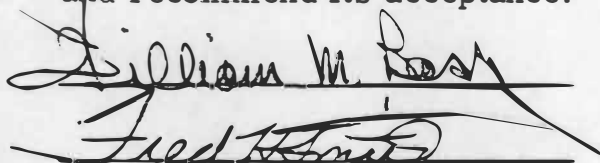


To the Graduate Council:

I am submitting herewith a thesis written by Robert S. Webb entitled "Sexual Differences, Fluctuating Asymmetry and Their Implications in the Growth Rate and Developmental Stability of the Permanent Dentition and the Primary Dermal Ridge of the Fingers and Palms." I recommend that it be accepted in partial fulfillment of the requirements for the degree of Master of Arts, with a major in Anthropology.


Richard L. Jantz, Major Professor

We have read this thesis
and recommend its acceptance:



Accepted for the Council:

Vice Chancellor
Graduate Studies and Research

SEXUAL DIFFERENCES, FLUCTUATING ASYMMETRY AND THEIR
IMPLICATIONS IN THE GROWTH RATE AND DEVELOPMENTAL
STABILITY OF THE PERMANENT DENTITION AND
THE PRIMARY DERMAL RIDGES OF
THE FINGERS AND PALMS

A Thesis

Presented for the

Master of Arts

Degree

The University of Tennessee, Knoxville

Robert S. Webb

December 1977

ACKNOWLEDGEMENTS

I would like to thank Dr. Richard L. Jantz for his advice and guidance, Dr. William M. Bass for his encouragement, and Dr. Fred H. Smith for his helpful criticism. Thanks should also be extended to Fred Fohl and Ken Parham for their assistance in collecting the data and to the students who participated in this study.

ABSTRACT

This study presents evidence that specific genetic material influences the growth rate and developmental stability of two genetic systems, dermal ridges and permanent dentition, having mutually exclusive developmental periods. Dermal ridges develop rapidly early in fetal life, while the permanent dentition develops slowly in a post-natal environment. Theories centering on the nature and effects of the specific genetic material (heterochromatin) are discussed and applied whenever possible.

The data for this investigation were collected from students in introductory anthropology classes at the University of Tennessee, Knoxville. The sample consists of fifty males and fifty females. Finger prints, palm prints and casts of the permanent dentition were obtained from each subject. In addition, each subject filled out a questionnaire eliciting genetic and dental information.

Statistical analysis of the data was carried out utilizing the Statistical Package for the Social Sciences (SPSS). Summary statistics, Pearson product-moment correlation coefficients and canonical correlations were calculated for all dental and dermal variables. Fluctuating asymmetry was used as a measure of developmental stability. The degree of fluctuating asymmetry was determined by taking the absolute right-left difference for each variable, and by calculating

the correlation coefficient between sides. Canonical correlation analysis was used to evaluate the degree to which dermal and dental systems are integrated.

The results indicate that there are sexual differences in size and shape of the teeth, ridge counts, fluctuating asymmetry and the degree to which the two systems are integrated. The most significant finding is that the dental and dermal systems are integrated. This may be due to the nature of the genetic material responsible for them, or because they develop and grow under similar controlling factors. Although the findings suggest that there is some common control throughout pre-natal and postnatal development, they do not supply evidence as to their specific nature.

TABLE OF CONTENTS

CHAPTER	PAGE
I. INTRODUCTION	1
II. BACKGROUND	4
III. OBJECTIVES	23
IV. MATERIALS AND METHODS	25
V. RESULTS AND DISCUSSION	36
BIBLIOGRAPHY	79
VITA	85

LIST OF TABLES

TABLE	PAGE
1. Mean developmental time and sex differences for formation of the permanent mandibular dentition.	21
2. Means, standard deviations, mean asymmetry values and right-left correlation coefficients for dental measurements.	37
3. Means (in percent), standard deviations, mean asymmetry values and correlation coefficients for crown indices. . .	40
4. Means, standard deviations, mean asymmetry values and correlation coefficients for digital and palmar ridge counts	42
5. Correlation coefficients between raw dental measurements of the upper right quadrant and digital ridge counts - males.	49
6. Correlation coefficients between raw dental measurements of the upper left quadrant and digital ridge counts - males.	50
7. Correlation coefficients between raw dental measurements of both lower quadrants and digital ridge counts - males.	51
8. Correlation coefficients between raw dental measurements of the upper right quadrant and digital ridge counts - females.	52
9. Correlation coefficients between raw dental measurements of the upper left quadrant and digital ridge counts - females.	53
10. Correlation coefficients between raw dental measurements of the lower right quadrant and digital ridge counts - females.	54

TABLE

PAGE

11.	Correlation coefficients between raw dental measurements of the lower left quadrant and digital ridge counts - females.	55
12.	Correlation coefficients between raw dental measurements of both upper quadrants and palmar ridge counts - males	56
13.	Correlation coefficients between raw dental measurements of both lower quadrants and palmar ridge counts - males	57
14.	Correlation coefficients between raw dental measurements of both upper quadrants and palmar ridge counts - females.	58
15.	Correlation coefficients between raw dental measurements of the lower right quadrant and palmar ridge counts - females.	59
16.	Correlation coefficients between raw dental measurements of the lower left quadrant and palmar ridge counts - females.	60
17.	Correlation coefficients between crown indices of both upper quadrants and digital ridge counts - males	61
18.	Correlation coefficients between crown indices of both lower quadrants and digital ridge counts - males	62
19.	Correlation coefficients between crown indices of all quadrants and digital ridge counts - females	63
20.	Correlation coefficients between crown indices of both upper quadrants and palmar ridge counts - males	64
21.	Correlation coefficients between crown indices of both lower quadrants and palmar ridge counts - males	65
22.	Correlation coefficients between crown indices of all quadrants and palmar ridge counts - females	66

TABLE	PAGE
23. Correlation coefficients between dental asymmetry values from the maxillary dentition and asymmetry values from digital and palmar ridge counts - males. . .	67
24. Correlation coefficients between dental asymmetry values from the mandibular dentition and asymmetry values from digital and palmar ridge counts - males. . .	68
25. Correlation coefficients between asymmetry values from the maxillary dentition and those from the digital and palmar ridge counts - females	69
26. Correlation coefficients between asymmetry values from the mandibular dentition and those from the digital and palmar ridge counts - females	70
27. Canonical correlations between dermal and dental values, and supporting statistics	74

LIST OF FIGURES

FIGURE		PAGE
1.	Ridge counts used in study.	26
2.	Absolute differences of right minus left (x axis) versus right plus left (y axis) for individual D-radial ridge counts .	29
3.	Absolute difference of right minus left (x axis) versus right plus left (y axis) for individual C-ulnar ridge counts .	30
4.	Absolute difference of right minus left (x axis) versus right plus left (y axis) for individual radial ridge counts of Digit II	31
5.	Absolute difference of right minus left (x axis) versus right plus left (y axis) for individual ulnar ridge counts of Digit II	32

CHAPTER I

INTRODUCTION

This thesis deals with certain aspects of growth and development. The major aspect will hopefully measure the degree of stability during the development of the human dentition and the primary dermal ridges of the fingers and palms. Stability is measured by the random differences between sides and is known as fluctuating asymmetry (Van Valen, 1962). Asymmetry will be explained in the next chapter. Another aspect considers sex differences in developmental stability, gross size, and shape of the dentition. Sex differences in ridge counts and ridge count asymmetry are also examined. The author will expound on these aspects in Chapter V.

Sex differences in size, shape, growth and developmental stability are explained in the theories set forth by Mittwoch (1973), Barlow (1973), Hamerton (1968), Brown (1966) and Commoner (1964). These theories generally state that the constitution, state and number of sex chromosomes affect growth and stability during development.

The present study offers a genetic explanation for the sexual differences in the dermal ridges and permanent dentition. Since these two genetic systems (dermatoglyphs and the permanent dentition) are popular in population studies as genetic markers, any new

information concerning differences between the sexes might be of value.

According to Krogman (1972), growth and development are intimately related but separate entities and can be measured accordingly. Growth can be measured by size (tooth diameters) and shape (crown indices) in the dentition, and by number (ridge counts) in the dermal ridges. Stability during development can be assessed by the degree of difference and variability between right and left sides in size and number (fluctuating asymmetry).

When fluctuating asymmetry is used as a measure of developmental stability (in dermatoglyphic studies) serious problems arise. Many statistical methods used to obtain asymmetry values do not give true reflections of the asymmetry between the hands. This is of particular concern in the digital ridge counts and will be discussed thoroughly in Chapter IV.

The statistical methods employed in measuring dermatoglyphic asymmetry vary considerably from author to author. For example, Holt (1954) evaluated asymmetry by subtracting the summed left hand digital ridge count from that of the right hand. Parsons (1964) took the absolute differences between corresponding digits, while Singh (1968) used the mean of the squared differences between corresponding digits. None of these methods are comparable.

Problems in measuring asymmetry in the permanent dentition are mostly dependent on the long developmental period of the teeth. Bilateral differences in the dentition might be attributed to both genetic and environmental factors, but it is almost impossible to discern the amount of asymmetry contributed by specific genetic or environmental factors.

In the present study different statistical procedures are employed which will hopefully clarify the role of fluctuating asymmetry in the dentition and dermal ridges. This may elucidate the part fluctuating asymmetry plays in growth and development.

CHAPTER II

BACKGROUND

One way in which control of developmental systems has been measured is through fluctuating asymmetry. It is reasonable to assume that in organisms which are normally bilaterally symmetrical, the genotype is identical for both sides (Adams and Niswander, 1967). When an organism is unable to produce two perfectly symmetrical sides it may be termed non-directional or fluctuating asymmetry (Van Valen, 1962). Adams and Niswander (1967) believe this to be the result of an inability by the organism to buffer itself against environmental and developmental stress factors. Waddington (1957, 1960), in his studies on different strains of Drosophila, refers to the cause of fluctuating asymmetry as developmental noise. The term noise refers to the effect that environmental factors such as disease or nutrition have on intrauterine growth.

In some bilateral traits used in populational studies there is a tendency for one side to be consistently larger than the other. This is commonly called directional asymmetry (Van Valen, 1962). An example of this is clearly illustrated in the tendency for the right arm bones to be larger than the left ones in most populations. In this study, directional asymmetry will not be dealt with to any degree.

More recent studies have revealed that nutritional and socio-economic factors (Perzigian, 1977; Anderson et al., 1975; Bailit, 1975; Enwonwu, 1973; Goose and Lee, 1973; Bailit et al., 1968; Goose, 1967), heat stress (Siegel et al., 1976), cold stress (Siegel and Doyle, 1975b) and audiogenic stress (Siegel and Doyle, 1975a, Siegel and Smookler, 1973) all seem to be responsible for some degree of fluctuating asymmetry in the dentition of humans and rats when they are introduced into the animal's environment during dental development.

Although the studies on rats and fruit flies are important because they attribute fluctuating asymmetry to specific stressors, much research needs to be done on human populations. Bailit et al. (1970) offer an explanation for fluctuating asymmetry in the human dentition based on data from four populations. Each population had a different degree of environmental stress imposed upon it, and the gene pools differed in size. The authors found that the most inbred of the four groups (Tristanites) was also the most asymmetrical. These people also inhabited the least favorable environment, and the investigators believe this to be the major causative factor of fluctuating asymmetry. Bader (1965) presents data showing that inbred rats exhibit a greater degree of dental asymmetry, while random-bred rats show the least amount. There are also many pathological and nutritional factors which may complicate the task of attributing fluctuating asymmetry to either inbreeding or environment. Studying genetically unusual

groups might also confuse the issue. In the situations described above perhaps inbreeding can be considered as an environmental stressor since it alters the genetic environment.

Perzigian (1977) approaches fluctuating asymmetry using skeletal populations (one prehistoric hunting group, two late prehistoric farming groups, one modern cadaver group) with different nutritional and subsistence backgrounds. His hypothesis is that dental asymmetry may reflect growth disturbances mediated by prenatal and post-natal environments. Perzigian finds that those groups under the best subsistence conditions (modern cadaver and late prehistoric farming groups) were skeletally larger and showed less fluctuating asymmetry in their dentitions. This supports the hypothesis that environmental factors in the form of living conditions affect growth and can be reflected by dental asymmetry.

Potter et al. (1976), in a study centering on the discordance, fluctuating asymmetry and mirror imagery in the dentition of monozygotic and dizygotic twins, do not find any genetic basis for fluctuating dental asymmetry. Holt (1954) believes there is a heritable component for fluctuating asymmetry in the dermal ridges, when it is measured in terms of variability. Parsons (1964) thinks that his measure of asymmetry (absolute difference of the right ridge count minus the corresponding left ridge count) also shows some degree of heritability, but he does not expound on it. Singh (1970) found a low,

but significant hereditary component in ridge count asymmetry but he believes that most of the asymmetry is due to environmental effects or chance.

