5								
6								
7								

P4 Stages × I2 Stages

P4 Stages	1	2	3	4	5	6	7	n
1								
2								
3								
4						83.3	16.7	6
5								
6								
7								

Spitalfields

M1 Stages \ I2 Stages	1	2	3	4	5	6	7	*n*
1		60.0	20.0	20.0				5
2		28.6	57.1	14.3				7
3	3.3	3.0	48.5	30.3	15.2			33
4			60.0	10.0	30.0			10
5								
6						83.3	16.7	6
7								

M2 Stages \ I2 Stages	1	2	3	4	5	6	7	*n*
1								
2								
3								
4				16.7		66.6	16.7	6
5								
6								
7								

di1 Stages \ I2 Stages	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6								
7		9.1	72.7	9.1	9.1			11

Poundbury

M1 Stages \ I2 Stages	1	2	3	4	5	6	7	*n*
1								
2								
3								
4			22.2	77.8				9
5				40.0	40.0	20.0		5
6						88.9	11.1	9
7					20.0	20.0	60.0	5

M2 Stages \ I2 Stages	1	2	3	4	5	6	7	*n*
1								
2								
3				40.0	40.0	20.0		5
4								
5						100		6
6								
7								

di1 Stages \ I2 Stages	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6								
7								

Burlington

M1 Stages \ I2 Stages	1	2	3	4	5	6	7	*n*
1								
2								
3			20.0	80.0				10
4			9.1	36.4	54.5			26
5					100			26
6					44.1	35.3	20.6	34
7						18.8	81.3	16

M2 Stages \ I2 Stages	1	2	3	4	5	6	7	*n*
1				62.5	37.5			8
2					100			9
3					93.5	6.5		31
4					23.1	35.9	41.0	39
5							100	10
6							100	7
7								

di1 Stages \ I2 Stages	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6								
7								

Belleville

M1 Stages \ I2 Stages	1	2	3	4	5	6	7	*n*
1								
2								
3			44.4	44.4	11.1			9
4				84.6	7.7			26
5								
6				16.7		66.7	16.7	6
7								

M2 Stages \ I2 Stages	1	2	3	4	5	6	7	*n*
1								
2								
3								
4						62.5	37.5	8
5								
6								
7								

di1 Stages \ I2 Stages	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6		71.4	26.6					
7			36.4	54.5	9.1			

Spitalfields

dl2 Stages × I2 Stages

dl2 Stages	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6	10.0	10.0	70.0	10.0				10
7			14.3	85.7				7

dc Stages × I2 Stages

dc Stages	1	2	3	4	5	6	7	*n*
1								
2								
3								
4		33.3	66.7					6
5	20.0		80.0					5
6			50.0	50.0				12
7			60.0	30.0	10.0			10

dm1 Stages × I2Stages

dm1 Stages	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5		33.3	66.7					6
6		6.3	75.0	12.5	6.3			16
7	8.3		41.7	41.7	8.3			12

Poundbury

dl2 Stages × I2 Stages

dl2 Stages	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6								
7								

dc Stages × I2 Stages

dc Stages	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6								
7								

dm1 Stages × I2Stages

dm1 Stages	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6								
7								

Burlington

dl2 Stages × I2 Stages

dl2 Stages	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6								
7								

dc Stages × I2 Stages

dc Stages	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6								
7								

dm1 Stages × I2Stages

dm1 Stages	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6								
7								

Belleville

dl2 Stages × I2 Stages

dl2 Stages	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6		44.4	44.4	11.1				9
7			7.7	84.6	7.7			13

dc Stages × I2 Stages

dc Stages	1	2	3	4	5	6	7	*n*
1								
2								
3								
4		50.0	50.0					10
5								
6			12.5	81.3	6.3			16
7				85.7	7.1	7.1		14

dm1 Stages × I2Stages

dm1 Stages	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5		16.7	83.3					5
6			21.4	78.6				6
7				93.8	6.3			14

Spitalfields

dm2 Stages × I2 Stages

	1	2	3	4	5	6	7	*n*
1								
2								
3		80.0	20.0					5
4		16.7	83.3					6
5	7.7		53.8	23.1	15.4			13
6			70.6	29.4				17
7			50.0	37.5	12.5			8

I1 Stages × C Stages

	1	2	3	4	5	6	7	*n*
1								
2								
3	10.7	35.7	50.0		3.6			28
4	7.1	7.1	85.7					14
5		10.0	40.0	40.0	10.0			10
6			20.0		60.0	20.0		5
7								

I2 Stages × C Stages

	1	2	3	4	5	6	7	*n*
1								
2								
3	10.3	31.0	55.2		3.4			29
4		6.3	93.8					16
5								
6					50.0	50.0		6
7								

Poundbury

dm2 Stages × I2 Stages

	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6								
7								

I1 Stages × C Stages

	1	2	3	4	5	6	7	*n*
1								
2								
3								
4			16.7	66.7	16.7			6
5			20.0	20.0	60.0			5
6			40.0	20.0		40.0		5
7								

I2 Stages × C Stages

	1	2	3	4	5	6	7	*n*
1								
2								
3								
4			25.0	75.0				8
5								
6				12.5	50.0	37.5		8
7						60.0	40.0	5

Burlington

dm2 Stages × I2 Stages

	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6								
7								

I1 Stages × C Stages

	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5			92.9	7.1	65.2			46
6					95.2	4.8		21
7					16.7	83.3		30

I2 Stages × C Stages

	1	2	3	4	5	6	7	*n*
1								
2								
3								
4			93.8	6.3				16
5			3.7	16.7	79.6			54
6					56.3	43.8		16
7					14.7	82.4	2.6	34

Belleville

dm2 Stages × I2 Stages

	1	2	3	4	5	6	7	*n*
1								
2								
3								
4		41.7	50.0	8.3				12
5			30.0	70.0				10
6				84.2	15.8			19
7				71.4		28.6		7

I1 Stages × C Stages

	1	2	3	4	5	6	7	*n*
1								
2								
3								
4			44.0	56.0				25
5								
6				50.0	33.3	16.7		6
7						100		6

I2 Stages × C Stages

	1	2	3	4	5	6	7	*n*
1								
2								
3		40.0	60.0					5
4			31.0	69.0				29
5								
6				22.2	22.2	55.6		9
7								

Spitalfields

P3 Stages × C Stages

P3	1	2	3	4	5	6	7	*n*
1		7.7	76.9	15.4				13
2			62.5	25.0	12.5			8
3								
4								
5					12.5	87.5		8
6						80.0	20.0	5
7								

P4 Stages × C Stages

P4	1	2	3	4	5	6	7	*n*
1			50.0	50.0				8
2								
3								
4					40.0	60.0		5
5								
6						80.0	20.0	5
7								

M1 Stages × C Stages

M1	1	2	3	4	5	6	7	*n*
1								
2	40.0	40.0			20.0			5
3	6.5	32.3	54.8	3.2	3.2			31
4			70.0	30.0				10
5								
6					42.9	57.1		7
7								

Poundbury

P3 Stages × C Stages

P3	1	2	3	4	5	6	7	*n*
1								
2								
3				71.4	28.6			7
4				33.3	55.6	11.1		9
5								
6						77.8	22.2	9
7						28.6	71.4	7

P4 Stages × C Stages

P4	1	2	3	4	5	6	7	*n*
1								
2								
3				42.9	42.9	14.3		7
4								
5					14.3	71.4	14.3	7
6						33.3	66.7	6
7								

M1 Stages × C Stages

M1	1	2	3	4	5	6	7	*n*
1								
2								
3		14.3	14.3	71.4				7
4			36.4	45.5	9.1	9.1		11
5				66.7	33.3			6
6			9.1	9.1	54.5	18.2	9.1	11
7						69.2	30.8	13

Burlington

P3 Stages × C Stages

P3	1	2	3	4	5	6	7	*n*
1								
2			83.3	11.1	5.6			18
3			15.4	30.8	53.8			26
4					100			12
5					59.6	40.4		52
6					2.4	88.1	9.5	42
7						42.4	57.6	33

P4 Stages × C Stages

P4	1	2	3	4	5	6	7	*n*
1			100					12
2			33.3	41.7	25.0			12
3				14.8	85.2			27
4					91.7	8.3		12
5					43.1	54.9	2.0	51
6						78.3	21.7	46
7						47.6	52.4	21

M1 Stages × C Stages

M1	1	2	3	4	5	6	7	*n*
1								
2								
3			100					10
4			50.0	31.8	18.2			22
5				11.5	88.5			26
6					62.5	35.0	2.5	40
7					5.3	81.6	13.2	38

Belleville

P3 Stages × C Stages

P3	1	2	3	4	5	6	7	*n*
1								
2								
3			12.5	75.0	12.5			16
4				82.9	29.4	17.6		17
5					16.7	83.3		6
6					9.1	72.7	18.2	11
7							100	7

P4 Stages × C Stages

P4	1	2	3	4	5	6	7	*n*
1								
2								
3				77.8	22.2			9
4				28.6	28.6	42.9		14
5					14.3	71.4	14.3	7
6						42.9	57.1	7
7							100	6

