To the Graduate Council:

We are submitting herewith a dissertation written by Stephen David Ousley entitled "The Quantitative Genetics of Epidermal Ridges Using Multivariate Maximum Likelihood Estimation". We have examined the final copy of this dissertation for form and content and recommend that it be accepted in partial fulfillment of the requirements for the degree of Doctor of Philosophy, with a major in Anthropology.

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The Quantitative Genetics of Epidermal Ridges Using Multivariate Maximum Likelihood Estimation

A Dissertation
Presented for the
Doctor of Philosophy
Degree

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Stephen David Ousley

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For

Heinz Brehme
1927 - 1993

Meta Neumann
1896 - 1996

Daniel Edward Ortmeyer
1947 - 1995

Leonard Paris Ousley
1900 - 1995

Wie an dem Tag, der dich die Welt verliehen,
Die Sonne stand zum Gruße der Planeten,
Bist alsobald und fort und fort gediehen
Nach dem Gesetz, wonach du angetreten.

So mußt du sein, dir kannst du nicht entfliehen,
So sagten schon Sibyllen, so Propheten;
Und keine Zeit und keine Macht zerstückelt
Geprägte Form, die lebend sich entwickelt.

Goethe (*Urworte* (extract), 1821)

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appreciated. Unfortunately, he did not live to see these results. Thanks to his work, many more results are sure to come.

ABSTRACT

Finger prints have been used for identification purposes for many hundreds of years. They were first used as a secure signature for financial agreements by merchants and rulers, later employed for forensic identification by law enforcement officials, and more recently adopted by anthropologists for estimating population relationships. Anthropologists believed that the dermatoglyphic observations accurately reflected the genetic relationships among observed populations due to their early development *in utero*, high heritability, and selective neutrality. A highly heritable and neutral trait would reflect the genetic relationships between populations more accurately. These qualities have prompted several anthropologists to suggest that dermatoglyphic traits reflect population relationships more accurately than anthropometrics or single-locus markers. While the quantitative genetics of anthropometrics and implications for estimated population relationships have been ascertained recently, these have not been investigated adequately for dermatoglyphics.

Dermatoglyphic traits appear to have a significant genetic component, though many aspects are uncertain. Holt firmly established the idea that dermatoglyphic traits, especially TRC (Total Ridge Count, the sum of the greater of the radial and ulnar ridge counts of each finger), were highly heritable with little environmental or nonadditive variation. Holt interpreted population differences in dermatoglyphics as evidence for natural selection, but many others assumed selective neutrality. More recent results

suggest that Holt's models were inadequate and that the genetic qualities of finger ridges include major gene, dominance, epistasis, and maternal effects. Like Holt, later researchers frequently analyzed composite variables, such as TRC (Total Ridge Count). TRC was used because of its more normal distribution and computational ease.

The inheritance of TRC alone does not explain the genetic properties of dermatoglyphics adequately, however. As Weninger and others pointed out, TRC simplified what could be vastly different finger configurations. At the level of the individual fingers, results from factor analysis of phenotypic values strongly suggested a "field effect" during the embryonic formation of epidermal ridges. Additionally, there has been little research concerned with the genetics of toe ridge counts, so a more comprehensive view of epidermal ridge formation in humans is still lacking.

Previous quantitative genetic analyses of dermatoglyphics have been limited to relatively small data sets and pedigrees that were divided into parent-child regressions and sibling-sibling correlations. To date, the largest dermatoglyphic sample with pedigree data was collected from over 800 German and Austrian families by Heinz Brehme and was analyzed here for the first time. The data comprise samples from normal families, families with dizygotic twins, and families with monozygotic twins. All of these family configurations were analyzed using the method of maximum likelihood estimation.

The results of this study strongly confirm the additive genetic basis of dermatoglyphic variability as well as the role of dominance and a "field effect" in the development of epidermal ridges in the fingers and toes. The contribution of additive genes to finger ridge counts was found to be much lower than previously estimated. This result was due to larger samples, better methodologies, and the avoidance of summary variables such as TRC, which were shown empirically to inflate the additive component. Additionally, finger ridge counts, palm ridge counts, and toe ridge counts are largely under separate genetic control.

The components of dermatoglyphic variation are best understood developmentally. Ridge count heritabilities are lower due to significant dominance and environmental effects, but also to a high level of developmental integration, as shown by high genetic correlations among variables. Epidermal Growth Factor, with its widespread action on the developing fetus and the epidermis, may be an especially important regulatory hormone for developmental integration.