The hypothesis that fluctuating asymmetry is the result of environmental stress is challenged in a pilot study by Jantz and Webb (N. D.) and a report by Jantz (1977). Both studies support the hypothesis that fluctuating asymmetry is genetically controlled. There are two reasons for this. First, there are significant differences in fluctuating asymmetry among Negroid, Caucasoid and Mongoloid groups and secondly, there are significant differences between sexes. It is unlikely that environmental factors could account for these differences. The authors therefore believe that fluctuating asymmetry has a genetic basis.

Jantz (1975) explains populational differences in asymmetry in digital ridge counts by genetic control over developmental stability. He reasons that those populations under the most environmental stress require stricter genetic control over developmental stability (yielding less asymmetry), while populations subject to less environmental stress do not depend on this genetic mechanism as heavily and show more asymmetry. The studies by Jantz and Webb (N. D.) and Jantz (1975, 1977) imply that fluctuating asymmetry is a measure of an organism's genetic ability to adapt to its environment.

Garn et al. (1967a) have suggested that bilateral asymmetry in the crown dimensions of the permanent dentition is greater in males than females because males lack the replicate X chromosome and are therefore more susceptible to developmental accidents. They believe that the replicate X chromosome acts as a stabilizing agent during development. As evidence for this hypothesis, they note that males exhibit more fluctuating asymmetry in buccolingual tooth diameters. In earlier studies, Garn et al. (1963b, 1964a) demonstrated that absence and size reduction of other teeth characterized persons with third molar agenesis. Garn and his associates (1963a) also conceptualized third molar agenesis as being part of a field responsible for size variation and number reduction in the dentition. Garn suggests that this field is controlled by genes which regulate an individual's developmental timing and ability to buffer the dentition against developmental stress. A similar model is conceivable for the dermal ridges but at present there are no references to support such a model. Are the controlling factors that Garn suggests for the dentition related to those that may be controlling dermal ridge development? Are the controlling agents in utero the same as those active during postnatal development? The following discussion may help elucidate these questions.

According to Jantz (Personal Communication), chromosomal content and the growth rate may affect the number of dermal ridges

formed and the degree of fluctuating asymmetry present. Such a theory is also logical for the dental dimensions.

Following this line of reasoning it is necessary to refer to Mittwoch (1973) for an explanation of how this theory might work. Mittwoch suggests that growth rates are affected by differences in the constitution of the sex chromosomes. For example, she notes that males are usually larger at birth than females and she gives evidence that X and Y chromosomes differ in their influence over the total ridge count. This was first noted by Penrose (1969) in persons with sex chromosome abnormalities.

Penrose believes that the addition of an X chromosome to the karyotype causes a reduction in the total ridge count. For example, when the total ridge count of an individual with Turner's syndrome (XO) is considered, it is much higher than that of a normal female (XX). This emphasizes the importance of the replicate X chromosome in association with slower growth rates. Shiono et al. (1975), in their study on individuals with Klinefelter's syndrome (XXY, XXYY, XXXY, XXXXY), demonstrate that extra X or Y chromosomes cause a reduction in the a-b ridge count. The reduction in ridge count is markedly greater when there are extra X chromosomes. While there is a reduction in ridge count when additional Y chromosomes are present, it is much less when extra X's are present.

Mittwoch (1973) explains the above phenomena in part by a theory developed by Lyon (1962). This theory states that when more than one X chromosome is present, the duplicate X's are late replicating and inactive. Mittwoch thinks that the presence of the extra chromosome, even though it is genetically inactive, slows down the rate of growth considerably because of its heterochromatic state. Heterochromatic regions of chromosomes are thought to be genetically inert (Mittwoch, 1973). This state is known as constitutive heterochromatin. These regions are called heterochromatic because they are normally more condensed than the other regions of the chromosome and stain much darker than the euchromatic or active regions (Mittwoch, 1973). When a replicate X chromosome becomes heterochromatic while the other X remains active or euchromatic, the replicate chromosome is said to be in a state of facultative heterochromatin. This state is the result of the replicate X chromosome's slower development. While both X's contain the same genetic material and include areas that are in facultative heterochromatin, the late replicating X chromosome becomes facultatively heterochromatic and remains inactive. Constitutive heterochromatization affects regions within the chromosome, and facultative heterochromatization affects the entire chromosome. Genes are known to exist in facultatively heterochromatic regions, but there is no evidence that they exist in the constitutive regions (Brown, 1966; Mittwoch, 1973).

Approximately half of the active X chromosome is constitutively heterochromatic. The inactive X and the Y chromosomes are facultatively heterochromatic. It is important to remember that the Y chromosome is only about one third the size of the X. In comparison, females still have more heterochromatin than males because of the difference in size between X and Y chromosomes (Mittwoch, 1973). Mittwoch postulates that the heterochromatin content of an extra Y chromosome causes only a slight decrease in the rate of growth, while the presence or absence of X chromosomes is much more influential in determining increases or decreases in growth rates.

Barlow's (1973) study on the effects of inactive extra X and Y chromosomes shows that the mitotic cycle of the cell is slower when these extra chromosomes are present. He supports this theory with data showing lower birth weights, ridge counts, heights and immunoglobulin levels in individuals with supernumerary X and Y chromosomes (Barlow, 1973).

The proposition in this study is that growth rate controlling factors are influenced by the constitution and presence of X and Y chromosomes. These factors regulate the development of specific target tissues at specific times. Secretion of hormones provides an excellent example of such factors. Hormones and other cellular products are controlled by protein synthesis in the cells where they are produced (Commoner, 1964; Vander et al., 1975). Protein

synthesis is controlled by the mitotic rate of those cells; and for this reason, heterochromatin could influence hormone production. Hormones are known to affect the mitotic cycle of the cells in their target tissues (Epifanova, 1971). Mittwoch (1973) believes that soon after gonadal differentiation takes place, steroid hormones under the influence of the sex chromosomes affect the growth rate of specific target organs which yield the secondary sex characteristics. Whether or not the hormones are of male or female proportions may depend on the chromosomes present and their constitution.

For a more specific example which shows the wide-ranging effects that heterochromatin might be indirectly responsible for, the hormone oestradiol can be used. Oestradiol is a steroid of the oestrogen group which checks growth in females at a very early age, possibly by inhibiting the production of growth hormone (Greene, 1970). In terms of the theory set forth above, oestradiol would be responsible for slowing down the mitotic cycle among the eosinophil cells of the anterior pituitary, where the growth hormone is produced. A similar hypothesis could be postulated for a number of other hormones which affect growth. The important point here is that growth and development is a very complex and integrated process which is largely dependent on hormonal levels, which are dependent to some extent on sex.

Hamerton (1968) thinks that the heterochromatic regions of the X and Y chromosomes have controlling centers which affect sex determination and control certain somatic characteristics. He suggests that sex is determined in males by inhibiting cortical development of the gonad and stimulating the medullary area, while in females the reverse is true. The result of this is that the testicles are formed from the primary sex chords of the medullary portions of the gonad and the ovaries are formed from the secondary sex chords of the cortex. It is known that in the absence of testicular development, ovaries form (Vander et al., 1975). The testicles must therefore form in about the ninth week in utero several weeks earlier than the ovaries. The presence of the testicles earlier might cause male hormones to be active earlier, thus giving males a slight edge in development. This is only speculation.

Hamerton advocates that the Y chromosome acts as a medullary stimulator and cortical inhibitor. He notes that the heterochromatic X chromosome in females might regulate oestrogen production and therefore somatic development and growth rate. Hamerton has added to Greene's theory that X heterochromatin acts as a regulator for a number of metabolic pathways.

Hamerton believes that growth control centers are located on the Y chromosome and the short arm of the heterochromatic X. He theorizes that growth is controlled by the genetic effects of

heterochromatic DNA, which regulates certain metabolic pathways. What phenotypic expressions result depend on the specific amount of this DNA, its composition, and what time during development it is synthesized. Technically the phenotypic expressions are achieved by exposing the DNA template, or genetic code, at specific times to messenger RNA in the presence of RNA-polymerase. RNA-polymerase is an enzyme which binds free nucleotides in specific sequences to messenger RNA. Messenger RNA is the substance which transfers the genetically coded information to sites of protein synthesis called ribosomes in the cytoplasm. Here the chain of ordered nucleotides are used to order the sequences of amino acids necessary to form proteins. These proteins are utilized by the cell to build specific structures and produce specific chemicals such as hormones and enzymes (Vander et al., 1975; Brown, 1966).

The sequence of nucleotides on the messenger RNA is obviously important because it must be a mirror image of the DNA sequence to produce the desired amino acid chains. An alteration in this sequence might result in a polymorphism such as an abnormal hemoglobin (Buettner-Janusch, 1973) or a protein which affects growth. How heterochromatic DNA reacts during this process and how its amount affects this process may be important. The amount of heterochromatic DNA in the sex chromosomes might be directly proportional to the amount of time allowed for messenger RNA coding and

protein synthesis. This in turn may affect the performance of the RNA-polymerase. Commoner (1964) points out that the availability of free nucleotides used in synthesizing both DNA and messenger RNA may also be affected by concentrations of heterochromatin at different points on the chromosome. Such an elaborate theory explains how the basic genetic material is put into action, and how differing amounts of heterochromatic DNA might affect these actions to produce sexual differences.

Unlike euchromatin, the environment can affect the influence of heterochromatin. Commoner (1964) notes that the density of the heterochromatin varies with the physical condition of the cell (i. e., temperature, nutritional state, disease) and because of this it can influence the synthesis of DNA in euchromatic genes by some sort of diffusive mechanism. Heterochromatic regions fluctuate in size and density as a direct response to environmental changes (Commoner, 1964). This might explain why heat, cold and audio-genic stressors cause fluctuating asymmetry.

Considering the theories about heterochromatin developed so far, normal males (XY) would have a higher rate of growth than normal females (XX) because they have less heterochromatin in their chromosomes than females do. Consequently, males might be expected to have higher ridge counts and larger dental dimensions than females. The increased rate of growth may also be responsible for

higher asymmetry values in males. Jantz (Personal Communication) has recently found that as digital ridge counts increase, fluctuating asymmetry increases also, except for very high ridge counts where low asymmetry is the rule. To confirm this, the investigator made a series of graphs plotting right plus left digital ridge counts against right minus left digital ridge counts for corresponding digits. The results follow Jantz's observations. Lower ridge counts are accompanied by lower asymmetry values, while higher ridge counts correspond with higher asymmetry values. Very high counts have low asymmetry values. This seems to indicate that digital and palmar patterns develop in a similar fashion, since they are affected by fluctuating asymmetry in the same manner.

The literature reveals that males generally have higher ridge counts than females (Parsons, 1964; Holt, 1968; Jantz, 1975). The same holds true for mesiodistal and buccolingual measurements and tooth shape variability (Goose, 1963; Garn et al., 1964b; Garn et al., 1967b; Rosenzweig, 1970; Moss and Moss-Salentin, 1977).

To further develop the theories which are basic to this study, it is necessary to consider the developmental time involved in the formation of the dentition and the dermal ridges. The permanent dentition represents a polygenic system which develops over a considerable length of time, from about .2 years of age (genesis of the first permanent molar) until about 12.3 years of age (completion of

the permanent third molar crown) (Moorrees et al., 1963). During this time the permanent dentition can be subjected to a profusion of environmental stressors, any of which might be responsible for some degree of fluctuating asymmetry (Perzigian, 1977). On the other hand, the primary dermal ridges are completely formed by the fourth month in utero (Holt, 1968). This does not allow much time for developmental anomalies to occur. The dermal ridges are relatively devoid of the stressors to which the teeth are subjected.

If there are significant correlations between the raw measurements and between the asymmetry values in these two genetic systems with mutually exclusive developmental periods, they would provide an excellent basis for expanding the idea of genetic controlling factors covering more than a single polygenic system or developmental period. This returns to the theory propounded by Mittwoch, that growth rate is determined at least in part by the amount of heterochromatin present in the individual's sex chromosomes. Differences in growth rate would be reflected in sexual differences among the variables of size, shape and asymmetry.

The theories concerning heterochromatic function might help explain the phenomenon of third molar agenesis related anomalies. Garn et al. (1963a, b; 1966a) noted that there is a higher incidence of third molar agenesis in females than males; however, when third molar agenesis occurs in males, it is accompanied by a greater

degree of size reduction in other teeth than when it occurs in females (Garn et al., 1963a, b; Baum and Cohen, 1971).