M1 Stages × C Stages

M1	1	2	3	4	5	6	7	*n*
1								
2								
3		25.0	50.0	25.0				16
4			39.3	57.1	3.6			28
5				77.8	22.2			9
6				28.6	28.6	42.9		14
7					13.3	60.0	26.7	15

Spitalfields

M2 Stages × **C Stages**

M2 Stages	1	2	3	4	5	6	7	*n*
1								
2								
3								
4					20.0	80.0		5
5								
6								
7								

di1 Stages × **C Stages**

di1 Stages	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6	60.0	40.0						5
7		50.0	40.0		10.0			10

di2 Stages × **C Stages**

di2 Stages	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6	40.0	40.0	10.0		10.0			10
7		50.0	50.0					6

Poundbury

M2 Stages × **C Stages**

M2 Stages	1	2	3	4	5	6	7	*n*
1								
2								
3				42.9	57.1			7
4								
5						100		7
6						33.3	66.7	6
7								

di1 Stages × **C Stages**

di1 Stages	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6								
7								

di2 Stages × **C Stages**

di2 Stages	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6								
7								

Burlington

M2 Stages × **C Stages**

M2 Stages	1	2	3	4	5	6	7	*n*
1			62.5	25.0	12.5			8
2			22.2	22.2	55.6			9
3				6.5	90.3	3.2		31
4					52.4	47.6		42
5					6.7	83.3	10.0	30
6						63.0	37.0	46
7						28.6	71.4	7

di1 Stages × **C Stages**

di1 Stages	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6								
7								

di2 Stages × **C Stages**

di2 Stages	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6								
7								

Belleville

M2 Stages × **C Stages**

M2 Stages	1	2	3	4	5	6	7	*n*
1								
2								
3				70.0	10.0	20.0		10
4				11.8	17.6	64.7	5.9	17
5					14.3	42.9	42.9	7
6								
7								

di1 Stages × **C Stages**

di1 Stages	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6								
7		11.1	55.6	33.3				9

di2 Stages × **C Stages**

di2 Stages	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6			50.0	20.0				5
7			53.8	46.2				13

Spitalfields

I1 Stages × P3 Stages

	1	2	3	4	5	6	7	*n*
1								
2								
3	83.3	8.3	8.3					12
4								
5	33.3	55.6	11.1					9
6		20.0		40.0	40.0			5
7								

C Stages × P3 Stages

	1	2	3	4	5	6	7	*n*
1								
2								
3	83.3	8.3	8.3					12
4	28.6	28.6	42.9					7
5	16.7	83.3						6
6				33.3	66.7			6
7								

C Stages × P3 Stages

	1	2	3	4	5	6	7	*n*
1								
2								
3	55.6	27.8	16.7					18
4								
5								
6					63.6	36.4		11
7								

Poundbury

I1 Stages × P3 Stages

	1	2	3	4	5	6	7	*n*
1								
2								
3								
4			66.7	33.3				6
5								
6			16.7	33.3		50.0		6
7					40.0	40.0	20.0	5

I2 Stages × P3 Stages

	1	2	3	4	5	6	7	*n*
1								
2								
3								
4		33.3	50.0	16.7				15
5								55
6			12.5	37.5	12.5	37.5		16
7					16.7	66.7	16.7	32

C Stages × P3 Stages

	1	2	3	4	5	6	7	*n*
1								
2								
3								
4		27.3	45.5	27.3				11
5			33.3	66.7				6
6				8.3	16.7	58.3	16.7	12
7						28.6	71.4	7

Burlington

I1 Stages × P3 Stages

	1	2	3	4	5	6	7	*n*
1								
2								
3								
4	7.1	85.7	7.1					14
5		13.0	52.2	21.7	13.0			46
6			4.8	9.5	85.7			21
7					57.1	28.6	14.3	28

I2 Stages × P3 Stages

	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6								
7								

C Stages × P3 Stages

	1	2	3	4	5	6	7	*n*
1								
2								
3	5.0	75.0	20.0					20
4		20.0	80.0					10
5		1.7	23.7	20.3	52.5	1.7		59
6					29.2	51.4	19.4	72
7						13.6	86.4	22

Belleville

I1 Stages × P3 Stages

	1	2	3	4	5	6	7	*n*
1								
2								
3								
4		11.1	77.8	11.1				
5								
6			33.3	66.7				
7					50.0	50.0		

I2 Stages × P3 Stages

	1	2	3	4	5	6	7	*n*
1								
2								
3								
4		10.0	70.0	20.0				10
5								
6				55.6	22.2	22.2		9
7								

C Stages × P3 Stages

	1	2	3	4	5	6	7	*n*
1								
2								
3			100					12
4		12.5	50.0	37.5				25
5								
6				18.8	31.3	50.0		9
7								

Spitalfields

P4 Stages × P3 Stages

P4 \ P3	1	2	3	4	5	6	7	n
1	37.5	62.5						8
2								
3								
4				20.0	80.0			5
5								
6						85.7	14.3	7
7								

M1 Stages × P3 Stages

M1 \ P3	1	2	3	4	5	6	7	n
1								
2								
3	66.7	33.3						
4	66.7	33.3						
5								
6				28.6	71.4			
7								

M2 Stages × P3 Stages

M2 \ P3	1	2	3	4	5	6	7	n
1								
2								
3								
4			16.7		83.3			6
5								
6						100		5
7								

Poundbury

P4 Stages × P3 Stages

P4 \ P3	1	2	3	4	5	6	7	n
1								
2								
3			40.0	60.0				10
4				100				5
5						100		5
6						37.5	62.5	8
7								

M1 Stages × P3 Stages

M1 \ P3	1	2	3	4	5	6	7	n
1								
2								
3								
4		12.5	75.0		12.5			8
5		9.1	45.5	45.5				11
6				66.7	8.3	25.0		12
7					7.1	50.0	42.9	14

M2 Stages × P3 Stages

M2 \ P3	1	2	3	4	5	6	7	n
1								
2								
3		10.0		90.0				10
4			16.7	50.0		33.3		6
5					22.2	66.7	11.1	9
6						28.6	71.4	7
7						20.0	80.0	5

Burlington

P4 Stages × P3 Stages

P4 \ P3	1	2	3	4	5	6	7	n
1	8.3	91.7						12
2		8.3	91.7					12
3		3.7	51.9	40.7	3.7			27
4			8.3	8.3	83.3			12
5					80.0	20.0		50
6					2.2	66.7	31.3	45
7						5.0	95.0	20

M1 Stages × P3 Stages

M1 \ P3	1	2	3	4	5	6	7	n
1								
2								
3	11.1	88.9						9
4		40.9	59.1					22
5		3.8	49.2	34.6	15.4			26
6				7.5	75.0	12.5	5.0	40
7					30.8	46.2	23.1	39

M2 Stages × P3 Stages

M2 \ P3	1	2	3	4	5	6	7	n
1		62.5	37.5					8
2			100					9
3		2.2	29.0	32.3	35.5			31
4				4.8	83.3	11.9		42
5					17.9	714	10.7	28
6					2.3	37.2	60.5	43
7						100		5

Belleville

IP4 Stages × P3 Stages

IP4 \ P3	1	2	3	4	5	6	7	n
1								
2								
3			50.0	50.0				10
4				61.5	30.8	7.7		13
5				10.0	30.0	50.0	10.0	10
6						66.7	33.3	6
7							100	7

M1 Stages × P3 Stages

M1 \ P3	1	2	3	4	5	6	7	n
1								
2								
3								
4	7.7	23.1	61.5	7.7				13
5			60.0	40.0				10
6				73.3	6.7	20.0		15
7					33.3	44.4	22.2	18

M2 Stages × P3 Stages

M2 \ P3	1	2	3	4	5	6	7	n
1								
2								
3			11.1	77.8	11.1			9
4				31.6	21.1	42.1	5.3	19
5					28.6	57.1	14.3	7
6								
7								

Spitalfields

I1 Stages × P4 Stages

I1 Stages	1	2	3	4	5	6	7	n
1								
2								
3								
4								
5	83.3	16.7						6
6	20.0		20.0	60.0				5
7								

I2 Stages × P4 Stages

I2 Stages	1	2	3	4	5	6	7	n
1								
2								
3								
4	40.0	20.0	40.0					5
5	100							5
6			16.7	83.3				6
7								

C Stages × P4 Stages

C Stages	1	2	3	4	5	6	7	n
1								
2								
3	50.0	16.7	33.3					6
4								
5								
6			9.1	27.3	27.3	36.4		11
7								

Poundbury

I1 Stages × P4 Stages

I1 Stages	1	2	3	4	5	6	7	n
1								
2								
3								
4								
5								
6			20.0	40.0	40.0			5
7								

I2 Stages × P4 Stages

I2 Stages	1	2	3	4	5	6	7	n
1								
2								
3								
4								
5								
6				60.0	40.0			5
7								