Differences between the additive genetic and phenotypic variance-covariance matrices produced disparities in population relationships using phenotypic and additive genetic values derived from the data with pedigrees. Dermatoglyphic phenotypes do not accurately reflect the genetic relationships among these populations. When analyzing population structure, dermatoglyphic traits cannot be used as a surrogate for genetic data, and will produce distorted relationships among populations.

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Chapter 1
INTRODUCTION

Human finger prints are a subset of dermatoglyphics, the ridge patterns that are formed on the volar surface of the hand and on the plantar surface of the foot. They are the result of the processes of dermal and epidermal development *in utero* and remain virtually unchanged throughout life. Dermatoglyphics are of interest to anthropologists because of their early development, permanence, and variation among human populations. Anthropologists compare dermatoglyphic relationships among populations to linguistic, geographic, and ethnohistorical relationships. Additionally, most researchers have presumed that dermatoglyphic traits are highly heritable and selectively neutral (Jantz 1987), and will thus reflect only genetic drift and migration among populations. For these reasons, anthropologists have assumed that the phenotypic dermatoglyphic relationships among populations accurately reflect their phylogenetic relationships. A test of this assumption is only possible through examining the genetics of dermatoglyphic traits.

Despite decades of research, the quantitative genetics of dermatoglyphic traits are not well established. Holt's (1952, 1956, 1957, 1968) investigations have indicated a near-total degree of genetic determination for the composite variable TRC (Total Ridge Count, the sum of the greater ridge counts from the radial and ulnar side of each finger pattern), though the validity of her results and the applicability to individual dermatoglyphic traits are disputed (Roberts and Coope 1975; Weninger 1964; Weninger *et al.* 1976; Weninger

1983). Population comparisons are much more reliable and valuable if they utilize many variables rather than one variable such as TRC, because they represent more of the variation among populations. Dermatoglyphic traits are certainly genetically influenced, as shown by dermatoglyphic peculiarities associated with genetic syndromes and medical conditions including Down's Syndrome, cleft palate, mental illnesses, and various chromosomal abnormalities (Jantz and Hunt 1986; Penrose 1963; Schaumann and Alter 1976). However, dermatoglyphic configurations can be influenced by environmental factors such as testosterone level (Sorenson Jamison *et al.* 1993, 1994) and diseases such as rubella and leukemia during pregnancy (Schaumann and Alter 1976).

Many of the genetic features of dermatoglyphic traits remain uncertain because most researchers have utilized data indirectly, through analyzing summary variables such as TRC, assessing population differences, or associating certain dermatoglyphic peculiarities with genetic disorders. If the data have included pedigrees (family relationship information), researchers have used univariate comparisons or composite variables, nullifying the genetic relationships among variables. Univariate analyses involved correlation and regression, which relied on paired data sets, most often from twins. The relationships among variables are important in a multivariate analysis and can also provide insights to their developmental commonalities (Atchley 1984; Cheverud 1984). Maximum likelihood (ML) estimation makes use of all variables and all pedigree relationships in multivariate estimates of genetic parameters. There have been very few multivariate studies using primary variables and pedigrees due to the computational rigor and large

sample required. As a result, the quantitative genetics of individual fingers and the genetic relationships between fingers have not been sufficiently explored. This multivariate quantitative genetic investigation will establish a more accurate picture of how dermatoglyphic features are related to each other genetically, how relatives are related to each other dermatoglyphically, and by extension, how populations are related to each other genetically through their dermatoglyphics.

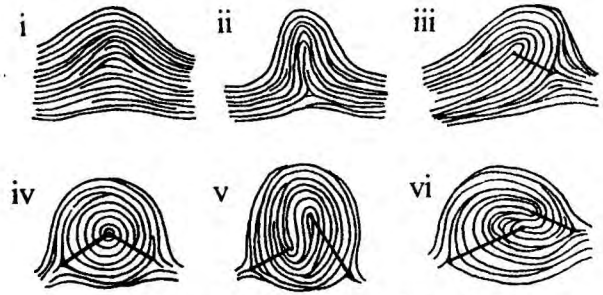
Dermatoglyphics and Population Comparisons

Dermatoglyphic data, especially in the form of finger ridge counts, have many desirable qualities for use in comparing human populations. Epidermal ridges are formed early in development, and thereafter retain their configurations throughout life barring significant trauma to the skin. By the 25th fetal week, after the ridges have completely formed, further environmental influences have no effect on ridge configurations (Babler 1991; Okajima 1991). The early formation and persistence of dermatoglyphic features are the basis for the widespread assumption that "comparisons between populations can be less concerned with environmental effects than those dealing with anthropometric variables." (Meier 1991: 253). Because ridge counts do not change over time and do not change with an individual's body size, dermatoglyphic data from children and adults among populations can be utilized more easily. Larger sample sizes produce more reliable population comparisons, and more of the quantitative variation in populations can be represented. Also, there are no indications of assortative mating in regard to ridge counts, allowing simpler statistics. Collecting dermatoglyphic data from individuals is less invasive