It is generally accepted that some phenotypic expressions are controlled by polygenes. The dentition is probably one such expression. Commoner (1964) has illustrated that in Drosophila polygenes are commonly located in heterochromatic regions of the chromosomes. Mittwoch (1973) has noted that genetic anomalies which result from alterations in heterochromatic material, do not have deleterious effects on the organism. It may be postulated that polygenes residing in heterochromatic regions are not essential to the survival of the organism. Brown (1966) implies that constitutive heterochromatization may be an evolutionary process by which unneeded genes are inactivated and eventually phased out of the chromosome. Certain aspects of the dentition such as tooth size and third molar agenesis might be placed in the non-essential category. If the genes responsible for tooth size reduction and agenesis are intimately related biochemically, reside in a heterochromatic region, and are in the process of being eliminated through heterochromatization, then tooth size reduction and third molar agenesis might be expected to occur simultaneously. The fact that females have a higher occurrence of third molar agenesis but less tooth size reduction may be a direct response to their greater amount of heterochromatin, slower growth rate and greater developmental stability. The same

principle would hold true for males with third molar agenesis. Tooth size reduction might be greater in males because of lower amounts of heterochromatin and a more intimate relationship between the genetic material responsible for these two dental conditions. Sexual differences in heterochromatin quantity and/or density might be responsible for other sexually dimorphic traits as well.

If one considers heterochromatin as an evolutionary mechanism, it might be employed by scholars of hominid evolution to help explain reduction in sexual dimorphism in the genus Homo over time. If this were done, then size would seem to be the major variable involved. Unfortunately, there is no way to prove such a theory.

A similar model to the one created for the dentition might also be constructed around the dermal ridges. The polygenes responsible for them could be located in heterochromatic regions of a chromosome, while the diversity which they exhibit may indicate that they are not essential to the survival of the individual. This also implies that they are not under stringent genetic control.

These theories are highly speculative and rest on assumptions for which there is little evidence. The key to understanding the effects of heterochromatin on growth rate and stability seems to be in the amount of time allowed for messenger RNA coding and the condition of the DNA active in the coding process. This might affect the proteins synthesized and therefore the specific products of the cells.

Time allowed for coding messenger RNA seems to be dependent in part on the amount of heterochromatin in the chromosome, which affects the mitotic cycle of the cell. This appears to be a complex feedback system.

Following the hypothesis that heterochromatin affects growth rate and that this differs between the sexes, one might examine the possibility that it affects the degree of fluctuating asymmetry in the teeth. Garn et al. (1967a) have shown that males have larger dental dimensions and more fluctuating asymmetry than females do. In another article, Garn et al. (1967b) found that there are sex differences in tooth shape. This can be treated as a form of asymmetry.

The author inspected several charts from an article by Moorrees et al. (1963) which deals with the developmental time involved in the formation of the permanent mandibular canines, premolars and molars. Simple computations revealed that overall mean developmental times (cusp formation plus root formation) are usually more rapid in females, while developmental times for crown formation (cusp initiation to crown completion) are usually more rapid in males (see Table 1). The latter point is important because the crowns are the measurable traits. Females develop faster in the roots, and this accounts for their earlier eruption sequences. As in most biological data, there are a few exceptions to the rules.

Table 1. Mean developmental time and sex differences for formation of the permanent mandibular dentition (in years).

Tooth	Cusp			Cusp and Root		
	Initiation	Completion	Sex Differences	Initiation	Completion	Sex Differences
Canine						
Male	.5	2.2	1.7	.5	13.2	12.7
Female	.5	4.0	3.5	.5	11.4	10.9
Difference			1.8			1.8
First Premolar						
Male	1.8	5.2	3.4	1.8	13.4	11.6
Female	1.8	5.0	3.2	1.8	12.2	10.4
Difference			.2			1.2
Second Premolar						
Male	3.2	6.3	3.1	3.2	14.4	11.2
Female	2.9	6.3	3.4	2.9	13.7	10.8
Difference			.3			.4
First Molar						
Male	.2	2.2	2.0	.2	9.5	9.3
Female	.2	2.5	2.3	.2	9.0	8.8
Difference			.3			.5
Second Molar						
Male	3.7	6.4	2.7	3.7	14.8	11.1
Female	3.4	6.1	2.7	3.4	13.9	10.5
Difference			.0			.6
Third Molar						
Male	9.3	12.0	2.7	9.3	20.0	10.7
Female	9.5	12.3	2.8	9.5	20.7	11.2
Difference			.1			.5

The greatest sexual difference in developmental time is in the canine. In males the average time is 1.7 years, while in females it is 3.5 years, a difference of 1.8 years. It is generally accepted that the canine is the most sexually dimorphic and stable tooth in the human dentition (larger in males) (Garn et al., 1966b). It is only logical to assume that if there is greater fluctuating asymmetry in the canines of males, it might be attributed to the velocity at which they develop. In this study, tooth diameters do not conform to this theory as well as the crown index utilized (mesiodistal diameter/buccolingual diameter). Crown indices measure tooth shape; and according to the data in this study, asymmetry is higher in the male indices than the female indices. Crown indices are higher in females, indicating that the crown dimensions are more homologous. These data imply that males are not only more asymmetrical in canine shape, but more asymmetrical in intra-canine dimensions. This observation is generally true for the rest of the dentition as well.

CHAPTER III

OBJECTIVES

The prime objective of this study is to examine raw dental measurements and dermal ridge counts and to extract from them values which reflect the degree of fluctuating asymmetry present in these two genetic systems. Inter-systemic comparisons between raw values and asymmetry values are then made.

The investigator is searching for statistically significant correlation coefficients between these two systems, one of which develops quite rapidly early in utero, while the other develops more slowly in a post natal environment (permanent dentition). If correlation coefficients are significant between the raw values, one might formulate the hypothesis that the two systems are controlled by common factors throughout development. If correlations between asymmetry values are significant, developmental stability can also be considered to be controlled similarly throughout development. If heterochromatin is responsible for these control mechanisms, sexual differences in correlations will provide evidence, since differences in heterochromatin content are supposed to affect growth rate and somatic expressions (Hamerton, 1968).

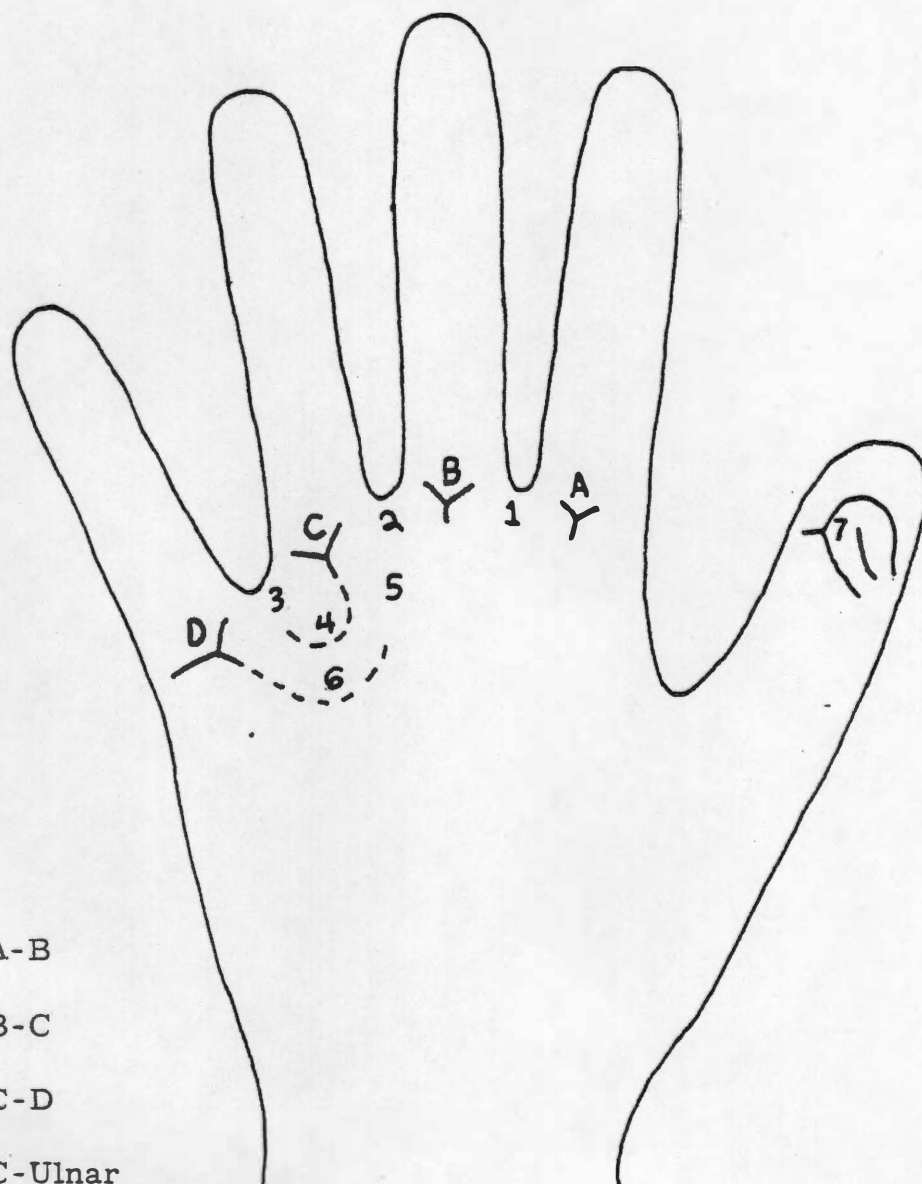
A combination of significant raw and asymmetry values would support the proposed hypothesis that heterochromatin controls growth rate and developmental stability. This is the desired goal. This study is concerned with presenting evidence that genetic material influences the general growth rate, and therefore the development of the dermal ridges and permanent dentition. Fluctuating asymmetry is simply a reflection of how well this control mechanism works, while raw values demonstrate that the mechanism is present.

CHAPTER IV

MATERIALS AND METHODS

The data for this investigation were collected from students of Euro-American descent in introductory anthropology classes during the Fall quarter, 1976, Winter quarter, 1977 and Spring quarter, 1977, at the University of Tennessee in Knoxville. Fifty males and fifty females compose the sample. For each individual, digital and palmar prints were taken in the usual manner using printer's ink. Dental impressions were taken using Jeltrate casting compound and Castone plaster. Each individual was also required to fill out two questionnaires, one pertaining to human variation and the other to dental history. All ridge counts were taken under a low powered binocular microscope, and all dental measurements were taken with a Helios dial caliper with modified measuring arms to the nearest half millimeter. Once all of the data were properly recorded, they were punched on standard 80-column computer cards. All computer analysis was done on the IBM 360 computer at the University of Tennessee Computer Center.

The measurements taken from the dermal ridges included all digital ridge counts and six palmar ridge counts (a-b, b-c, c-d, d-radial, c-ulnar, c-radial). Figure 1 illustrates where each ridge count is located.



- 1) A-B
- 2) B-C
- 3) C-D
- 4) C-Ulnar
- 5) C-Radial (not shown)
- 6) D-Radial
- 7) Digital Ridge Count

Figure 1. Ridge counts used in study.

The method used for recording ridge counts is taken from Holt (1968). All primary ridges, bifurcations and islands crossing an imaginary line from the triradius to the core are counted, while secondary ridges are not.

The buccolingual and mesiodistal diameters for all permanent teeth present were used, with the exception of the third molars. Both of these tooth diameters are defined by Bass (1971) respectively, as the maximum distance between the mesial and distal contact points of the crown and the maximum distance from the buccal edge to the lingual edge of the crown, at a right angle to the mesiodistal diameter. Since dental casts were used, the contact points were not usually accessible, so the points nearest to them were used.

The statistical analysis in this study is broken down into four basic steps. The first of these is simply the computation of summary statistics (means, standard deviations) for all males and females, rights and lefts. These computations reveal sex differences in size, shape and variance.

The second step entails the calculation of asymmetries for the dentition and the dermal ridges for all individuals. Three methods are used to calculate fluctuating asymmetry, since certain problems are unique to digital ridge counts and interdigital pattern ridge counts.

With the digital and interdigital palmar counts (d-radial, c-ulnar and c-radial) there is the problem of limitation because only a certain

number of ridges can possibly develop in the area between the pattern triradius and its core. Another problem is that as an individual's pattern ridge count increases initially, so does his fluctuating asymmetry; however after a certain point, the fluctuating asymmetry drops markedly (with high ridge counts). Figures 2 through 5 demonstrate this. In such a situation, a mere percentage figure does not reflect the true relationship between the ridge counts and their asymmetry values. This would only produce a bias, since all pattern ridge counts would be treated equally, and not relatively as is statistically correct. If these two problems are not corrected for in the pattern-type ridge counts, then all ridge counts would be treated equally, creating yet another bias.