C Stages × P4 Stages

C Stages	1	2	3	4	5	6	7	n
1								
2								
3								
4	16.7	16.7	50.0	16.7				6
5			42.9	42.9	14.3			7
6					55.6	22.2	11.1	9
7					16.7	66.7	16.7	6

Burlington

I1 Stages × P4 Stages

I1 Stages	1	2	3	4	5	6	7	n
1								
2								
3								
4	80.0	20.0						
5	8.9	22.2	53.3	11.1	4.4			
6			14.3	28.6	57.1			21
7					63.3	30.0	6.7	30

I2 Stages × P4 Stages

I2 Stages	1	2	3	4	5	6	7	n
1								
2								
3								
4	75.0	25.0						12
5		17.0	50.9	18.9	13.2			53
6				12.5	87.5			16
7					61.8	32.4	5.9	34

C Stages × P4 Stages

C Stages	1	2	3	4	5	6	7	n
1								
2								
3	75.0	25.0						16
4		55.6	44.4					9
5			41.1	19.6	39.3			56
6				1.3	36.8	48.7	13.2	76
7								

Belleville

I1 Stages × P4 Stages

I1 Stages	1	2	3	4	5	6	7	n
1								
2								
3								
4								
5								
6								
7				40.0	40.0	20.0		10

I2 Stages × P4 Stages

I2 Stages	1	2	3	4	5	6	7	n
1								
2								
3								
4								
5								
6			14.3	71.4	14.3			7
7								

C Stages × P4 Stages

C Stages	1	2	3	4	5	6	7	n
1								
2								
3								
4		15.4	53.8	30.8				13
5			28.6	57.1	14.3			7
6				42.9	35.7	21.4		14
7					9.1	36.4	54.5	11

Spitalfields

P3 Stages \ P4 Stages	1	2	3	4	5	6	7	*n*
1								
2	100							5
3								
4								
5			12.5	50.0	37.5			8
6					100			6
7								

M1 Stages \ P4 Stages	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6			28.6	57.1	14.3			7
7								

M2 Stages \ P4 Stages	1	2	3	4	5	6	7	*n*
1								
2								
3								
4				66.7	16.7			6
5								
6				14.3		85.7		7
7								

Poundbury

P3 Stages \ P4 Stages	1	2	3	4	5	6	7	*n*
1								
2								
3								
4		8.3	50.0	41.7				12
5								
6					62.5	37.5		8
7						55.6	44.4	9

M1 Stages \ P4 Stages	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5	12.5	25.0	37.5	25.0				8
6			36.4	36.4	27.3			11
7					33.3	50.0	16.7	12

M2 Stages \ P4 Stages	1	2	3	4	5	6	7	*n*
1								
2								
3	9.1	9.1	36.4	45.5				11
4								
5					50.0	50.0		6
6						83.3	16.7	6
7					16.7	16.7	66.7	6

Burlington

P3 Stages \ P4 Stages	1	2	3	4	5	6	7	*n*
1								
2	84.6	7.7	7.7					13
3		42.3	53.8	3.8				26
4			91.7	8.3				12
5			1.9	19.2	76.9	1.9		52
6					24.4	73.2	1.9	41
7						41.2	58.8	36

M1 Stages \ P4 Stages	1	2	3	4	5	6	7	*n*
1								
2								
3	100							7
4	26.3	57.9	15.8					19
5			76.9	19.2	3.8			26
6			10.0	15.0	62.5	12.5		40
7				2.6	38.5	46.2	12.8	39

M2 Stages \ P4 Stages	1	2	3	4	5	6	7	*n*
1	40.0	60.0						5
2		33.3	66.7					9
3			58.1	25.8	16.1			31
4			7.1	9.5	83.3			42
5					37.9	62.1		29
6						63.0	37.0	46
7							100	5

Belleville

P3 Stages \ P4 Stages	1	2	3	4	5	6	7	*n*
1								
2								
3		20.0	80.0					5
4			35.7	57.1	7.1			14
5				57.1	42.9			7
6				10.0	50.0	40.0		10
7					10.0	20.0	70.0	10

M1 Stages \ P4 Stages	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5			85.7	14.3				7
6			23.1	53.8	23.1			13
7				23.5	29.4	29.4	17.6	17

M2 Stages \ P4 Stages	1	2	3	4	5	6	7	*n*
1								
2								
3			57.1	42.9				7
4			15.0	30.0	40.0	10.0	5.0	20
65				33.3	16.7	50.0		
6								
7								

Spitalfields

I1 Stages (rows) × M1 Stages (columns)

	1	2	3	4	5	6	7	*n*
1								
2								
3								
4			16.7	66.7	16.7			6
5								
6				14.3		85.7		7
7								

I2 Stages (rows) × M1 Stages (columns)

	1	2	3	4	5	6	7	*n*
1								
2	50.0	33.3	6.7					6
3	3.6	14.3	57.1	21.4	3.6			28
4	6.3	6.3	62.5	6.3	18.8			16
5			100					5
6						100		5
7								

C Stages (rows) × M1 Stages (columns)

	1	2	3	4	5	6	7	*n*
1	2.9	28.6	28.6					7
2		16.7	83.3					12
3	3.4		58.6	24.1	13.8			29
4								
5		20.0	20.0			60.0		5
6						66.7	33.3	6
7								

Poundbury

I1 Stages (rows) × M1 Stages (columns)

	1	2	3	4	5	6	7	*n*
1								
2								
3	9.1	9.1	36.4	45.5				11
4								
5					50.0	50.0		6
6						83.3	16.7	6
7					16.7	16.7	66.7	6

I2 Stages (rows) × M1 Stages (columns)

	1	2	3	4	5	6	7	*n*
1								
2								
3			40.0	40.0			20.0	5
4			7.7	76.9	17.4			13
5								
6				9.1	9.1	72.7	9.1	11
7								

C Stages (rows) × M1 Stages (columns)

	1	2	3	4	5	6	7	*n*
1								
2								
3			16.7	66.7		16.7		6
4		6.3	31.3	31.3	25.0	6.3		16
5				11.1	22.2	66.7		9
6				8.3		16.7	75.0	12
7						20.0	80.0	5

Burlington

I1 Stages (rows) × M1 Stages (columns)

	1	2	3	4	5	6	7	*n*
1								
2								
3								
4			42.9	57.1				14
5			8.7	30.4	47.8	13.0		46
6					20.0	80.0		20
7						42.9	57.1	21

I2 Stages (rows) × M1 Stages (columns)

	1	2	3	4	5	6	7	*n*
1								
2								
3								
4			50.0	50.0				16
5	1.9			22.2	48.1	27.8		54
6						80.0	20.0	15
7						35.0	65.0	20

C Stages (rows) × M1 Stages (columns)

	1	2	3	4	5	6	7	*n*
1								
2								
3			47.6	52.4				21
4				18.4	7.9	65.8	7.9	38
5				5.6	31.9	19.4	43.1	72
6						16.7	83.3	6
7								

Belleville

I1 Stages (rows) × M1 Stages (columns)

	1	2	3	4	5	6	7	*n*
1								
2								
3	25.0	25.0	50.0					8
4			25.9	66.7	7.4			27
5								
6				40.0		60.0		5
7						33.3	66.7	6

I2 Stages (rows) × M1 Stages (columns)

	1	2	3	4	5	6	7	*n*
1								
2								
3			14.3	57.1	28.6			7
4				14.3	78.6	3.6	3.6	28
5								
6					14.3	57.1	28.6	7
7								

C Stages (rows) × M1 Stages (columns)

	1	2	3	4	5	6	7	*n*
1								
2								
3			30.8	42.3	26.9			26
4			15.4	61.5	7.7	15.4		26
5				14.3		57.1	28.6	7
6						40.0	60.0	15
7								

Spitalfields

P3 Stages \ M1 Stages	1	2	3	4	5	6	7	*n*
1	7.7		46.2	46.2				13
2			37.5	50.0	12.5			8
3								
4								
5						83.3	16.7	6
6								
7								

P4 Stages \ M1 Stages	1	2	3	4	5	6	7	*n*
1			42.9	57.1				7
2								
3								
4				16.7		66.7	16.7	6
5								
6								
7								

M2 Stages \ M1 Stages	1	2	3	4	5	6	7	*n*
1								
2								
3					40.0	60.0		5
4								
5								
6								
7								

Poundbury

P3 Stages \ M1 Stages	1	2	3	4	5	6	7	*n*
1								
2								
3				54.5	45.5			11
4					38.5	61.5		13
5								
6						30.0	70.0	10
7							100	5

P4 Stages \ M1 Stages	1	2	3	4	5	6	7	*n*
1								
2								
3				30.0	30.0	40.0		10
4					33.3	66.7		6
5						33.3	66.7	6
6							100	6
7								

M2 Stages \ M1 Stages	1	2	3	4	5	6	7	*n*
1								
2								
3				7.7	53.8	38.5		13
4					11.1	77.8	11.1	9
5						22.2	77.8	9
6				10.0			90.0	10
7								