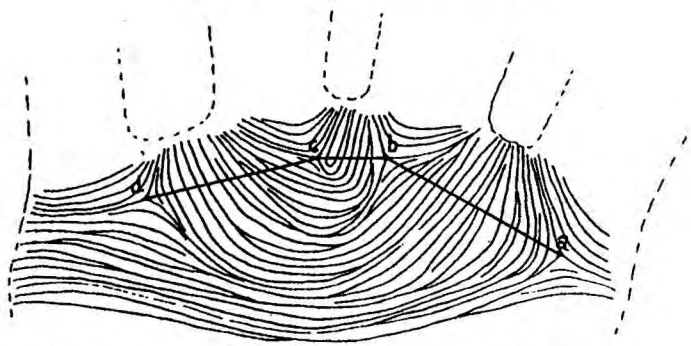
than drawing blood or taking anthropometric measurements, and the permanence of dermatoglyphic records enables later analyses as new techniques are developed (Meier 1991).

Ridge counts are meristic traits, which can have only integer values, like abdominal bristle number in *Drosophila*. However, the range and distribution of ridge counts make them quasi-continuous, allowing less complicated statistical methods than those for discrete data. Finger print configurations are represented by three basic patterns, arch, loop, and whorl (Figure 1a). The most common method of quantifying finger print configurations is to count the number of ridges between the triradius (the intersection of three ridges) and the pattern core in fingers, and between the triradii in palms (Figure 1b). Arches, with no triradius, have a count of zero. Loops, with one triradius, have either an ulnar or radial count depending on the orientation of the pattern (Figure 1c). Whorls, with two triradii, have ulnar and radial counts. The three palm counts are designated by the two triradii that they include (ab, bc, and cd). Toe patterns have tibial and fibular ridge counts depending on their orientation.

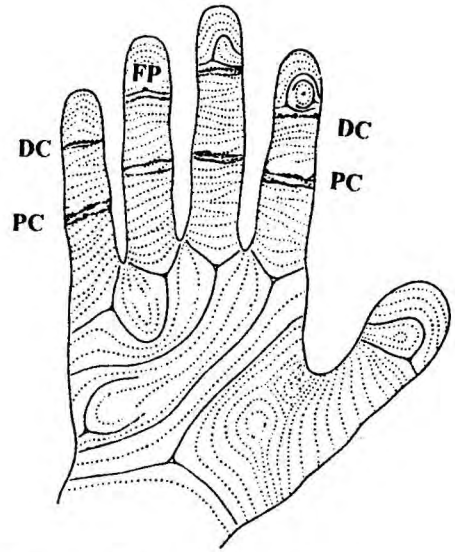
Froehlich and Giles (1981a, 1981b) outlined several advantages of dermatoglyphic traits in comparison to other biological traits. They concluded that dermatoglyphics reflect ancient population relationships better than anthropometrics and blood markers because dermatoglyphics are highly heritable, less subject to selection, and resistant to gene drift. They found that in spite of recent gene flow, finger ridge counts reflected linguistic



a. Finger Patterns and Counting.



b. Palm (Interdigital) Ridge Counting (Right Hand).



c. Finger ridges, creases, and patterns.

Figure 1. Finger and Palm Ridge Counting. The finger patterns shown (a) are plain arch (i), tented arch (ii), loop (iii), monocentric whorl (iv), and two bicentric whorls (v,vi). The heavy lines shown are drawn between triradii and pattern core for fingers, and between triradii (ab, bc, cd) in palms (b). The number of ridges that they cross is the ridge count.
Figure 1c shows the location of a finger pad (FP) and the proximal and distal interphalangeal creases (PC, DC). Also shown are an arch (little finger), radial loop (middle finger), ulnar loop (thumb), and whorl (index finger).
(Figures 1a and 1b adapted from Figure 4 in Hauser, 1990: Dermatoglyphic recording and scoring techniques. In *Trends in Dermatoglyphic Research*, N.M. Durham and C.C. Plato, eds. Boston: Kluver, pgs. 16-52. Figure 1c adapted from Figure 11 in Cummins, 1926: Epidermal-ridge configurations in developmental defects, with particular reference to the ontogenetic factors which condition ridge direction. *American Journal of Anatomy* 38: 89-151).

distances among several New Guinea populations better than anthropometric variables, and cited results from other studies that supported their view. Highly heritable neutral traits would show little environmental influences, and Holt's (1952, 1956, 1957, 1968) results indicated a very high heritability for finger prints. The polygenic nature of dermatoglyphics was believed to be the reason for a more conservative rate of change over time, because random changes across many loci would cancel each other out (Birdsell 1950; Froehlich and Giles 1981b).