The above problems were solved by utilizing a correction factor worked out by Jantz (personal communication). It involves calculating contingency tables of the absolute value of right minus left ($R-L$) by right plus left ($R+L$), for all digital and interdigital patterns. The table places each ridge count in an interval, each covering two ridges. Once each individual is placed in the proper interval at the proper asymmetry level, a maximum asymmetry value is recorded for every ridge count interval. The maximum asymmetry value is then divided by the absolute value of each individual's right minus left ridge count value, in that ridge count interval. This exercise produces an asymmetry value, in percent, which is relative to the

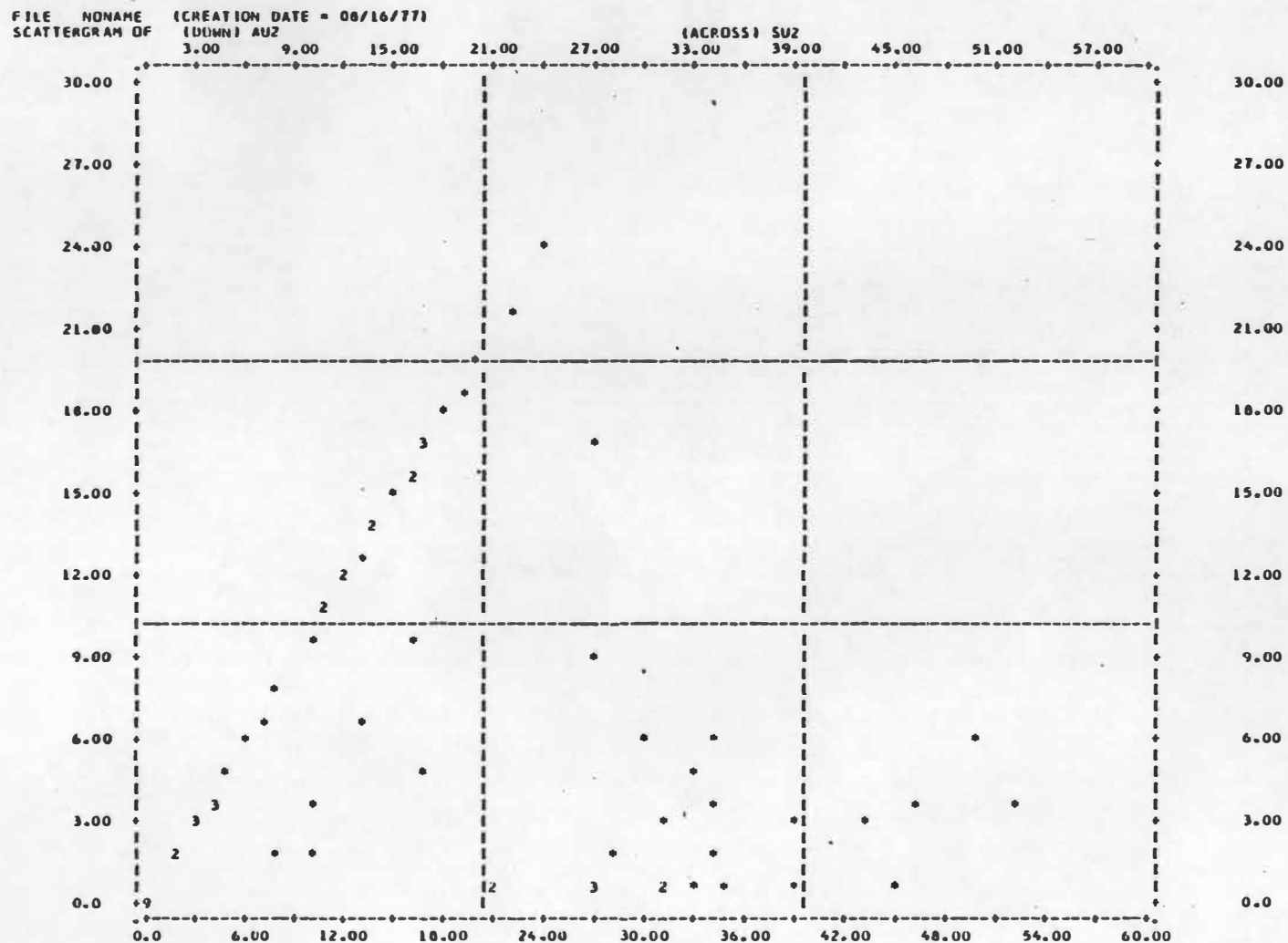


Figure 2. Absolute differences of right minus left (x-axis) versus right plus left (y-axis) for individual D-radial ridge counts.

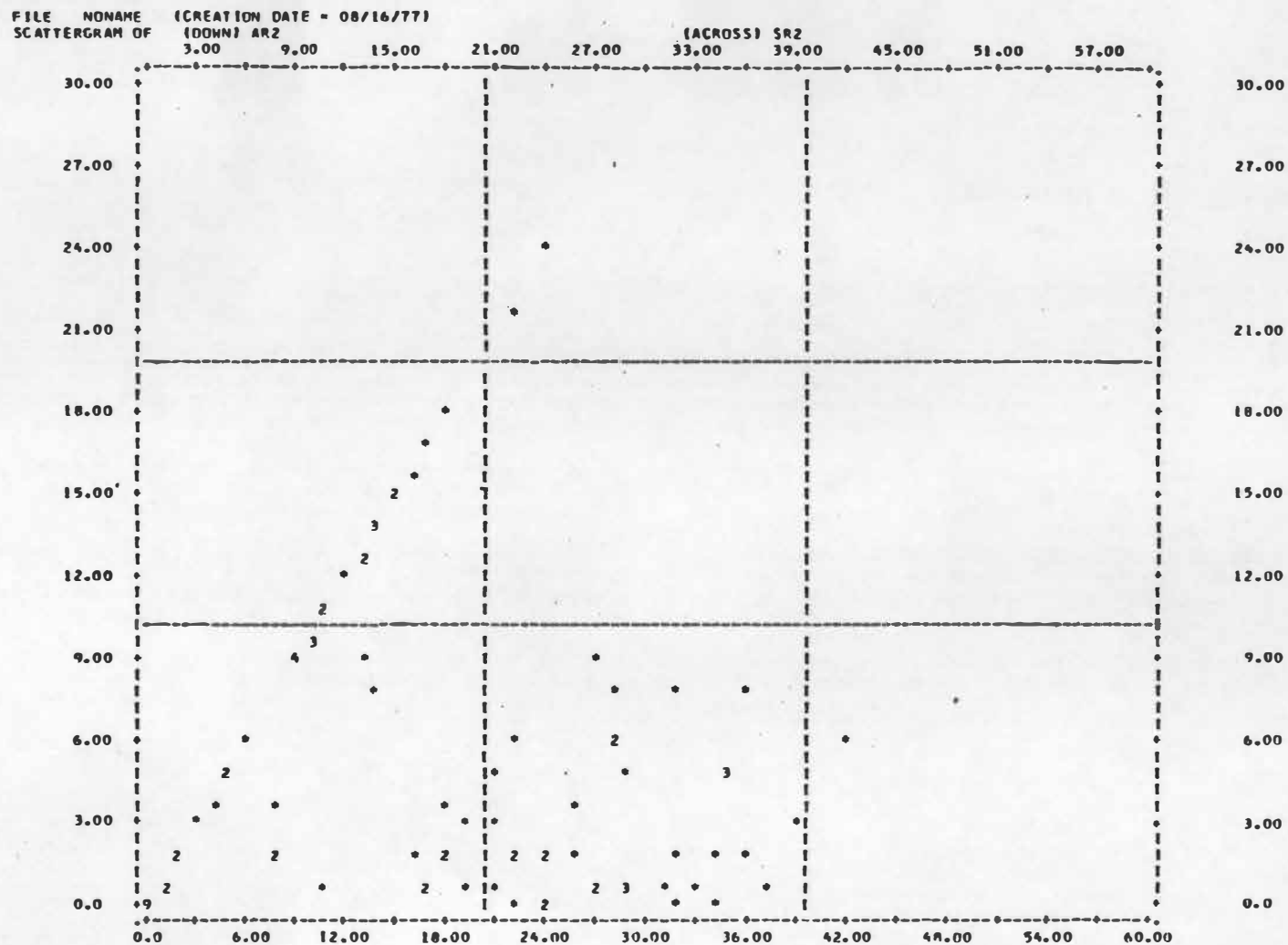


Figure 3. Absolute difference of right minus left (x axis) versus right plus left (y axis) for individual C-ulnar ridge counts.

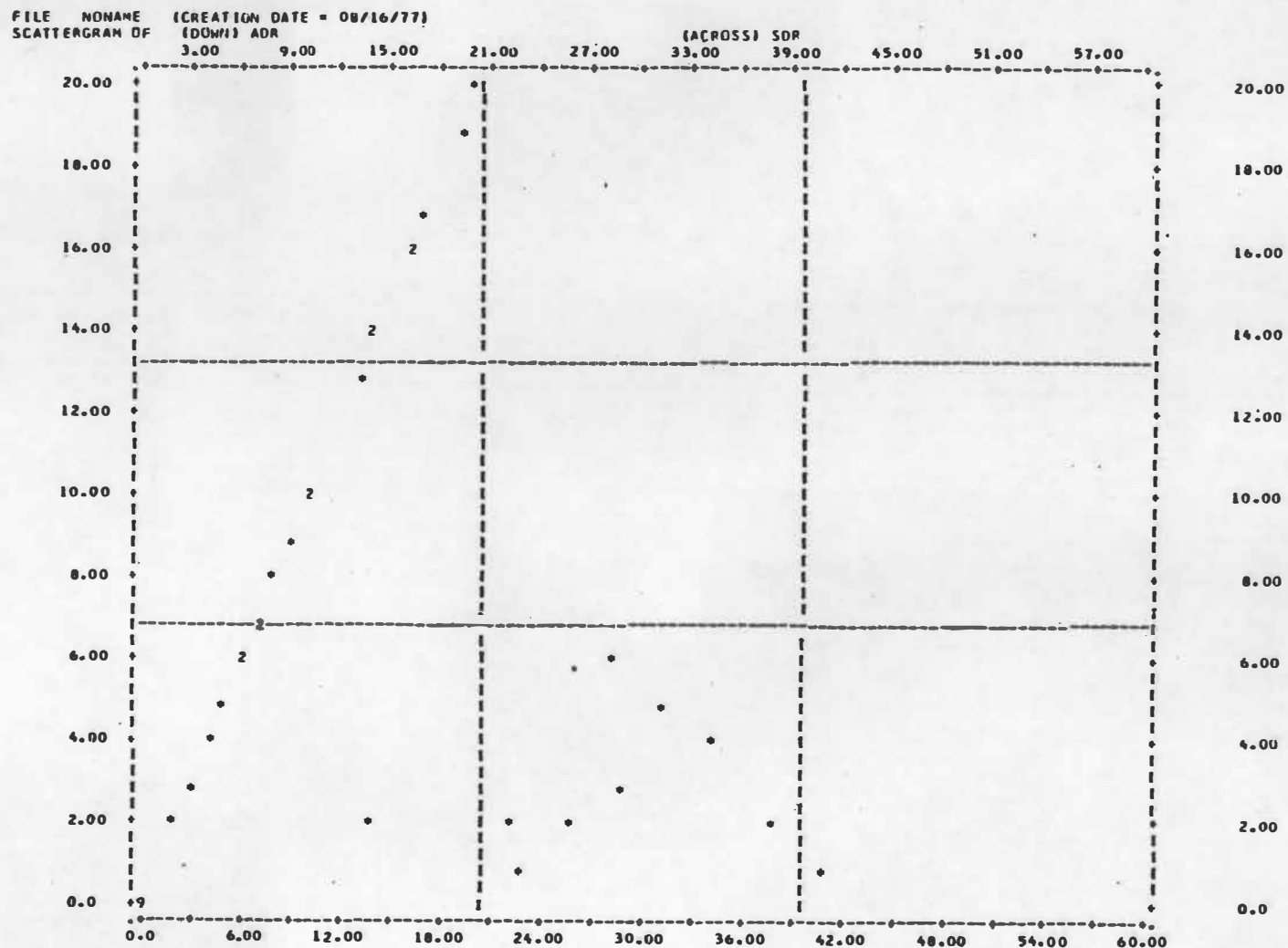


Figure 4. Absolute difference of right minus left (x axis) versus right plus left (y axis) for individual radial ridge counts of Digit II.