Burlington

P3 Stages \ M1 Stages	1	2	3	4	5	6	7	*n*
1								
2			44.4	50.0	5.6			18
3				52.0	48.0			25
4					75.0	25.0		12
5					8.7	65.2	26.1	46
6						21.7	78.3	23
7						20.0	80.0	10

P4 Stages \ M1 Stages	1	2	3	4	5	6	7	*n*
1	7.7		53.8	38.5				13
2				100				11
3				10.0	76.7	13.3		30
4					41.7	50.0	8.3	12
5					2.4	61.0	36.6	41
6						21.7	78.3	23
7							100	5

M2 Stages \ M1 Stages	1	2	3	4	5	6	7	*n*
1								
2								
3					71.0	29.0		31
4					2.6	64.1	33.3	39
5						16.7	83.3	12
6						19.0	81.0	21
7								

Belleville

P3 Stages \ M1 Stages	1	2	3	4	5	6	7	*n*
1								
2								
3			12.5	50.0	37.5			16
4				6.3	25.0	68.8		16
5						14.3	85.7	7
6						27.3	72.7	11
7								

P4 Stages \ M1 Stages	1	2	3	4	5	6	7	*n*
1								
2								
3				10.0	60.0	30.0		10
4					8.3	58.3	33.3	12
5						42.9	57.1	7
6							100	5
7								

M2 Stages \ M1 Stages	1	2	3	4	5	6	7	*n*
1								
2								
3					62.5	25.0	12.5	8
4					10.0	45.0	45.0	20
5							100	6
6								
7								

Spitalfields

P3 Stages × M1 Stages

P3 \ M1	1	2	3	4	5	6	7	*n*
1	7.7		46.2	46.2				13
2			37.5	50.0	12.5			8
3								
4								
5						83.3	16.7	6
6								
7								

P4 Stages × M1 Stages

P4 \ M1	1	2	3	4	5	6	7	*n*
1			42.9	57.1				7
2								
3								
4				16.7		66.7	16.7	6
5								
6								
7								

M2 Stages × M1 Stages

M2 \ M1	1	2	3	4	5	6	7	*n*
1								
2								
3					40.0	60.0		5
4								
5								
6								
7								

Poundbury

P3 Stages × M1 Stages

P3 \ M1	1	2	3	4	5	6	7	*n*
1								
2								
3				54.5	45.5			11
4					38.5	61.5		13
5								
6						30.0	70.0	10
7							100	5

P4 Stages × M1 Stages

P4 \ M1	1	2	3	4	5	6	7	*n*
1								
2								
3				30.0	30.0	40.0		10
4					33.3	66.7		6
5						33.3	66.7	6
6							100	6
7								

M2 Stages × M1 Stages

M2 \ M1	1	2	3	4	5	6	7	*n*
1								
2								
3				7.7	53.8	38.5		13
4					11.1	77.8	11.1	9
5						22.2	77.8	9
6				10.0			90.0	10
7								

Burlington

P3 Stages × M1 Stages

P3 \ M1	1	2	3	4	5	6	7	*n*
1								
2			44.4	50.0	5.6			18
3				52.0	48.0			25
4					75.0	25.0		12
5					8.7	65.2	26.1	46
6						21.7	78.3	23
7						20.0	80.0	10

P4 Stages × M1 Stages

P4 \ M1	1	2	3	4	5	6	7	*n*
1	7.7		53.8	38.5				13
2				100				11
3				10.0	76.7	13.3		30
4					41.7	50.0	8.3	12
5					2.4	61.0	36.6	41
6						21.7	78.3	23
7							100	5

M2 Stages × M1 Stages

M2 \ M1	1	2	3	4	5	6	7	*n*
1								
2								
3					71.0	29.0		31
4					2.6	64.1	33.3	39
5						16.7	83.3	12
6						19.0	81.0	21
7								

Belleville

P3 Stages × M1 Stages

P3 \ M1	1	2	3	4	5	6	7	*n*
1								
2								
3			12.5	50.0	37.5			16
4				6.3	25.0	68.8		16
5						14.3	85.7	7
6						27.3	72.7	11
7								

P4 Stages × M1 Stages

P4 \ M1	1	2	3	4	5	6	7	*n*
1								
2								
3				10.0	60.0	30.0		10
4					8.3	58.3	33.3	12
5						42.9	57.1	7
6							100	5
7								

M2 Stages × M1 Stages

M2 \ M1	1	2	3	4	5	6	7	*n*
1								
2								
3					62.5	25.0	12.5	8
4					10.0	45.0	45.0	20
5							100	6
6								
7								

Spitalfields

di1 Stages / M1 Stages

	1	2	3	4	5	6	7	*n*
1								
2								
3								
4	80.0		20.0					5
5	60.0	40.0						5
6	23.1	53.8	23.1					13
7	7.7	15.4	61.5	7.7			7.7	13

di2 Stages / M1 Stages

	1	2	3	4	5	6	7	*n*
1								
2								
3								
4	71.4		28.6					7
5	78.6	21.4						14
6	14.3	42.9	42.9					14
7		14.3	85.7					7

dc Stages / M1 Stages

	1	2	3	4	5	6	7	*n*
1								
2								
3	83.3	8.3	8.3					12
4	31.3	43.8	25.0					16
5	20.0		80.0					5
6	9.1		90.9					11
7	11.1		44.4	33.3	11.1			9

Poundbury

di1 Stages / M1 Stages

	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5	71.4	14.3	14.3					7
6		12.5	87.5					8
7			100					5

di2 Stages / M1 Stages

	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5	16.7	33.3	50.0					6
6			100					6
7								

dc Stages / M1 Stages

	1	2	3	4	5	6	7	*n*
1								
2								
3								
4	25.0	16.7	58.3					12
5			100					6
6			50.0	37.5		12.5		8
7								

Burlington

di1 Stages / M1 Stages

	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6								
7								

di2 Stages / M1 Stages

	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6								
7								

dc Stages / M1 Stages

	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6								
7								

Belleville

di1 Stages / M1 Stages

	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6	25.0	55.0	20.0					20
7			73.3	26.7				15

di2 Stages / M1 Stages

	1	2	3	4	5	6	7	*n*
1								
2								
3								
4	50.0	33.3		16.7				6
5	20.0	60.0	20.0					5
6	8.0	24.0	68.0					25
7			35.7	64.3				14

dc Stages / M1 Stages

	1	2	3	4	5	6	7	*n*
1								
2								
3								
4	19.2	46.2	30.8	3.8				26
5	20.0		80.0					5
6		3.3	53.3	43.3				30
7			9.3	90.5				21

Spitalfields

dm1 Stages × M1 Stages

dm1 \ M1	1	2	3	4	5	6	7	*n*
1								
2								
3								
4	61.5	23.1	15.4					13
5	25.0	50.0	25.0					12
6	19.0	23.8	57.1					21
7	8.3		91.7					12

dm2 Stages × M1 Stages

dm2 \ M1	1	2	3	4	5	6	7	*n*
1								
2								
3	70.0	30.0						20
4	25.0	43.6	31.3					16
5		15.4	84.6					13
6			75.0	25.0				16
7	12.5		25.0	37.5	25.0			8

I1 Stages × M2 Stages

I1 \ M2	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6	20.0	20.0	20.0	40.0				5
7								

Poundbury

dm1 Stages × M1 Stages

dm1 \ M1	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5		18.2	72.7	9.1				11
6			72.7	27.3				11
7								

dm2 Stages × M1 Stages

dm2 \ M1	1	2	3	4	5	6	7	*n*
1								
2								
3	60.0		40.0					5
4		18.2	72.7	9.1				11
5		16.7	83.3					6
6			25.0	62.5	12.5			8
7			16.7		50.0	33.3		6

I1 Stages × M2 Stages

I1 \ M2	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6				71.4	28.6			7
7								

Burlington

dm1 Stages × M1 Stages

dm1 \ M1	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6								
7								

dm2 Stages × M1 Stages

dm2 \ M1	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6								
7								

I1 Stages × M2 Stages

I1 \ M2	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5	10.5	23.7	55.3	10.5				38
6			42.9	57.1				21
7			3.3	56.7	20.0	16.7	3.3	30

Belleville

dm1 Stages × M1 Stages

dm1 \ M1	1	2	3	4	5	6	7	*n*
1								
2								
3								
4	38.5	61.5						13
5	18.8	18.8	56.3	6.3				16
6		2.6	59.0	33.3	5.1			39
7			4.8	76.2	19.0			21

dm2 Stages × M1 Stages

dm2 \ M1	1	2	3	4	5	6	7	*n*
1								
2								
3								
4	17.9	35.7	46.4					28
5			58.6	41.4				29
6			18.2	72.7	4.5	4.5		22
7				47.1	35.3	17.6		17

I1 Stages × M2 Stages

I1 \ M2	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6								
7			16.7	66.7	19.7			5

Spitalfields

I2 Stages × M2 Stages

I2 \ M2	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6		16.7	16.7	66.7				6
7								