Dow *et al.* (1987) also compared population relationships based on several types of biological data from the Solomon Islands, and concluded that genetic drift largely explained the observed patterns of variation. Using more powerful matrix comparison tests than Froehlich and Giles (1981a, 1981b), they found significant agreement between dermatoglyphic (finger and palm ridge counts) and linguistic distances, controlling for geography. Blood marker, anthropometric, and odontometric matrices showed insignificant and/or lower correlations with language, which the authors attributed to stronger selection on these traits (Dow *et al.* 1987).

Variables subject to natural selection would reflect the effects of selection rather than genetic drift on population differentiation, and would produce distorted population phylogenies. A possible functional role for dermatoglyphics that would be subject to selection comes from Loesch and Martin's (1984) findings that finger patterns are correlated with tactile sensitivity. Fingers with loops and ridge counts near the mean were

more sensitive than fingers with other patterns and ridge counts. There is conflicting evidence for selection in the relationship between dermatoglyphics and fertility. Loesch and Wolanski (1985) found that women with a history of miscarriages had significantly lower ridge counts and fewer loops and whorls than women with successful births. Also, women with more loops and whorls, especially for some fingers, had more surviving offspring. However, Blangero (1988) found no evidence for fertility selection in samples from Nepalese villages, and concluded that dermatoglyphics are selectively neutral.

Rogers and Harpending (1983) compared blood marker, dermatoglyphic, and anthropometric differentiation among Bougainville island populations using F_{ST} (Wright 1951), a measure of within-group variation relative to among-group variation. They found that the level of among-group phenotypic differentiation for finger ridge counts was at the same level as ostensibly neutral blood markers. In contrast, anthropometrics showed much higher levels of differentiation, suggesting significant selective pressures. However, the level of genetic differentiation (calculated from phenotypic differentiation) depends on heritability, and Rogers and Harpending (1983) assumed a heritability of 1 for anthropometric and dermatoglyphic traits. They concluded that there was no evidence for selection in dermatoglyphics and that phenotypic distances equaled genetic distances for dermatoglyphic traits.

It is true that as a trait approaches a heritability of 1, the phenotypic relationships among populations will better represent the genetic relationships, because less room

remains for nongenetic variation. However, Cheverud (1988) noted that high heritabilities are not a necessary condition for phenotypic distances to accurately represent genetic distances. Rather, the phenotypic variance-covariance matrix of a trait set must merely be proportional to the genetic variance-covariance matrix. He then explicitly tested matrix proportionality using published analyses of morphometric data with pedigrees and found that phenotypic relationships accurately reflected the known genetic relationships. Most traits had an average heritability of only .35, demonstrating that high heritabilities are not a requirement for accurate population comparisons using phenotypic traits. However, assumptions of proportionality were not necessarily valid for population-level comparisons. Fitch and Atchley (1987) found disagreement between known mouse population phylogenies and population relationships estimated from mandibular measurements.

There have been few explicit examinations of the phenotypic and genetic relationships among human populations. In the studies of Froehlich and Giles (1981a, 1981b) and Dow *et al.* (1987), the authors did not perform an explicit test of their conclusions. In these studies, the "ancient" population relationships were based on linguistic distances rather than actual phylogenetic information. In an explicit test of proportionality, Konigsberg and Ousley (1995) analyzed anthropometric data from pedigrees and found that anthropometric relationships accurately reflected the genetic relationships among relatives. At the population level, Relethford and Crawford's (1995)

analysis of 20th century Irish anthropometrics revealed the residual biological influences of invasions and occupation of several areas by Vikings over 1000 years earlier.

To date, explicit tests on the genetics of dermatoglyphics have not been performed. The primary concern addressed so far has been that of selective neutrality. However, the extent and diversity of dominance and environmental effects among traits will also influence the phenotypic relationships among populations (Falconer 1989). The discrepancies between dermatoglyphic and anthropometric results and their implications for population comparisons can only be resolved through a quantitative genetic study of dermatoglyphic traits.