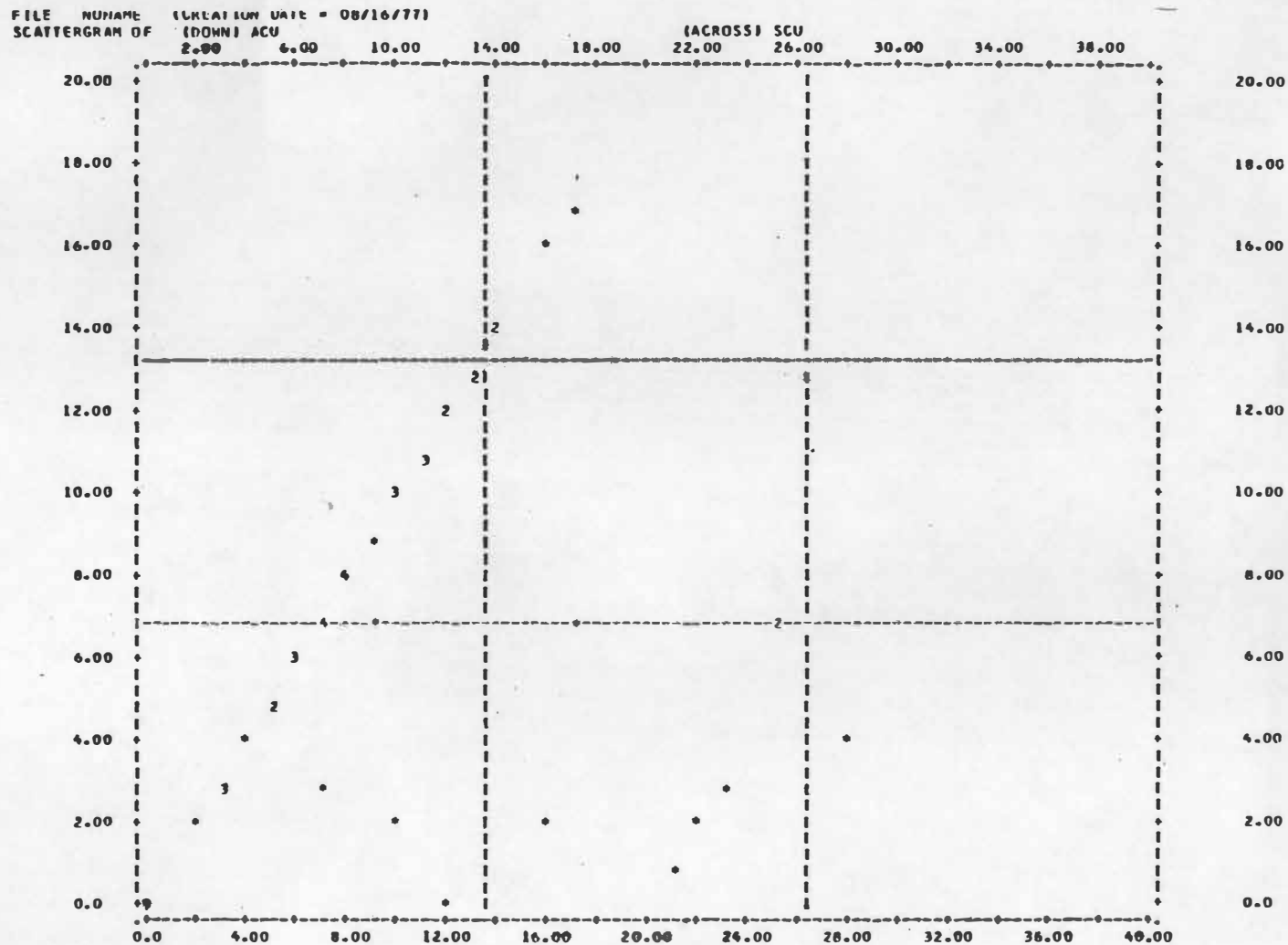


Figure 5. Absolute difference of right minus left (x axis) versus right plus left (y axis) for individual ulnar ridge counts of Digit II.

number of ridges on a given digit or palm, for a given individual.

This procedure was done for all intervals, radial and ulnar, for all digital and palmar pattern-type counts.

Another method for measuring fluctuating asymmetry was incorporated whenever it was relevant (dental and non-pattern ridge counts). This involves taking the Pearson product-moment correlation coefficients between corresponding right and left measurements. The values of the coefficients reflect homogeneity if they are high (as r approaches one, the variables being correlated approach equality), and heterogeneity if they are low (as r approaches zero, the variables being correlated become more dissimilar). This method is often more helpful in examining fluctuating asymmetry than the absolute value of right minus left, because it reflects correlations which simple mean differences can not.

An interjection might be made here about the sign accompanying the correlation coefficient. This indicates whether the variables being correlated are proceeding in the same general direction. If they do, the coefficients will be positive; if they do not the sign will be negative. For example, if dental dimensions increase as ridge counts increase one would expect positive correlations and vice versa. The sign's usefulness will become more apparent in the next chapter.

Step three requires that all raw dental values be correlated with all raw dermal values. Using this method, significant correlations

were identified and relationships between tooth size and shape and ridge counts could be established. The same thing was done with the asymmetry values. Correlation coefficients were first computed on the population as a whole, and then by sex to examine sexual differences.

The final step in this analysis involves the use of canonical correlation. This is the process of correlating one set of variables (dental) with another (dermal), and deriving a linear combination from each, such that the correlation between them is maximized (Nie et al., 1975). In canonical correlation the object is to account for as much of the variance as possible between the two sets of variables. This "accounting" is reflected in what is called an eigenvalue. In order to directly measure the amount of correlation between the two sets of variables, in which all within-group correlation has been removed, a value called a canonical correlation is presented. This is simply the square root of the eigenvalue and can be read exactly the same as a Pearson product-moment correlation coefficient.

This procedure is tested for significance by means of chi-square. A significance level of .05 is considered acceptable, but all correlations with a significance level of .10 or less is considered worthy of mention.

All summary statistics, Pearson product-moment correlation coefficients and canonical correlations were done utilizing SPSS

package programs (Nie et al. , 1975), while computation of asymmetry and corrected asymmetry values were done in part manually, and partly using Fortran computer programs.

In the following chapter this statistical probe provides evidence for the theories supporting generalized controlling agents over growth rate and developmental stability, prenatally and postnatally. The results indicate that there are sexual differences in these agents.

CHAPTER V

RESULTS AND DISCUSSION

Results were obtained through the statistical analysis just discussed and will be presented in the same order. Each section refers the reader to tables containing the appropriate statistics, followed by an assessment of any significant values, and how they support or refute the theories postulated about growth rate, developmental stability and related sex differences.

The summary statistics (Tables 2 through 4) provide a general verification of what is already known; males generally have greater dental dimensions and dermal ridge counts than females. Males show larger mesiodistal and buccolingual measurements in both dental arches (Table 2) and have higher ridge counts in 100% of the radial ridge counts and 70% (7 of 10) of the ulnar counts (Table 4). On the palms, females have higher ridge counts in all non-pattern-type ridge counts (6), and in 50% (3 of 6) of the interdigital pattern-type counts. This deviates greatly from the proposed hypothesis. Females also show more and higher palmar asymmetry values than males (83%).

Females have higher values in 72% (20 of 28) of the crown indices (Table 3). It must be remembered that the crown index utilized here

Table 2. Means, standard deviations, mean asymmetry values and right-left correlation coefficients for dental measurements (in millimeters).

Variable	Males				Females			
	Mean	S. D.	Asym.	r	Mean	S. D.	Asym.	r
Mesiodistal								
URM2	10.33	.61	.40	.638	10.04	.75	.30	.867
ULM2	10.17	.61			10.05	.65		
URM1	10.75	.66	.24	.888	10.40	.68	.27	.874
ULM1	10.70	.63			10.37	.67		
URPM2	6.72	.45	.24	.807	6.48	.48	.24	.603
ULPM2	6.78	.44			6.55	.57		
URPM1	6.81	.34	.17	.812	6.58	.44	.14	.911
ULPM1	6.80	.38			6.57	.41		
URC	7.72	.44	.17	.841	7.33	.46	.19	.871
ULC	7.69	.42			7.38	.44		
URI2	6.47	.67	.23	.914	6.40	.66	.25	.808
ULI2	6.49	.72			6.47	.50		
URI1	8.58	.81	.18	.921	8.19	.54	.20	.872
ULI1	8.63	.83			8.20	.49		
LRM2	10.80	.64	.13	.762	10.33	.78	.31	.826
LLM2	10.79	.58			10.25	.69		
LRM1	11.29	.63	.32	.850	10.72	.63	.23	.897
LLM1	11.17	.63			10.70	.64		
LRPM2	7.25	.54	.22	.743	6.90	.44	.20	.861
LLPM2	7.22	.48			6.91	.52		
LRPM1	6.98	.41	.28	.789	6.60	.33	.17	.790

Table 2 (continued)

Variable	Males				Females			
	Mean	S. D.	Asym.	r	Mean	S. D.	Asym.	r
Mesiodistal								
LLPM1	6.92	.38			6.66	.31		
LRC	6.70	.39	.21	.885	6.26	.40	.18	.850
LLC	6.71	.37			6.27	.42		
LRI2	5.87	.42	.15	.866	5.65	.44	.17	.880
LLI2	5.88	.40			5.63	.41		
LRI1	5.33	.38	.17	.820	5.19	.37	.15	.843
LLI1	5.31	.35			5.18	.35		
Buccolingual								
URM2	11.77	.73	.31	.817	11.04	.61	.27	.875
ULM2	11.79	.66			11.18	.70		
URM1	11.84	.59	.21	.905	11.20	.69	.20	.913
ULM1	11.80	.64			11.18	.65		
URPM2	9.55	.62	.18	.911	9.01	.64	.20	.910
ULPM2	9.54	.62			9.01	.63		
URPM1	9.21	.54	.24	.824	8.92	.52	.16	.929
ULPM1	9.18	.48			8.87	.48		
URC	8.13	.59	.22	.892	7.72	.55	.24	.853
ULC	8.16	.63			7.63	.54		
URI2	6.19	.54	.25	.821	5.92	.73	.30	.833
ULI2	6.17	.57			5.90	.68		
URI1	7.18	.52	.28	.781	6.81	.57	.26	.820

Table 2 (continued)

Variable	Males				Females			
	Mean	S. D.	Asym.	r	Mean	S. D.	Asym.	r
Buccolingual								
ULI1	7.16	.56			6.84	.58		
LRM2	10.60	.65	.32	.728	10.13	.53	.29	.708
LLM2	10.58	.57			10.12	.60		
LRM1	10.80	.59	.26	.909	10.33	.56	.24	.829
LLM1	10.84	.68			10.38	.56		
LRPM2	8.52	.58	.28	.845	8.12	.58	.20	.871
LLPM2	8.58	.63			8.19	.52		
LRPM1	7.88	.49	.22	.858	7.37	.47	.20	.815
LLPM1	7.92	.50			7.43	.46		
LRC	7.36	.59	.24	.841	6.84	.45	.23	.821
LLC	7.38	.52			6.85	.47		
LRI2	6.20	.49	.30	.656	5.90	.43	.23	.776
LLI2	6.17	.46			5.88	.41		
LRI1	6.02	.49	.27	.689	5.69	.35	.19	.820
LLI1	6.06	.67			5.69	.42		

Table 3. Means (in percent), standard deviations, mean asymmetry values and correlation coefficients for crown indices.

Variable	Males				Females			
	Mean	S. D.	Asym.	r	Mean	S. D.	Asym.	r
Crown Indices								
URM2	.866	.06	.04	.638	.902	.05	.04	.674
ULM2	.882	.06			.911	.06		
URM1	.907	.05	.03	.730	.928	.05	.04	.547
ULM1	.908	.05			.930	.05		
URPM2	.712	.03	.02	.526	.728	.05	.03	.617
ULPM2	.705	.03			.722	.05		
URPM1	.742	.03	.03	.549	.741	.04	.02	.783
ULPM1	.740	.04			.738	.04		
URC	.944	.06	.04	.595	.969	.07	.04	.659
ULC	.952	.06			.951	.06		
URI2	1.055	.11	.06	.753	1.107	.13	.06	.843
ULI2	1.048	.11			1.090	.13		
URI1	1.213	.11	.05	.777	1.206	.12	.07	.756
ULI1	1.202	.10			1.209	.12		
LRM2	1.021	.05	.04	.494	1.013	.05	.04	.544
LLM2	1.020	.06			1.020	.06		
LRM1	1.032	.05	.03	.615	1.033	.06	.03	.629
LLM1	1.047	.05			1.039	.04		
LRPM2	.845	.07	.04	.732	.845	.05	.04	.649

Table 3 (continued)

Variable	Males				Females			
	Mean	S. D.	Asym.	r	Mean	S. D.	Asym.	r
Crown								
Indices								
LLPM2	.854	.08			.852	.06		
LRPM1	.876	.05	.04	.616	.897	.06	.03	.667
LLPM1	.887	.05			.899	.06		
LRC	.913	.07	.04	.787	.919	.07	.04	.799
LLC	.914	.08			.918	.06		
LRI2	.957	.09	.05	.656	.961	.07	.05	.657
LLI2	.952	.09			.961	.07		
LRI1	.886	.10	.05	.760	.913	.07	.04	.748
LLI1	.891	.09			.915	.07		

Table 4. Means, standard deviations, mean asymmetry values and correlation coefficients for digital and palmar ridge counts (in ridges).