C Stages × M2 Stages

C \ M2	1	2	3	4	5	6	7	*n*
1								
2								
3	40.0	40.0	20.0					5
4								
5								
6			18.2	36.4	18.2	27.3		11
7								

P3 Stages × M2 Stages

P3 \ M2	1	2	3	4	5	6	7	*n*
1								
2	100							5
3								
4								
5			25.0	62.5	12.5			8
6					16.7	83.3		6
7								

Poundbury

I2 Stages × M2 Stages

I2 \ M2	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6			11.1	66.7	22.2			9
7					33.3	16.7	50.0	6

C Stages × M2 Stages

C \ M2	1	2	3	4	5	6	7	*n*
1								
2								
3								
4		20.0	60.0	20.0				5
5			66.7	33.3				6
6				9.1	63.6	27.3		11
7						57.1	42.9	7

P3 Stages × M2 Stages

P3 \ M2	1	2	3	4	5	6	7	*n*
1								
2								
3								
4			75.0	25.0				12
5								
6				18.2	54.5	18.2	9.1	11
7					10.0	50.0	40.0	10

Burlington

I2 Stages × M2 Stages

I2 \ M2	1	2	3	4	5	6	7	*n*
1								
2								
3								
4	100							4
5	6.0	18.0	58.0	18.0				50
6			12.5	87.5				16
7				47.1	29.4	20.6	2.9	34

C Stages × M2 Stages

C \ M2	1	2	3	4	5	6	7	*n*
1								
2								
3	71.4	28.6						7
4	20.0	40.0	40.0					5
5	1.7	8.6	48.3	37.9	3.4			58
6			1.3	26.3	31.6	38.2	2.6	76
7					12.5	70.8	16.7	24

P3 Stages × M2 Stages

P3 \ M2	1	2	3	4	5	6	7	*n*
1								
2								
3	14.3	42.9	42.9					21
4			83.3	16.7				12
5			21.2	67.3	9.6	1.9		52
6				11.9	47.6	38.1	2.4	42
7					8.8	76.5	14.7	34

Belleville

I2 Stages × M2 Stages

I2 \ M2	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6			25.0	62.5	12.5			
7				60.0	20.0	20.0		

C Stages × M2 Stages

C \ M2	1	2	3	4	5	6	7	*n*
1								
2								
3								
4		18.2	63.6	18.2				11
5			16.7	66.7	16.7			6
6			12.5	68.8	18.8			16
7				11.1	33.3	33.3	22.2	9

P3 Stages × M2 Stages

P3 \ M2	1	2	3	4	5	6	7	*n*
1								
2								
3								
4			53.8	46.2				13
5			14.3	57.1	28.6			7
6				66.7	33.3			12
7				12.5	12.5	50.0	25.0	8

Spitalfields

P4 Stages (rows) × M2 Stages (columns)

P4 \ M2	1	2	3	4	5	6	7	*n*
1								
2								
3								
4			16.7	66.7		16.7		6
5								
6					12.5	72.0	12.5	8
7								

M1 Stages (rows) × M2 Stages (columns)

M1 \ M2	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6		14.3	42.9	42.9				7
7								

I1 Stages (rows) × di1 Stages (columns)

I1 \ di1	1	2	3	4	5	6	7	*n*
1								
2								
3				7.1	7.1	21.4	64.3	14
4								
5								
6								
7								

Poundbury

P4Stages (rows) × M2 Stages (columns)

P4 \ M2	1	2	3	4	5	6	7	*n*
1								
2								
3			80.0	20.0				5
4			83.3	16.7				6
5				33.3	50.0		16.7	6
6					33.3	55.6	11.1	9
7						20.0	80.0	5

M1 Stages (rows) × M2 Stages (columns)

M1 \ M2	1	2	3	4	5	6	7	*n*
1								
2								
3								
4		66.7				33.3		8
5			87.5	12.5				15
6			33.3	46.7	13.3		6.7	19
7				5.3	36.8	47.4	10.5	12

I1 Stages (rows) × di1 Stages (columns)

I1 \ di1	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6								
7								

Burlington

P4 Stages (rows) × M2 Stages (columns)

P4 \ M2	1	2	3	4	5	6	7	*n*
1								
2	50.0	50.0						6
3		19.2	69.2	11.5				26
4			66.7	33.3				12
5			9.8	68.8	21.6			51
6					38.3	61.7		47
7						77.3	22.7	22

M1 Stages (rows) × M2 Stages (columns)

M1 \ M2	1	2	3	4	5	6	7	*n*
1								
2								
3								
4	57.1	42.9						14
5		11.5	84.6	3.8				26
6			22.5	62.5	5.0	10.0		40
7				32.5	25.0	42.5		40

I1 Stages (rows) × di1 Stages (columns)

I1 \ di1	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6								
7								

Belleville

P4 Stages (rows) × M2 Stages (columns)

P4 \ M2	1	2	3	4	5	6	7	*n*
1								
2								
3			57.1	42.9				7
4			27.3	54.5	18.2			11
5				80.0	10.0	10.0		10
6				28.6	42.9	14.3	14.3	7
7				16.7		33.3	50.0	6

IM1 Stages (rows) × M2 Stages (columns)

IM1 \ M2	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5			71.4	28.6				7
6			18.2	81.8				11
7			5.9	52.9	35.3		5.9	17

I1 Stages (rows) × di1 Stages (columns)

I1 \ di1	1	2	3	4	5	6	7	*n*
1								
2								
3								
4						66.7	33.3	9
5						10.0	90.0	10
6								
7								

Spitalfields

I2 Stages × dl1 Stages

I2 Stages	1	2	3	4	5	6	7	*n*
1								
2				16.7	33.3	33.3	16.7	8
3						33.3	66.7	12
4								
5								
6								
7								

C Stages × dl1 Stages

C Stages	1	2	3	4	5	6	7	*n*
1				16.7	16.7	66.7		6
2						28.6	71.4	7
3								
4								
5								
6								
7								

M1 Stages × dl1 Stages

M1 Stages	1	2	3	4	5	6	7	*n*
1			15.4	30.8	23.1	23.1	7.7	13
2					18.2	63.6	18.2	11
3						25.0	66.7	12
4								
5								
6								
7								

Poundbury

I2 Stages × dl1 Stages

I2 Stages	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6								
7								

C Stages × dl1 Stages

C Stages	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6								
7								

M1 Stages × dl1 Stages

M1 Stages	1	2	3	4	5	6	7	*n*
1								
2								
3					8.3	58.3	33.3	12
4								
5								
6								
7								

Burlington

I2 Stages × dl1 Stages

I2 Stages	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6								
7								

C Stages × dl1 Stages

C Stages	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6								
7								

M1 Stages × dl1 Stages

M1 Stages	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6								
7								

Belleville

I2 Stages × dl1 Stages

I2 Stages	1	2	3	4	5	6	7	*n*
1								
2						100		5
3						33.3	66.7	6
4							100	6
5								
6								
7								

C Stages × dl1 Stages

C Stages	1	2	3	4	5	6	7	*n*
1								
2								
3							100	5
4								
5								
6								
7								

M1 Stages × dl1 Stages

M1 Stages	1	2	3	4	5	6	7	*n*
1						100		5
2						100		11
3						26.7	73.3	15
4								
5								
6								
7								

Spitalfields

di2 Stages × di1 Stages

	1	2	3	4	5	6	7	*n*
1								
2								
3			66.7	33.3				9
4				50.0	40.0		10.0	10
5					80.0	20.0		5
6						69.2	30.8	13
7								

dc Stages × di1 Stages

	1	2	3	4	5	6	7	*n*
1								
2								
3			6.3	43.8	43.8	6.3		16
4					9.1	90.9		11
5								
6						16.7	83.3	6
7								

dm1 Stages × di1 Stages

	1	2	3	4	5	6	7	*n*
1								
2								
3			12.5	62.5	25.0			8
4				30.0	50.0	20.0		10
5					12.5	75.0	12.5	8
6				7.7		30.8	61.5	13
7								

Poundbury

di2 Stages × di1 Stages

	1	2	3	4	5	6	7	*n*
1								
2								
3			60.0	40.0				5
4				53.8	38.5	7.7		13
5					13.3	86.7		15
6						77.8	22.2	9
7								

Dc Stages × di1 Stages

	1	2	3	4	5	6	7	*n*
1								
2								
3			12.5	50.0	25.0	12.5		8
4				5.0	30.0	65.0		20
5						83.3	16.7	6
6							100	5
7								

dm1 Stages × di1 Stages

	1	2	3	4	5	6	7	*n*
1								
2								
3			10.0	50.0	30.0	10.0		10
4				8.3	25.0	66.7		12
5					8.3	83.3	8.3	12
6						33.3	66.7	6
7								