Variable	Males				Females			
	Mean	S. D.	Asym.	r	Mean	S. D.	Asym.	r
Digital								
Radial								
R5	15.60	5.90	.46	.589	12.46	5.96	.49	.750
L5	14.12	4.92			11.26	5.41		
R4	15.36	7.94	.61	.699	14.62	6.41	.52	.696
L4	15.94	6.02			12.90	7.22		
R3	11.26	5.34	.47	.660	10.32	6.15	.62	.758
L3	11.80	5.75			9.96	6.97		
R2	10.02	7.64	.58	.567	8.60	6.63	.41	.404
L2	7.70	7.09			6.30	6.22		
R1	17.78	6.84	.63	.561	16.76	6.68	.49	.684
L1	16.44	5.83			14.04	5.79		
Ulnar								
R5	3.66	7.46	.95	.257	2.24	4.35	.93	.330
L5	1.02	3.35			1.76	4.38		
R4	6.52	7.46	.87	.611	5.12	7.25	.86	.784
L4	4.54	7.30			4.48	7.49		
R3	1.88	5.47	.99	.581	1.90	4.55	.97	.366
L3	2.32	5.82			1.98	4.75		
R2	6.36	7.64	.82	.525	5.62	8.20	.84	.565
L2	7.44	8.23			5.94	8.55		

Table 4 (continued)

Variable	Males				Females			
	Mean	S. D.	Asym.	r	Mean	S. D.	Asym.	r
Ulnar								
R1	4.06	7.31	.94	.483	5.30	7.76	.89	.539
L1	4.70	8.06			4.24	7.11		
Palmar								
RD-Rad	2.52	6.05	.97	.472	1.88	4.91	.97	.616
LD-Rad	3.06	6.03			2.38	4.78		
RC-Uln	2.42	4.33	.93	.062	2.14	4.63	.95	.333
LC-Uln	2.68	4.35			2.78	4.80		
RC-Rad	3.24	4.84	.92	.258	4.60	5.70	.86	.177
LC-Rad	1.42	3.18			2.18	4.24		
RA-B	36.02	6.82	2.66	.780	38.86	6.74	2.82	.797
LA-B	35.80	6.73			37.30	7.48		
RB-C	27.66	5.01	2.82	.775	29.30	5.55	3.36	.627
LB-C	27.04	5.57			29.06	4.97		
RC-D	40.96	4.19	3.62	.664	42.28	5.16	3.64	.695
LC-D	41.82	4.81			42.62	4.92		

(mesiodistal/buccolingual) reflects shape and not size. These higher values in females denote more uniform crown dimensions. This supports the speculation that females are more symmetrical and more stable during development. Males tend to have more elongated teeth, according to their crown indices (Table 3).

The idea that males grow faster and therefore achieve a greater size in a shorter developmental period is supported by the mean values in Table 2. This corresponds well with evidence shown earlier in Table 1 (page 21).

The hypothesis that males are more asymmetrical than females is not clearly demonstrable in the summary statistics. Visual scanning of means and standard deviations is not an acceptable method for determining the degree of fluctuating asymmetry. For this reason asymmetry values and correlation coefficients were included in the summary statistics tables.

In terms of percentages, males are more asymmetrical than females in 70% (7 of 10) of the corrected digital asymmetry values, and in 33% (1 of 3) of the corrected palmar values. Males had lower raw asymmetry values in all (3) of the non-pattern-type palmar counts (Table 4). This corresponds with the means of the palmar ridge counts, in that females generally have higher counts.

In the dentition the results are somewhat different. Raw asymmetry values of the tooth diameters (Table 2) in the maxillary dentition

show males with greater asymmetry values in only 43% (6 of 14) of the variables. All but one of these is associated with the posterior teeth. In the mandibular dentition males exhibit greater asymmetry in 86% (12 of 14) of the variables. Overall, males show higher asymmetry values in 64% of the raw dental measurements. Seven are mesiodistal diameters, while eleven are buccolingual. The raw asymmetry values obtained from the crown indices do not clearly reflect any sex differences, perhaps because the absolute value of right minus left does not reflect shape differences.

With the exception of the crown indices, the raw asymmetry values reflect sex differences which support the proposed theories, but the correlation coefficient is probably a better indicator of fluctuating asymmetry. Pearson product-moment correlation coefficients measure the degree to which variation in one variable is related to variation in another. They summarize the strength of the relationship between two variables (Nie et al., 1975). Absolute mean differences do not consider variability, and for this reason are not as accurate indicators of fluctuating asymmetry.

When correlation coefficients replace absolute differences, males show higher asymmetry (lower coefficients) in 57% (8 of 14) of the raw dental values in the maxillary teeth and 64% (9 of 14) of the values in the mandible. Overall, males are more asymmetrical in 60% of the measurements.

Even though this is a lower overall percentage than was obtained through absolute differences, it reflects less radical differences between the maxillary and mandibular teeth. Correlation coefficients reveal that males are slightly more asymmetrical in both arches, rather than extremely asymmetrical in one arch. This would seem more statistically correct since other investigators (Garn et al., 1966a) do not find extreme differences in asymmetry between the upper and lower dentition.

Correlation coefficients are presented with the digital and interdigital pattern-type ridge count data, but they do not reflect the true asymmetry between corresponding counts. Corrected values were calculated instead and have already been discussed.

The coefficients for the non-pattern-type palmar ridge counts reflect true relationships and sexual differences, contrary to what absolute differences show (Table 4). The coefficients show that males are more asymmetrical in the a-b and c-d ridge counts, while females are quite asymmetrical in the b-c ridge count. The absolute values showed that females were more asymmetrical in all three of these counts, which is not a true representation. It should be mentioned that these absolute differences were not corrected as were the other counts. This demonstrates the effectiveness of the correction factor and the superiority of the correlation coefficient as an indicator of fluctuating asymmetry.

Mean absolute differences apparently do not reflect shape differences well because they prove to be of little use when analyzing the crown indices for asymmetry. The correlation coefficient however, revealed sexual differences. Female indices were higher in 71% (10 of 14) of the coefficients. Female index correlations were higher in 70% (5 of 7) of the maxillary indices and 70% of the mandibular indices. This shows that females are generally more symmetrical in tooth shape than males. This corresponds with the index means, which are almost exactly the same, and demonstrates that females have more symmetrical intra-tooth dimensions and less asymmetry between teeth. Garn et al. (1967b) found significant sex differences in tooth shape and attributed them to greater variability in males. Intratooth uniformity and asymmetry in shape are not discussed in this article. This could be of particular importance when discussing developmental stability.

The results favor the theories proposed on growth rate and developmental stability. The developmental timing table (Table 1, page 21) demonstrates that male tooth crowns grow and develop faster. The summary statistics for the dental measurements (Table 2, page 37) support the hypothesis that faster growth and development in males yields larger tooth size. The means for the digits and palms also support the theory that males develop and grow faster than females (Table 4, page 42). The asymmetry values and correlation

coefficients generally support the idea that faster development not only yields greater size but also greater asymmetry.

The graphs on right plus left ridge counts versus their absolute differences, presented in Chapter II (Figures 2 through 4, pages 29 through 31), clearly show this. The crown indices lucidly show that males exhibit more intra-tooth and inter-tooth asymmetry than females. This is important because a crown index converts two size variables, each of which reflect unilineal development, into one shape variable which reflects multilineal development. The crown indices may therefore better represent developmental stability in terms of intra-tooth and inter-tooth asymmetry.

So far the data support the theory that the heterochromatic material in chromosomes is a good candidate for regulating growth and stability during development. The evidence also indicates that heterochromatin could be responsible for controlling the growth rate of systems developing during mutually exclusive time periods (i. e., prenatal versus postnatal). To this point the evidence has been indirect because no one-to-one correlations have been made between the dentition and the dermal ridges. We will now turn to direct evidence.

Tables 5 through 26 provide this evidence in the form of several hundred correlation coefficients (dental measurements versus dermal

Table 5. Correlation coefficients between raw dental measurements of the upper right quadrant and digital ridge counts - males ($p \leq .025$).

	BLM2	MDM1	BLM1	BLPM2	BLPM2	BLC	BLI2
LR4			-.3146	-.4003**			
LU4				.3246			
LR3				-.4375**	-.3783*		
LR2	-.3314			-.3722**			
LU2		-.2882	-.2859	-.3078		-.3802**	-.4734**
RU5							-.3676**
RR4				-.3336*			
RU4				-.2969			
RR3				-.3278*	-.3540*		
RU2	-.3128		-.2979	-.3065			

* $p \leq .01$

** $p \leq .005$

Table 6. Correlation coefficients between raw dental measurements of the upper left quadrant and digital ridge counts - males ($p \leq .025$).

	MDM2	BLM2	MDPM2	BLPM2	BLPM1	MDC	BLC	BLI2
LR4				-.3140				
LU4			-.3204	-.3139	-.3540*			
LR3				-.3340*		-.2835		
LR2				-.3777**				
LU2				-.2994			-.3188	-.4178**
LR1								-.2867
RU5							-.3144	
RR4	-.3504							
RU4	-.4175**	-.3480*	-.3119					
RR3			-.3045	-.3841**	-.3686*	-.3393*		
RR2					-.3825**			
RU2				-.2932				
RU1								

* $p \leq .01$

** $p \leq .005$

Table 7. Correlation coefficients between raw dental measurements of both lower quadrants and digital ridge counts - males ($p \leq .025$).

	RBLPM2	RBLPM1	RMDC	RBLC	RBLI2	RBLI1	LMDPM2	LBLPM2	LBLPM1	LMDC	LBLC	LBLI2
LR4			-.2863									
LR3		-.3264									-.2928	
LU3					-.4801**							
LR2						-.2985					-.3659**	
LU2			-.3333*	-.2888						-.3113	-.4909**	
LU1											-.3530*	
RR5											-.3004	
RU5			-.2839					-.3007	-.3031		-.3322*	
RR2	-.2819		-.4020**					-.3059			-.4589**	-.2924
RU2					-.3558*							
RU1				-.2933							-.3146	

* $p \leq .01$

** $p \leq .005$

Table 8. Correlation coefficients between raw dental measurements of the upper right quadrant and digital ridge counts - females ($p \leq .025$).

	MDM2	BLM2	MDPM2	MDPM1	MDC	BLC	BLI2	MDI1	BLI1
LR5					.5154**			.3112	
LR4					.4253**			.3779**	
LU4					.2978			.2906	.3101
LR3					.4416**			.3714**	
LR2		.3867*		.4111*	.3278				
LU2		.3741*						.4370**	
LU1			.3145			.2945			.3412*
RR5					.4290**			.5118**	
RU5							.3162	.3077	.3553*
RR4					.3811**			.3246	
RU4								.2839	
RR3	.3274	.3608*			.3761**			.4432**	
RR2		.3306							
RU2					.2808			.3967**	
RR1	.3535								
RU1			.3183		.2870			.3276	

* $p \leq .01$

** $p \leq .005$

Table 9. Correlation coefficients between raw dental measurements of the upper left quadrant and digital ridge counts - females ($p \leq .025$).

	MDM2	BLM2	MDM1	BLM1	MDPM1	MDC	BLC	MDI1	BLI1
LR5		.3218				.4071**			.2951
LR4						.3059		.2949	
LU4				.2966					.3305*
LR2		.4183**			.3762**				
LU2		.3233						.3365*	
LU1	.4140**								.3902**
RR5		.3023				.3651**		.4885**	
RU5								.3302*	.3619**
RR4									.2865
RU4								.3056	
RR3		.3103						.3312*	.3213
RR2							.3419*	.4032**	
RU2									
RR1	.4001*								
RU1						.3351*			

* $p \leq .01$

** $p \leq .005$

Table 10, Correlation coefficients between raw dental measurements of the lower right quadrant and digital ridge counts - females ($p \leq .025$).

	MDM2	BLM2	MDM1	BLM1	MDPM2	BLPM2	MDPM1	BLPM1	MDC	BLC	MDI2	BLI2	MDI1	BLI1
LR5				.3556*		.3480*	.3186	.3522*	.4577**					.3694**
LU5												.3112		
LR4				.4475**		.3311		.3404*	.3144					.3113
LU4				.4155**		.3071			.3379*					.2927
LR3				.4278**		.3483*			.3229					
LR2				.3197		.3946**			.2850					
LU2			.3839**	.4221**	.3226				.3485*		.2966	.3328	.3326*	
LU1	.4476**	.3833*	.3363*	.3651**	.3308				.3594**	.3136				.4141**
RR5		.2952		.4354**		.4023**			.4326**		.3010		.2825	.2876
RU5				.4143**		.2882			.2805					
RR4						.2986								.3088
RU4				.4194**										
RR3		.3648**	.5295**			.3663*			.3483*					.3137
RU3									.3137			.3546**		
RR2				.4426**		.4059**			.4250**	.3314*	.3738**		.3177	.3702**
RU2				.3128										.3216
RU1	.3051			.2839					.2909	.2955				.3216

* $p \leq .01$

** $p \leq .005$

Table 11. Correlation coefficients between raw dental measurements of the lower left quadrant and digital ridge counts - females ($p \leq .025$).