Burlington

di2 Stages × di1 Stages

	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6								
7								

dc Stages × di1 Stages

	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6								
7								

dm1 Stages × di1 Stages

	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6								
7								

Belleville

di2 Stages × di1 Stages

	1	2	3	4	5	6	7	*n*
1								
2								
3			28.6	71.4				7
4				65.5	13.8	20.7		29
5						100		7
6				4.8		42.9	52.4	21
7							100	8

dc Stages × di1 Stages

	1	2	3	4	5	6	7	*n*
1								
2								
3				75.0	18.8	6.3		16
4				3.6	3.6	78.6	14.3	28
5								
6						16.7	83.3	12
7								

dm1 Stages × di1 Stages

	1	2	3	4	5	6	7	*n*
1								
2								
3			5.9	94.1				17
4				25.9	18.5	55.6		27
5						58.3	41.7	12
6						13.3	86.7	15
7								

	dl1 Stages	**dl2 Stages**	**dl2 Stages**

Spitalfields

dm2 Stages × dl1 Stages

dm2	1	2	3	4	5	6	7	*n*
1			60.0	40.0				5
2				66.7	33.3			6
3			6.7	6.7	33.3	46.7	6.7	15
4				11.1	11.1	44.4	33.3	9
5				10.0		30.0	60.0	10
6								
7								

I1 Stages × dl2 Stages

I1	1	2	3	4	5	6	7	*n*
1								
2								
3				20.0	20.0	60.0		10
4						22.2	77.8	9
5								
6								
7								

I2 Stages × dl2 Stages

I2	1	2	3	4	5	6	7	*n*
1								
2								
3				7.7		46.2	46.2	13
4								
5								
6								
7								

Poundbury

dm2 Stages × dl1 Stages

dm2	1	2	3	4	5	6	7	*n*
1								
2			16.7	50.0	33.3			6
3			5.9	23.5	23.5	47.1		17
4					9.1	81.8	9.1	11
5								
6								
7								

I1 Stages × dl2 Stages

I1	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6								
7								

I2 Stages × dl2 Stages

I2	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6								
7								

Burlington

dm2 Stages × dl1 Stages

dm2	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6								
7								

I1 Stages × dl2 Stages

I1	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6								
7								

I2 Stages × dl2 Stages

I2	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6								
7								

Belleville

dm2 Stages × dl1 Stages

dm2	1	2	3	4	5	6	7	*n*
1			14.3	85.7				7
2				100				5
3				47.8	17.4	34.8		23
4						71.7	27.3	22
5							100	11
6								
7								

I1 Stages × dl2 Stages

I1	1	2	3	4	5	6	7	*n*
1								
2								
3				27.3	9.1	63.6		11
4						13.3	86.7	15
5								
6								
7								

I2 Stages × dl2 Stages

I2	1	2	3	4	5	6	7	*n*
1								
2				20.0		80.0		5
3					28.6	57.1	14.3	7
4						8.3	91.7	12
5								
6								
7								

Spitalfields

C Stages × dI2 Stages

C Stages	1	2	3	4	5	6	7	n
1				28.6	14.3	57.1		7
2				12.5		50.0	37.5	8
3								
4								
5								
6								
7								

M1 Stages × dI2 Stages

M1 Stages	1	2	3	4	5	6	7	n
1			23.5	29.4	35.3	11.8		17
2					30.0	60.0	10.0	10
3				14.3		42.9	42.9	14
4								
5								
6								
7								

dI1 Stages × dI2 Stages

dI1 Stages	1	2	3	4	5	6	7	n
1								
2								
3			100					6
4			37.5	62.5				8
5				50.0	50.0			8
6					16.7	83.3		12
7				11.1		44.4	44.4	9

Poundbury

C Stages × dI2 Stages

C Stages	1	2	3	4	5	6	7	n
1								
2								
3								
4								
5								
6								
7								

M1 Stages × dI2 Stages

M1 Stages	1	2	3	4	5	6	7	n
1								
2								
3				12.5	18.8	62.5	6.3	16
4								
5								
6								
7								

dI1 Stages × dI2 Stages

dI1 Stages	1	2	3	4	5	6	7	n
1								
2								
3								
4			22.2	77.8				9
5				71.4	28.6			7
6				4.8	61.9	33.3		21
7						40.0	60.0	5

Burlington

C Stages × dI2 Stages

C Stages	1	2	3	4	5	6	7	n
1								
2								
3								
4								
5								
6								
7								

M1 Stages × dI2 Stages

M1 Stages	1	2	3	4	5	6	7	n
1								
2								
3								
4								
5								
6								
7								

dI1 Stages × dI2 Stages

dI1 Stages	1	2	3	4	5	6	7	n
1								
2								
3								
4								
5								
6								
7								

Belleville

C Stages × dI2 Stages

C Stages	1	2	3	4	5	6	7	n
1								
2								
3						22.2	77.8	9
4						14.3	85.7	7
5								
6								
7								

M1 Stages × dI2 Stages

M1 Stages	1	2	3	4	5	6	7	n
1				50.0	16.7	33.3		6
2				18.2	27.3	54.5		11
3					4.3	73.9	21.7	23
4				10.0			90.0	10
5								
6								
7								

dI1 Stages × dI2 Stages

dI1 Stages	1	2	3	4	5	6	7	n
1								
2								
3								
4			20.0	76.0		4.0		25
5								
6				23.8	33.3	42.9		21
7						57.9	42.1	19

Spitalfields

dc Stages / dl2 Stages

dc Stages	1	2	3	4	5	6	7	*n*
1								
2								
3			26.3	52.6	21.1			19
4					38.5	61.5		13
5								
6								
7								

dm1 Stages / dl2 Stages

dm1 Stages	1	2	3	4	5	6	7	*n*
1								
2								
3			38.5	61.5				13
4			9.1	45.5	45.5			11
5					22.2	77.8		9
6				7.7	7.7	46.2	38.5	13
7								

dm2 Stages / dl2 Stages

dm2 Stages	1	2	3	4	5	6	7	*n*
1		11.1	77.8	11.1				9
2			20.0	80.0				5
3			10.5	36.8	31.6	15.8		11
4				11.1		55.6	33.3	9
5								
6								
7								

Poundbury

dc Stages / dl2 Stages

dc Stages	1	2	3	4	5	6	7	*n*
1								
2								
3			10.0	80.0	10.0			10
4				25.0	65.0	10.0		20
5				10.0		90.0		10
6						20.0	80.0	5
7								

dm1 Stages / dl2 Stages

dm1 Stages	1	2	3	4	5	6	7	*n*
1								
2								
3			9.1	72.7	18.2			12
4				35.7	57.1	7.1		14
5				6.3	43.8	50.0		14
6					11.1	66.7	22.2	8
7								

dm2 Stages / dl2 Stages

dm2 Stages	1	2	3	4	5	6	7	*n*
1								
2				100				6
3			5.6	33.3	55.6	5.6		18
4				5.9	41.2	52.9		17
5								
6								
7								

Burlington

dc Stages / dl2 Stages

dc Stages	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6								
7								

dm1 Stages / dl2 Stages

dm1 Stages	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6								
7								

dm2 Stages / dl2 Stages

dm2 Stages	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6								
7								

Belleville

dc Stages / dl2 Stages

dc Stages	1	2	3	4	5	6	7	*n*
1								
2								
3			12.5	87.5				16
4				25.8	25.8	48.4		31
5						100		5
6						47.6	52.4	21
7								

dm1 Stages / dl2 Stages

dm1 Stages	1	2	3	4	5	6	7	*n*
1								
2								
3			18.8	81.3				16
4				51.9	22.2	25.9		27
5				7.1	14.3	71.4	7.1	14
6						68.2	31.8	22
7								

dm2 Stages / dl2 Stages

dm2 Stages	1	2	3	4	5	6	7	*n*
1			42.9	57.1				7
2			20.0	80.0				5
3				72.7	13.6	13.6		22
4					18.5	70.4		27
5						58.8	41.2	17
6							100	9
7								

Spitalfields

I1 Stages (rows) × **dc Stages** (columns)

	1	2	3	4	5	6	7	*n*
1								
2			80.0	20.0				5
3			9.1	18.2	18.2	31.8	22.7	22
4				12.5		75.0	12.5	8
5								
6								
7								

I2 Stages (rows) × **dc Stages** (columns)

	1	2	3	4	5	6	7	*n*
1								
2								
3				19.0	19.0	33.3	28.6	21
4						66.7	33.3	9
5								
6								
7								

C Stages (rows) × **dc Stages** (columns)

	1	2	3	4	5	6	7	*n*
1		14.3	28.6	57.1				7
2				12.5	37.5	50.0		8
3						50.0	50.0	16
4								
5								
6								
7								

Poundbury

I1 Stages (rows) × **dc Stages** (columns)

	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6								
7								

I2 Stages (rows) × **dc Stages** (columns)

	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6								
7								

C Stages (rows) × **dc Stages** (columns)

	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6								
7								

Burlington

I1 Stages (rows) × **dc Stages** (columns)

	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6								
7								

I2 Stages (rows) × **dc Stages** (columns)

	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6								
7								

C Stages (rows) × **dc Stages** (columns)

	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6								
7								

Belleville

I1 Stages (rows) × **dc Stages** (columns)

	1	2	3	4	5	6	7	*n*
1								
2								
3				90.0	10.0			10
4				3.8		60.5	34.6	26
5								
6								
7								

I2 Stages (rows) × **dc Stages** (columns)

	1	2	3	4	5	6	7	*n*
1								
2				100				5
3				62.5	12.5	25.0		8
4						52.0	48.0	25
5								
6								
7								

C Stages (rows) × **dc Stages** (columns)

	1	2	3	4	5	6	7	*n*
1								
2				60.0	20.0	20.0		5
3				7.7		76.9	15.4	13
4						30.4	69.6	23
5								
6								
7								

Spitalfields

M1 Stages × dc Stages

	1	2	3	4	5	6	7	*n*
1		10.0	50.0	25.0	5.0	5.0	5.0	20
2			8.3	58.3	8.3	8.3	25.0	
3			4.3	17.4	17.4	43.5	17.4	25
4								
5								
6								
7								

di1 Stages × dc Stages

	1	2	3	4	5	6	7	*n*
1								
2								
3								
4		12.5	87.5				8	
5			87.5	12.5			8	
6			7.7	76.9	7.7	7.7	13	
7					37.5	62.5	8	

di2 Stages × dc Stages

	1	2	3	4	5	6	7	*n*
1								
2								
3	11.1	33.3	55.6					9
4		8.3	83.3		8.3			12
5			44.4	55.6				9
6				61.5	15.4	23.1		13
7					16.7	66.7	16.7	6

Poundbury

M1 Stages × dc Stages

	1	2	3	4	5	6	7	*n*
1								
2								
3				41.2	35.3	23.5		17
4								
5								
6								
7								

di1 Stages × dc Stages

	1	2	3	4	5	6	7	*n*
1								
2								
3								
4	14.3	14.3	57.1	14.3				7
5			25.0	75.0				8
6			5.3	68.4	26.3			19
7					16.7	83.3		6

di2 Stages × dc Stages

	1	2	3	4	5	6	7	*n*
1								
2								
3								
4	6.7		53.3	33.3	6.7			15
5			7.1	92.9				14
6				16.7	75.0	8.3		12
7								

Burlington

M1 Stages × dc Stages

	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6								
7								

di1 Stages × dc Stages

	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6								
7								

di2 Stages × dc Stages

	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6								
7								

Belleville

M1 Stages × dc Stages

	1	2	3	4	5	6	7	*n*
1			14.3	71.4	14.3			7
2				62.3		7.7	13	
3				26.7	13.3	53.3	6.7	30
4				3.7		48.1	48.1	27
5								
6								
7								

di1 Stages × dc Stages

	1	2	3	4	5	6	7	*n*
1								
2								
3								
4		7.1	85.7	7.1				14
5								
6			4.0	88.0		8.0		25
7				21.1	15.8	57.9	5.3	19

di2 Stages × dc Stages

	1	2	3	4	5	6	7	*n*
1								
2								
3								
4			63.6	36.4				22
5				100				8
6				50.0	16.7	33.3		30
7						73.3	26.7	15

Spitalfields

dm1 Stages (rows) × dc Stages (columns)

	1	2	3	4	5	6	7	*n*
1								
2								
3		18.2	81.8					11
4			69.2	30.8				13
5			20.0	70.0	10.0			10
6			6.3	25.0	18.8	50.0		16
7					11.1	55.6	33.3	69

dm2 Stages (rows) × dc Stages (columns)

	1	2	3	4	5	6	7	*n*
1	40.0	20.0	20.0	20.0				5
2								
3			66.7	28.6	4.8			21
4			6.7	53.3	26.7	13.3		15
5			9.1	18.2	18.2	54.5		11
6						66.7	33.3	9
7								

I1 Stages (rows) × dm1 Stages (columns)

	1	2	3	4	5	6	7	*n*
1								
2				80.0	20.0			5
3				8.3	20.8	50.0	20.8	24
4						37.5	62.5	8
5								
6								
7								

Poundbury

dm1 Stages (rows) × dc Stages (columns)

	1	2	3	4	5	6	7	*n*
1								
2								
3	8.3		66.7	25.0				12
4			14.3	78.6	7.1			14
5				57.1	42.9			14
6				12.5	37.5	50.0		8
7								

dm2 Stages (rows) × dc Stages (columns)

	1	2	3	4	5	6	7	*n*
1								
2	20.0		80.0					5
3			29.4	64.7	5.9			17
4				57.1	42.9			14
5								
6						100		5
7								

I1 Stages (rows) × dm1 Stages (columns)

	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6								
7								

Burlington

dm1 Stages (rows) × dc Stages (columns)

	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6								
7								

dm2 Stages (rows) × dc Stages (columns)

	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6								
7								

I1 Stages (rows) × dm1 Stages (columns)

	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6								
7								

Belleville

dm1 Stages (rows) × dc Stages (columns)

	1	2	3	4	5	6	7	*n*
1								
2								
3		14.3	85.7					7
4			25.0	75.0				28
5				70.6	11.8	11.8		17
6				5.6	13.9	75.0	5.6	36
7						31.3	68.8	16

dm2 Stages (rows) × dc Stages (columns)

	1	2	3	4	5	6	7	*n*
1								
2								
3			55.0	45.0				20
4				78.1	9.4	12.5		32
5					15.4	80.0	3.8	26
6						52.9	47.1	17
7				11.1			88.9	9

I1 Stages (rows) × dm1 Stages (columns)

	1	2	3	4	5	6	7	*n*
1								
2								
3				30.0	60.0	10.0		10
4					3.7	59.3	37.0	27
5								
6								
7								

Spitalfields

I2 Stages \ dm1 Stages	1	2	3	4	5	6	7	*n*
1								
2				50.0	33.3	16.7		6
3					19.0	57.1	23.8	21
4						28.6	71.4	7
5								
6								
7								

C Stages \ dm1 Stages	1	2	3	4	5	6	7	*n*
1			14.3	14.3	57.1	14.3		7
2					16.7	66.7	16.7	12
3				7.1		21.4	71.4	14
4								
5								
6								
7								

M1 Stages \ dm1 Stages	1	2	3	4	5	6	7	*n*
1			20.0	40.0	15.0	20.0	5.0	20
2				20.0	46.7	33.3		15
3				7.41	7.41	44.4	40.7	27
4								
5								
6								
7								

Poundbury

I2 Stages \ dm1 Stages	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6								
7								

C Stages \ dm1 Stages	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6								
7								

M1 Stages \ dm1 Stages	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6								
7								

Burlington

I2 Stages \ dm1 Stages	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6								
7								

C Stages \ dm1 Stages	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6								
7								

M1 Stages \ dm1 Stages	1	2	3	4	5	6	7	*n*
1								
2								
3			5.6		44.4	38.9	11.1	18
4					16.7	50.0	33.3	6
5								
6								
7								

Belleville

I2 Stages \ dm1 Stages	1	2	3	4	5	6	7	*n*
1								
2				80.0	20.0			5
3				11.1	55.6	33.3		9
4						42.3	57.7	26
5								
6								
7								

C Stages \ dm1 Stages	1	2	3	4	5	6	7	*n*
1								
2								
3					11.1	61.1	27.8	18
4						27.3	72.7	22
5								
6								
7								

M1 Stages \ dm1 Stages	1	2	3	4	5	6	7	*n*
1				62.5	37.5			8
2				63.6	27.3	9.1		11
3					27.3	69.7	3.0	33
4					3.3	43.3	53.3	30
5								
6								
7								

Spitalfields

dm1 Stages

di1 Stages	1	2	3	4	5	6	7	*n*
1								
2								
3								
4			62.5	25.0		12.5		8
5			25.0	62.5	12.5			8
6				15.4	46.2	30.8	7.7	13
7					9.1	72.7	18.2	11

dm1 Stages

di2 Stages	1	2	3	4	5	6	7	*n*
1								
2								
3	18.2	27.3	45.5	9.1				11
4			53.3	33.3		13.3		15
5				70.0	20.0	10.0		10
6					50.0	42.9	7.1	14
7						71.4	28.6	7

dm1 Stages

I2 Stages	1	2	3	4	5	6	7	*n*
1								
2								
3			40.0	45.0	10.0	5.0		20
4				26.7	46.7	26.7		15
5					20.0	60.0	20.0	5
6						61.5	38.5	13
7								

Poundbury

di1 Stages	1	2	3	4	5	6	7	*n*
1								
2								
3	40.0	40.0	20.0					5
4	20.0	10.0	60.0	10.0				10
5			42.9	42.9	14.3			7
6			4.8	38.1	47.6	9.5		21
7					14.3	57.1	28.6	7

di2 Stages	1	2	3	4	5	6	7	*n*
1								
2								
3								
4	12.5		50.0	31.3	6.3			16
5			11.1	44.4	38.9	5.6		18
6				6.7	53.3	40.0		15
7								

dc Stages	1	2	3	4	5	6	7	*n*
1								
2								
3	9.1		72.7	18.2				11
4			13.0	47.8	34.8	4.3		23
5				10.0	60.0	30.0		10
6						80.0	20.0	5
7								