	MDM2	BLM2	MDM1	BLM1	MDPM2	BLPM2	MDPM1	BLPM1	MDC	BLC	MDI2	BLI2	MDI1	BLI1
LR5					.3273	.3354*			.4472*		.3852**		.3593*	
LR4					.3337**				.3620**		.3909**		.3407*	
LU4				.3582*	.3245				.3031		.2922		.2881	
LR3					.3004				.3825**		.3820**			
LR2		.3298*			.3568*	.3212	.3043		.2851					
LU2			.4253**	.3509*	.3098				.3480*		.3417*			
LU1	.4791**	.3111	.2957	.3635*	.2875	.2895	.3743*		.3709**			.3256	.3861**	
RR5					.2856	.3230			.4206**		.4361**	.2930	.2907	
RU5	.2982	.2931				.3255								
RR4									.3157		.2948		.3127	
RU4				.3224							.2859			
RR3	.3147	.3032	.3219	.3701**	.3375*	.3549*			.3979**		.3762**	.2820		
RU3													.3604*	
RR2	.2958	.3052		.2962	.2973	.3996**			.4473**	.2901	.4628**	.3302*	.3401*	
RU2													.3342*	
RR1									.3197					
RU1	.3715**													

* $p \leq .01$

** $p \leq .005$

Table 12. Correlation coefficients between raw dental measurements of both upper quadrants and palmar ridge counts - males ($p \leq .025$).

	RMDM1	RMDI2	RMDI1	RBLI1	LMDM1	LBLM1	LMDPM2	LMDPM1	LMDI2	LMDI1
LAB								-.3053		
LBC						.3165				
LCD						.3107				
LCU		.3364*							.2845	
RCD	.3141	-.2848			.3423*					
RDR			.2821	-.2804			.3029			.3121
RCU		-.3789**							-.3475*	

* $p \leq .01$

** $p \leq .005$

Table 13. Correlation coefficients between raw dental measurements of both lower quadrants and palmar ridge counts - males ($p \leq .025$).

	RBLM1	RBLPM1	RBLC	RMDI1	RBLI1	LBLM2	LBLM1	BLPM2	LBLC	LMFI1
LBC			.3964**				.2859		.3469*	
LCD	.3199					.2879	.4160**	.4323**		.2991
LDR		.3961**	.2891						.2972	
LCU				.2950				-.3261		
RBC		.3444*								
RCD	.3111						.3799**	.3291		
RCU					.3159					

* $p \leq .01$

** $p \leq .005$

Table 14. Correlation coefficients between raw dental measurements of both upper quadrants and palmar ridge counts - females ($p \leq .025$).

	RMDM2	RBLM2	RMDM1	RBLM1	RMDPM2	RMDC	RBLC	RBLI2	LMDM1	LBLM1	LMDC	LMDI2	LBLI2
LAB						.3384*						.3198	
LBC							-.2867						-.2986
LCD	.3611*												
LCU	.3164	.4102**	.3602*			.3301*		.3580*	.3456*				
RAB												.2940	
RBC												.2865	
RCU		.3585*	.3291	.4013**				.4354**		.3015			

* $p \leq .01$

** $p \leq .005$

Table 15. Correlation coefficients between raw dental measurements of the lower right quadrant and palmar ridge counts - females ($p \leq .025$).

	MDM2	BLM2	MDM1	BLM1	MDPM2	BLPM2	BLPM1	MDC	MDI2	BLI2	MDI1
LAB								.3115	.2822		
LBC							-.3042				
LCD										-.2932	
LCU	.2899	.2827		.3402*		.3272		.3139	.2827		
RAB								.2869	.2790		
RCD										-.3913**	
RCU	.3920**	.2908	.3642**	.3923**	.4341**				.3834**		.3608**

* $p \leq .01$

** $p \leq .005$

Table 16. Correlation coefficients between raw dental measurements of the lower left quadrant and palmar ridge counts - females ($p \leq .025$).

	MDM2	BLM2	MDM1	BLM1	MDPM2	BLPM2	BLPM1	MDC	MDI2	BLI2	MDI1	BLI1
LAB								.3185				
LBC							-.3235					
LCU	.3787**	.3315*		.4144*		-.3171		.3999**	.3288*			.3179
RAB								.3745**	.2976			
RCD										-.3045		
RCU	.4367**	.3839**	.4458**	.4409**	.4669**			.3209	.3167		.4160**	

* $p \leq .01$

** $p \leq .005$

Table 17. Correlation coefficients between crown indices of both upper quadrants and digital ridge counts - males ($p \leq .025$).

	RM1	RPM1	RI1	LM2	LPM2	LPM1	LC	LI1
LU5						.3539*		
LR3					.3508*			
LU3	.2831			.3095	.3404*		.3656**	
LU2			.2791				.2994	.3117
RR5								.2996
RU5			.3208					.4222**
RR2		.3272						
RU2				.2959				

* $p \leq .01$

** $p \leq .005$

Table 18. Correlation coefficients between crown indices of both lower quadrants and digital ridge counts - males ($p \leq .025$).

	RM2	RM1	RPM2	RPM1	RC	LM2	LM1	LPM2	LPM1	LC	LI2	LI1
LR5									.3106			
LR4									.3133			
LR3									.3032			
LU3												-.3493*
LU1					.3227							
RR5		.2814					.3030		.2989			
RU5		.3123	.3222					.2832				
RR4												-.3075
RR3												-.3155
RR2	.3240	.2871		.2986	.3995**	.2886	.3890**		.3558*	.3276*		
RU2												-.3002
RU1					.3992**						.3065	

* $p \leq .01$

** $p \leq .005$

Table 19. Correlation coefficients between crown indices of all quadrants and digital ridge counts - females ($p \leq .025$).

	ULM1	ULPM2	ULPM1	ULC	ULI2	ULI1	LRPM2	LRPM1	LRC	LRI2	LRI1	LLPM2	LLI1
LR5				.3678**					.2929				
LR3				.2935					.3203	.4052**			
LU3									.3001				
LR2													-.3165
LU2					.2824								
LR1						-.3776**							
LU1		.3098						.3659**					
RR5									.2860	.3439*			
RU5				-.3113									
RR3										.3180			
RU2				.3300*							-.3295		-.3433*
RR1	-.3019						-.2872						
RU1			-.3285										

* $p \leq .01$

** $p \leq .005$

Table 20. Correlation coefficients between crown indices of both upper quadrants and palmar ridge counts - males ($p \leq .025$).

	RPM2	RC	RI2	RI1	LPM2	LPM1	LI2	LI1
LAB				.3489*				
LCU	.3811**			.	.2788	.4133**	.3783**	
RAB					.3264*			
RCD			-.3117				-.3830**	
RDR		.2847		.3543*				.4586**
RCU			-.4095**				-.4297**	

* $p \leq .01$

** $p \leq .005$

Table 21. Correlation coefficients between crown indices of both lower quadrants and palmar ridge counts - males ($p \leq .025$).

	RM1	RPM2	RPM1	RC	RI1	LPM2	LPM1	LC	LI2	LI1
LCD	-.3840**									
LDR							-.3712*			
LCU		.4077**	.3950**			.4351**	.3613*			
LCR		-.2874		-.3054					-.3053	
RBC								-.2906		
RCD	-.2901									
RCU					-.2917					-.3759**

* $p \leq .01$

** $p \leq .005$

Table 22. Correlation coefficients between crown indices of all quadrants and palmar ridge counts - females ($p \leq .025$).

	URPM2	URPM1	URC	URI2	ULC	ULI2	ULI1	LRPM2	LRC	LRI2	LRI1	LLC	LLI2
LAB				.4062**					.2844	.4047**			.2987
LBC						.2899							
LDR	-.4189**								-.2882				
LCR							.2857		.2840				
RAB			.2959						.3032	.4027**			.3822**
RBC		.3562*											
RCD											.2913		
RDR					-.3039				-.3187			-.3003	
RCU								.3538*					

* $p \leq .01$

** $p \leq .005$

Table 23. Correlation coefficients between dental asymmetry values from the maxillary dentition and asymmetry values from digital and palmar ridge counts - males ($p \leq .025$).

	MDM2	MDM1	BLM1	MDPM2	BLPM2	MDPM1	BLPM1	MDC	BLC	BLI1
R3	.3554*		-.3167		.2911					
R2			-.3778**							-.3221
U2								-.3018	.3329*	
R1		-.3247								
AB							.3309			
CD				.3112		.3074				
DR					-.2902					

* $p \leq .01$

** $p \leq .005$

Table 24. Correlation coefficients between dental asymmetry values from the mandibular dentition and asymmetry values from digital and palmar ridge counts - males ($p \leq .025$).

	MDM2	BLM2	MDM1	BLM1	MDPM2	BLPM2	MDPM1	BLPM1	BLC	BLI2	MDI1	BLI1
R5	-.3463			-.3942**				-.3546*				
R4										-.3710**		
U4	-.4019**	-.4547**	-.3033		-.2856							
R3	.3526*											
U3									-.3100			
R2							.3371					
U2						-.2836					-.3938**	
AB			.3054									
DR										-.6138**		
CU												-.5352**
CR							-.3231					

* $p \leq .01$

** $p \leq .005$

Table 25. Correlation coefficients between asymmetry values from the maxillary dentition and those from the digital and palmar ridge counts - females ($p \leq .025$).

	MDM2	MDM1	MDPM1	MDC	BLC	MDI2	BLI2	MDI1	BLI1
R5			.3363						
U5						-.4083**			
R3					.3272				
R2	.3249								
U2									
R1					-.4002**				
AB			.3545*					-.3241	
CD		.3048	.3988*		-.2808		-.2958		
DR									-.3828**
CU				.2998					

* $p \leq .01$

** $p \leq .005$

Table 26. Correlation coefficients between asymmetry values from the mandibular dentition and those from the digital and palmar ridge counts - females ($p \leq .025$).

	MDM2	MDPM2	BLPM2	BLPM1	MDI2	BLI2
U4		-.2919				
R3	.2978				.3474*	
U1				-.3866*	-.2809	
AB				.3307		.3010
BC					.3022	
DR			-.5072**			
CU			-.3226			

* $p \leq .01$

** $p \leq .005$

measurements, dental asymmetry values versus dermal asymmetry values), all of which are significant at the .025 level, and many at the .005 level. If this does not provide firm evidence that there are universal controlling factors (heterochromatin) influencing growth and development both prenatally and postnatally, then it at least indicates that these two genetic systems are correlated.

It is important to make this last point because some of the correlations may be present because of the relationship between the two systems. The systems may have certain genes in common, which are suppressory in nature, or place them on the same level of genetic priority because they are no longer essential to the survival of the organism. Similar correlations may be present between any two polygenic systems because of their very nature. There may be a particular set of genes responsible for activating polygenic systems.

Commoner (1964) has noted that in Drosophila many polygenes are located in heterochromatic regions of the chromosomes. If this is also the case in man then all correlations would be due to heterochromatin, but some for different reasons than others. The possibility that polygenes may reside in heterochromatic regions and that heterochromatic content affects growth and development are two different causative agents. The first causes correlations between all polygenes that reside in heterochromatin, while the second causes correlations between polygenes because of the overall amount of heterochromatin

present and the possible chemical effects it might have on mitotic cycle. The second type of correlation probably predominates.

The author can say this with some degree of confidence because there are sexual differences in what variables correlate with each other. For example, in males, most of the digital ridge counts are highly correlated with the buccolingual diameters of the upper second premolars (Tables 5 and 6) and the lower left canine (Table 7). In females, the digits are correlated with the mesiodistal diameters of the upper right canine and central incisor (Table 8), the buccolingual diameter of the upper left second molar, and both diameters of the upper left central incisor (Table 9). Female digital counts are also strongly correlated with the buccolingual diameters of the lower right and left first molars, second premolars, central incisors and the mesiodistal diameters of the canines (Tables 10 and 11). It is obvious that females exhibit more significant coefficients than males.

There are sexual differences in the correlations between the dental measurements and the palms, but they are less obvious. Females show more significant coefficients than males (Tables 12 through 16). Correlations between crown indices and digits reveal a higher number in males than females (Tables 17 through 19), while the number of correlations between crowns and palms are closer to equal (Tables 20 through 22).

When the asymmetry values were correlated with each other, males and females had only three coefficients in common (Tables 23 through 26). Males tended to exhibit more negative coefficients and more correlations between digits and palms and the mandibular dentition. Males and females were almost equal in correlations in the maxillary dentition.

The fact that males and females infrequently produce the same sets of correlated asymmetry values is strong evidence for sexual differences in developmental stability. Generally, there is a great deal of sexual difference in what variables correlate with each other. When the raw dental values and ridge counts are considered, this may be a reflection of sexual differences in growth rate. What variables correlate with each other probably depends on what elements developed at the same rate. These would probably be different between the sexes if heterochromatin is responsible for growth rates.