Burlington

di1 Stages	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6								
7								

di2 Stages	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6								
7								

dc Stages	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6								
7								

Belleville

di1 Stages	1	2	3	4	5	6	7	*n*
1								
2								
3								
4	3.7	11.1	59.3	25.9				27
5				100				5
6				71.0	22.6	6.5		13
7					26.3	68.4	5.3	19

di2 Stages	1	2	3	4	5	6	7	*n*
1								
2								
3	16.7	33.3	50.0					6
4		6.7	43.3	46.7	3.3			30
5				75.0	25.0			8
6				21.9	31.3	46.9		32
7					7.1	64.3	28.6	14

dc Stages	1	2	3	4	5	6	7	*n*
1								
2								
3		12.5	37.5	43.8	6.3			16
4				58.3	36.1	5.6		36
5					28.6	71.4		7
6					5.9	79.1	14.7	34
7						15.4	84.6	13

Spitalfields

dm1 Stages (columns) × **dm2 Stages** (rows)

	1	2	3	4	5	6	7	*n*
1	25.0	37.5	37.5					8
2			100					5
3			17.4	52.2	21.7	37.5		16
4					7.7	69.2	23.1	13
5						36.4	63.6	11
6								
7								

dm2 Stages (columns) × **I1 Stages** (rows)

	1	2	3	4	5	6	7	*n*
1								
2			100					5
3			9.7	19.4	22.6	35.5	12.9	31
4					40.0	40.0	20.0	10
5								
6								
7								

dm2 Stages (columns) × **I2 Stages** (rows)

	1	2	3	4	5	6	7	*n*
1								
2			66.7	16.7	16.7			6
3			3.4	17.2	24.1	41.4	13.8	29
4					27.3	45.5	27.3	11
5								
6								
7								

Poundbury

dm1 Stages (columns) × **dm2 Stages** (rows)

	1	2	3	4	5	6	7	*n*
1	16.7	50.0	33.3					6
2	14.3		85.7					7
3		4.5	22.7	59.1	13.6			22
4				10.0	75.0	15.0		20
5						87.5	12.5	8
6						85.7	14.3	7
7								

dm2 Stages (columns) × **I1 Stages** (rows)

	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6								
7								

dm2 Stages (columns) × **I2 Stages** (rows)

	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6								
7								

Burlington

dm1 Stages (columns) × **dm2 Stages** (rows)

	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6								
7								

dm2 Stages (columns) × **I1 Stages** (rows)

	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6								
7								

dm2 Stages (columns) × **I2 Stages** (rows)

	1	2	3	4	5	6	7	*n*
1								
2								
3								
4								
5								
6								
7								

Belleville

dm1 Stages (columns) × **dm2 Stages** (rows)

	1	2	3	4	5	6	7	*n*
1		28.6	71.4					7
2		16.7	83.3					6
3			20.8	70.8	8.3			24
4				38.9	38.9	22.2		36
5					6.1	93.9		33
6						30.0	70.0	20
7							100	7

dm2 Stages (columns) × **I1 Stages** (rows)

	1	2	3	4	5	6	7	*n*
1								
2								
3			10.0	90.0				10
4				9.7	38.7	35.5	16.1	31
5								
6								
7								

dm2 Stages (columns) × **I2 Stages** (rows)

	1	2	3	4	5	6	7	*n*
1								
2				100				5
3				66.7	33.3			9
4				3.4	24.1	55.2	17.2	29
5								
6								
7								

Spitalfields

C Stages × dm2 Stages

	1	2	3	4	5	6	7	n
1		14.3	28.6	28.6	28.6			7
2				33.3	50.0	16.7		12
3					13.0	60.9	26.1	23
4								
5								
6								
7								

M1 Stages × dm2 Stages

	1	2	3	4	5	6	7	n
1	4.8	4.8	66.7	19.0				21
2			40.0	46.7	13.3			15
3				16.7	36.7	40.0	6.7	30
4						70.0	30.0	10
5								
6								
7								

di1 Stages × dm2 Stages

	1	2	3	4	5	6	7	n
1								
2								
3								
4	25.0		50.0	12.5	12.5			8
5		25.0	62.5	12.5				8
6			50.0	28.6	21.4			14
7			8.3	25.0	50.0	8.3	8.3	12

Poundbury

C Stages × dm2 Stages

	1	2	3	4	5	6	7	n
1								
2								
3								
4					16.7	33.3	50.0	6
5								
6								
7								

M1 Stages × dm2 Stages

	1	2	3	4	5	6	7	n
1								
2								
3			11.1	44.4	27.8	11.1	5.186	
4				16.7		83.3	6	
5								
6								
7								

di1 Stages × dm2 Stages

	1	2	3	4	5	6	7	n
1								
2								
3								
4	11.1	44.4	44.4					9
5		14.3	57.1	14.3	14.3			7
6			44.4	50.0	5.6			18
7				14.3	28.6	42.9	14.3	7

Burlington

C Stages × dm2 Stages

	1	2	3	4	5	6	7	n
1								
2								
3								
4								
5								
6								
7								

M1 Stages × dm2 Stages

	1	2	3	4	5	6	7	n
1								
2								
3								
4								
5								
6								
7								

di1 Stages × dm2 Stages

	1	2	3	4	5	6	7	n
1								
2								
3								
4								
5								
6								
7								

Belleville

C Stages × dm2 Stages

	1	2	3	4	5	6	7	n
1								
2								
3				5.3	57.9	31.6	5.3	19
4				3.4	6.9	51.7	37.9	29
5								
6								
7								

M1 Stages × dm2 Stages

	1	2	3	4	5	6	7	n
1			37.5	62.5				8
2			9.1	90.9				11
3				38.2	50.0	11.8		34
4					34.3	45.7	20.0	35
5						14.3	85.7	7
6								
7								

di1 Stages × dm2 Stages

	1	2	3	4	5	6	7	n
1								
2								
3								
4	27.3	22.7	50.0					22
5								
6			33.3	66.7				24
7				30.0	55.0	15.0		20

Spitalfields

dl2 Stages × dm2 Stages

	1	2	3	4	5	6	7	n
1								
2								
3	63.6	9.1	18.2	9.1				11
4	7.1	28.6	50.0	7.1	7.1			14
5			66.7	33.3				9
6			21.4	42.9	35.7			14
7			14.3		42.9	42.9		7

dc Stages × dm2 Stages

	1	2	3	4	5	6	7	n
11								
22								
33	5.0	15.0	70.0	5.0	5.0			20
44			37.5	50.0	12.5			16
55			14.3	57.1	28.6			7
66				14.3	42.9	42.9		14
77						42.9	57.1	7

dm1 Stages × dm2 Stages

	1	2	3	4	5	6	7	n
1								
2								
3	23.1	38.5	30.8	7.7				13
4			85.7	14.3				14
5			38.5	53.8	7.7			13
6			9.5	28.6	42.9	19.0		21
7					25.0	58.3	16.7	12

Poundbury

dl2 Stages × dm2 Stages

	1	2	3	4	5	6	7	n
1								
2								
3								
4	12.5	37.5	37.5	6.3	6.3			16
5			58.8	41.2				17
6			8.3	75.0	16.7			12
7								

dc Stages × dm2 Stages

	1	2	3	4	5	6	7	n
11								
22								
33	18.2	36.4	45.5					11
44			57.1	38.1	4.8			21
55			12.5	75.0	12.5			8
66					25.0	62.5	12.5	8
77								

dm1 Stages × dm2 Stages

	1	2	3	4	5	6	7	n
1								
2								
3	15.4	46.2	38.5				13	
4			86.7	13.3			15	
5			16.7	83.3			18	
6				18.8	43.8	37.5	16	
7								

Burlington

I2 Stages × dm2 Stages

	1	2	3	4	5	6	7	n
1								
2								
3								
4								
5								
6								
7								

dc Stages × dm2 Stages

	1	2	3	4	5	6	7	n
11								
22								
33								
44								
55								
66								
77								

dm1 Stages × dm2 Stages

	1	2	3	4	5	6	7	n
1								
2								
3								
4								
5								
6								
7								

Belleville

dl2 Stages × dm2 Stages

	1	2	3	4	5	6	7	n
1								
2								
3								
4	14.3	14.3	57.1	10.7			3.6	28
5			37.5	62.5				8
6			9.4	59.4	31.3			32
7					41.2	52.9	5.9	17

dc Stages × dm2 Stages

	1	2	3	4	5	6	7	n
11								
22								
33	6.7	20.0	73.3					15
44			25.7	71.4			2.9	35
55				33.3	66.7			6
66				11.8	61.8	26.5		34
77					5.9	47.1	47.1	17

dm1 Stages × dm2 Stages

	1	2	3	4	5	6	7	n
1								
2								
3	33.3	33.3	33.3					15
4			54.8	45.2				31
5			11.1	77.8	11.1			18
6				17.8	68.9	13.3		45
7						66.7	33.3	21