With the sexual differences between correlation coefficients analyzed, we turn to another method of statistical analysis which provides verification that these two genetic systems are generally correlated without the consideration of sex. This technique is known as canonical correlation. As was explained earlier, canonical correlation correlates sets of variables and not single variables. Sample sizes were too small to run this procedure by sex when all cases of with missing data were removed. Table 27 shows the results obtained

Table 27. Canonical correlations between dermal and dental values, and supporting statistics.

Variables Correlated	Eigenvalue	Canonical Correlation	Significance
R. Radial and R. Palms vs. U. R. Mesiodistal	.4621	.6798	.002
R. Radial and R. Palms vs. L. R. Buccolingual	.3625	.6021	.021
R. Radial and R. Palms vs. Lower Mesiodistal Asymmetry Values	.4209	.6488	.090
R. Ulnar and R. Palms vs. L. R. Mesiodistal	.3457	.5880	.091
L. Radial and L. Palms vs. U. L. Mesiodistal	.4412	.6643	.003
L. Radial and L. Palms vs. Lower Mesiodistal Asymmetry Values	.3431	.5858	.034
L. Ulnar and L. Palms vs. U. L. Crowns	.4372	.6612	.103

Table 27 (continued)

Variables Correlated	Eigenvalue	Canonical Correlation	Significance
Right and Left Radial and Right and Left Palms vs. Upper and Lower Crown Asymmetry Values	.8714	.9335	.002
Digital and Palmar Asymmetry Values vs. L. L. Buccolingual	.4227	.6502	.059

through this statistical procedure. They generally support what has already been said.

The data were submitted by sides so that left teeth correlated with left dermatoglyphics and right teeth with right dermatoglyphics.

Radial and ulnar ridge counts had to be submitted separately because when submitted together they produced linear dependencies which would not allow the procedure to give true relationships between dental and dermal variables. The same was true of the mesiodistal and buccolingual tooth diameters.

The results seem to show general integration of the dental and dermal systems. The right radial and palmar ridge counts are correlated with the upper right mesiodistal and lower right buccolingual diameters, as well as the asymmetry values of the lower mesiodistal diameters. Right ulnar and palmar counts are correlated with mesiodistal diameters of the lower right quadrant. Left radial and palmar counts are correlated with the upper left mesiodistal diameters and the lower mesiodistal asymmetry values. Left ulnar and palmar counts are correlated with the upper left crown indices. Both right and left radial and palmar counts are correlated to the upper and lower crown index asymmetry values. This is somewhat unexpected since crown asymmetry values did not seem to show significant correlation coefficients earlier. This may be caused by a sample size bias.

The only case where digital and palmar asymmetry values were correlated was with the lower left tooth diameters.

These results provide evidence that dental and dermal systems are correlated. They may develop in similar fashions or at growth rates which are proportional to each other.

Overall this investigation has provided evidence that growth rate and developmental stability are controlled by similar if not the same agents. These agents influence both prenatal and postnatal development. Sexual differences are present in these agents and can be demonstrated in faster development of male tooth crowns, larger male tooth sizes, higher male ridge counts and generally higher asymmetry values in males. The reverse situation characterizes females.

The data fit well into the proposed models of growth and development regulation because they would be controlled by the quantity, distribution and biochemical effects of heterochromatic material in the chromosomes.

An important discovery is made with the correlation of the permanent dentition and the primary ridges of the digits and palms. This may be due to shared genetic material, shared residence in the chromosome (in heterochromatic regions), or similarities in growth and development. Most of the evidence seems to point to the last suggestion, but these results do not provide the basis needed to draw

such a conclusion. Instead they have stimulated discussion and will hopefully encourage further investigations in this area.

BIBLIOGRAPHY

BIBLIOGRAPHY

- Adams, M.S. and J.D. Niswander
1967 Developmental "Noise" and a Congenital Malformation.
Genetical Research 10: 313-317.
- Anderson, D.L., G.W. Thompson and F. Popovich
1975 Relation Between Weight, Socioeconomics and Maturity:
Dental, Skeletal, Statural, Sexual. Journal of Dental Research,
Special Issue A 54: 53 (Abstract).
- Bader, R.S.
1965 Fluctuating Asymmetry in the Dentition of the House Mouse.
Growth 29:291-300.
- Baillet, H.L.
1968 Maternal Effects on the Developing Dentition. Archives
of Oral Biology 13: 155-161.
- 1975 Dental Variation Among Populations, An Anthropologic View.
Dental Clinic of North America 19: 125-139.
- Baillet, H.L., J.D. Niswander and C.J. MacLean
1968 The Relationship Among Several Prenatal Factors and
Variation in the Permanent Dentition in Japanese Children.
Growth 32: 331-345.
- Baillet, H.L., P.L. Workman, J.D. Niswander and C.J. MacLean
1970 Dental Asymmetry as an Indicator of Genetic and Environ-
mental Conditions in Human Populations. Human Biology
42: 626-638.
- Barlow, P.W.
1973 The Influence of Inactive Chromosomes on Human Develop-
ment, Anomalous Sex Chromosome Complements and the
Phenotype. Human Genetik 18: 105-253.
- Bass, W.M.
1971 Human Osteology. Columbus, Missouri: Missouri Archaeo-
logical Society.
- Baum, B.J. and M.M. Cohen
1971 Patterns of Size Reduction in Hypodontia. Journal of Dental
Research 50: 779.

Brown, S.W.

1966 Heterochromatin. *Science* 151: 417-425.

Buettner-Janusch, J.

1973 *Physical Anthropology: A Perspective*. New York: John Wiley and Sons, Inc.

Commoner, B.

1964 Roles of Deoxyribonucleic Acid in Inheritance. *Nature* 202: 960-968.

Enwonwu, C.O.

1973 Influence of Socioeconomic Conditions on Dental Development in Nigerian Children. *Archives of Oral Biology* 18: 95-107.

Epifanova, O.I.

1971 Effects of Hormones on Cell Cycle. In the Cell Cycle and Cancer. R. Baserga ed., pp. 144-190, New York: Marcel Dekker.

Garn, S.M., A.B. Lewis and J.H. Vicinus

1963a Third Molar Polymorphism and Its Significance in Dental Genetics. *Journal of Dental Research, Supplement* 42: 1344-1363.

Garn, S.M., A.B. Lewis and R.S. Kerewsky

1963b Third Molar Agenesis and Size Reduction of the Remaining Teeth. *Nature* 200: 488-489.

1964a Sex Differences in Tooth Size. *Journal of Dental Research* 43: 306.

1965 Genetic, Nutritional and Maturational Correlates of Dental Development. *Journal of Dental Research* 44: 228-242.

1966a The Meaning of Bilateral Asymmetry in the Permanent Dentition. *Angle Orthodontist* 36: 55-62.

1967a Buccolingual Size Asymmetry and Its Developmental Meaning. *Angle Orthodontist* 37: 186-193.

1967b Sex Differences in Tooth Shape. *Journal of Dental Research* 46: 1470.

Garn, S.M., R.S. Kerewsky and D.R. Swindler

1966b Canine "Field" in Sexual Dimorphism of Tooth Size. *Nature* 212: 1501-1502.

Goose, D.H.

1963 Dental Measurement: An Assessment of Its Value in Anthropological Studies. In Dental Anthropology. D. Brothwell ed., pp. 125-148. London: Pergamon Press.

1967 Preliminary Study of Tooth Size in Families. Journal of Dental Research 46: 959-962.

Goose, D.H. and G.T. Lee

1973 Inheritance of Tooth Size in Immigrant Populations. Journal of Dental Research 52: 175.

Greene, R.

1970 Human Hormones. New York: McGraw-Hill.

Hamerton, J.L.

1968 Significance of Sex Chromosome Derived Heterochromatin in Mammals. Nature 219: 910-914.

Holt, S.B.

1954 Genetics of the Dermal Ridges: Bilateral Asymmetry in Finger Ridge Counts. Annals of Eugenics 18: 211-231.

1968 The Genetics of Dermal Ridges. Springfield, Illinois: Charles C. Thomas.

Jantz, R. L.

1975 Population Variation in Asymmetry and Diversity from Finger to Finger for Digital Ridge Counts. American Journal of Physical Anthropology 42: 215-224.

1977 On the Levels of Dermatoglyphic Variation. Presented at the Cummins Memorial Symposium, Gulf Shores, Alabama.

Jantz, R. L. and R. S. Webb

N. D. Racial and Sexual Differences in the Fluctuating Asymmetry of the a-b Ridge Count. Manuscript.

Krogman, W.M.

1972 Child Growth. Ann Arbor: University of Michigan Press.

Lyon, M. F.

1962 Sex Chromatin and Gene Action in the Mammalian X-Chromosome. American Journal of Human Genetics 14: 135-148.

Mittwoch, U.

1973 Genetics of Sex Differentiation. New York: Academic Press.

Moorrees, C.F., E.A. Fanning and E.E. Hunt, Jr.

1963 Age Variation of Formation Stages for Ten Permanent Teeth.
Journal of Dental Research 42: 1490-1502.

Moss, M.L. and L. Moss-Salentijn

1977 Analysis of Developmental Processes Possibly Related to
Human Dental Sexual Dimorphism in Permanent and Deciduous
Canines. American Journal of Physical Anthropology 46: 407-413

Nie, N.H., C.H. Hull, J. Jenkins, K. Steinbrenner and D.H. Bent

1975 Statistical Package for the Social Sciences. New York:
McGraw-Hill.

Parsons, P.A.

1964 Finger-print Pattern Variability. Acta Genetica Et Statistica
Medicae 14: 201-211.

Penrose, L.S.

1969 Dermatoglyphics. Scientific American 221: 72-83.

Perzigian, A.J.

1977 Fluctuating Dental Asymmetry: Variation Among Skeletal
Populations. American Journal of Physical Anthropology 47:
81-88.

Potter, R.H. and W.E. Nance

1976 A Twin Study of Dental Dimension. I. Discordance, Asymmetry
and Mirror Imagery. American Journal of Physical Anthropology
44: 391-396.

Potter, R.H., P.L. Yu, A.A. Dahlberg, A.D. Merritt and

P.M. Conneally

1968 Genetic Studies of Tooth Size Factors in Pima Indian
Families. American Journal of Human Genetics 20: 89-100.

Rosenzweig, K.A.

1970 Tooth Form as a Distinguishing Trait between Sexes and
Human Populations. Journal of Dental Research 49: 1423-1426.

Shiono, H., J. Kadowaki and H. Tanda

1975 The Palmar a-b Ridge Count and Sex Chromosomes. Human
Biology 47: 505-509.

Siegel, M.I., W.J. Doyle and C. Kelley

- 1976 Heat Stress, Fluctuating Asymmetry and Prenatal Selection in the Laboratory Rat. *American Journal of Physical Anthropology* 46: 121-126.

Siegel, M.I. and W.J. Doyle

- 1975a The Differential Effects of Prenatal and Postnatal Audiogenic Stress on Fluctuating Asymmetry. *Journal of Experimental Zoology* 191: 211-214.

- 1975b The Effects of Cold Stress on Fluctuating Asymmetry in the Dentition of the House Mouse. *Journal of Experimental Zoology* 193: 385-389.

Siegel, M.I. and H.H. Smookler

- 1973 Fluctuating Dental Asymmetry and Audiogenic Stress. *Growth* 37: 35-39.

Singh, S.

- 1968 A Measure of Asymmetry of Finger Ridge Counts. *Acta Genetica Et Statistica Medicae* 18: 599-605.

- 1970 Inheritance of Asymmetry in Finger Ridge Counts. *Human Heredity* 20: 403-408.

Vander, A.J., J.H. Sherman and D.S. Luciano

- 1975 *Human Physiology*. New York: McGraw-Hill.

Van Valen, L.

- 1962 A Study of Fluctuating Asymmetry. *Evolution* 16: 125-142.

Waddington, C.H.

- 1957 *The Strategy of Genes*. New York: MacMillan.

- 1960 Experiments on Canalizing Selection. *Genetical Research* 1: 140-150.

VITA

Robert Stephen Webb was born in Jacksonville, Florida on October 16, 1953. He attended elementary school in that city and graduated from St. John's Country Day School in nearby Orange Park, Florida in 1971. He entered Shorter College in Rome, Georgia the following fall. In the Fall of 1973 he attended the University of New Mexico for a semester. During the first nine months of 1974 he worked as an archaeological crew chief for the University of Georgia. In Fall, 1974, he was accepted at the University of Tennessee, Knoxville, and in December of 1975, he received his B.A. in anthropology.

Robert entered graduate school in the Winter Quarter of 1976, at the University of Tennessee. For the first seven months of that year he was involved in archaeological investigations in Jalisco, Mexico, where he served as a physical anthropologist and archaeological crew chief. More recently, he has been collecting biological data for the Anthropology Department at the University of Tennessee, Knoxville.

Robert is a member of the American Association of Physical Anthropologists, the American Dermatoglyphics Association and the American Anthropological Association. He is employed at Fels Research Institute in Yellow Springs, Ohio.

Robert is married to Elaine Williams of Maryville, Tennessee.