Estimating "Natural Inheritance"

Galton (1889) initiated the scientific study of the inheritance of qualitative variables, such as finger print patterns, and quantitative variables, such as stature, in the late 19th century. At that time there were two main schools of thought on the transmission of morphological information from one generation to the next: Particulate inheritance and blending inheritance. Particulate inheritance was the theory that animals obtained their features as discrete bits of information from their various ancestors. This explained how a child could have his mother's eyes and his father's nose, and also explained the perceived stability of racial "types". Blending inheritance maintained that a person was an average of his ancestors, especially his parents. A tall father and a short mother would produce intermediate offspring. However, under blending inheritance, variation would diminish

over time and variability, necessary for evolutionary change, would disappear. It was not until the rediscovery of Mendel's work in 1900 that evidence for particulate inheritance was widely published.

Galton first concentrated on quantitative variables and blending inheritance. In a study of stature, he also coined the term "regression" from the observation that in plotting midparental and sons' statures, "the Stature of the adult offspring must on the whole, be more mediocre [closer to the mean] than the stature of their parents" (Galton 1889:95). The stature of sons "regressed" to the population mean. For Galton, the fact of regression contradicted the total genetic determination of traits. Instead, he maintained that these traits would be influenced by "Family Likeness" (genetics) as well as "Individual Variation" (environment) (1889:9). He also maintained that if the traits were determined by the parents, then siblings or family units would show lower variability (within-pedigree variation) than random samples of children (among-pedigree variation). This approach is the basis for many genetic analytical methods including the traditional analysis of variance (ANOVA), and the intraclass correlation (Hopper 1993).

Galton tested blending vs. particulate inheritance by examining families with parents similar in a trait compared to families with parents differing in a trait. Particulate inheritance would imply that the children of similar parents would be more similar to each other than children of parents that were very different. The children of very different parents would be expected to be more similar to one or the other parent. His initial results

indicated that stature appeared to be a blending trait. Galton invented the "midparent" value, an average of the two parental values. He found that unrelated children with the same midparental stature showed greater differences than siblings with the same midparental stature. Blending alone could not explain these discrepancies, which he attributed to different individual parental values, to different values for their ancestors, and to different environments.

Galton also examined the distribution and inheritance of finger print patterns (Galton 1892). Fingers are more easily referred to by number, and are numbered sequentially starting with the thumb, digit 1, to the little finger, digit 5. He noted different pattern frequencies for the fingers and differences in symmetry and similarity: fingers 2-3-4 (index, middle, ring) showed few bilateral differences in pattern type and greater differences among fingers. In contrast, fingers 1 (thumb) and 5 (little finger) showed more differences in bilateral asymmetry yet fewer differences between each finger on the same hand. He also detected a "proximity effect" in fingers, with the greatest frequency of arches occurring on digit 2, and arch frequencies for other fingers diminishing with greater distance. Galton explored the inheritance of dermatoglyphics by comparing the frequencies of arches, loops, and whorls between pairs of brothers and compared them to frequencies from random pairings and found that the brother pairs were more similar than the random pairs.

Fisher (1918) introduced a new method and theory for analyzing quantitative traits in a Mendelian framework. At that time, the prevailing theories of inheritance were mutually exclusive: Traits were either blended or discrete. After the rediscovery of Mendel's work at the turn of the century, most genetic research was directed to qualitative traits, for which there were now well-founded methods (such as counting discrete phenotypes), theories, and results. Fisher's model was a fusion of the particulate and blending inheritance models, explaining the blending effect due to large numbers of genes and estimating the particulate component through dominance (Hopper 1993). Continuous variation was the summed small effects of many "particles", while the particulate variation was due to few "particles" with larger effects. Among relatives, individual traits should be approximately normally distributed, and the relationships between several traits determined by many genes could be analyzed in terms of variances and covariances. In short, Fisher's theory allowed phenotypic variation in traits to be analyzed in terms of specific underlying genetic influences (Hopper 1993).

Fisher's (1918) breakthrough involved examining the variance among relatives and decomposing it into variance due to additive genetic, dominance, and environmental effects. For example, the expected covariance between a parent and offspring should equal half the additive genetic variance because they share exactly half of their alleles. The covariance between sibs should equal 1/2 the additive genetic variance, because they share on average half of their alleles, plus 1/4 of the dominance variance, because 50% of the alleles they have in common will be allele pairs at the same locus. Parameter estimations

can be based on each available relationship category (e.g. parent-child, sib-sib), so many possible equations can be solved. Fortunately, Fisher (1912) also invented the method of maximum likelihood estimation for solving simultaneous linear equations such as these (Crow 1986). With a mathematical model available, it was now possible to compare continuous data from various combinations of family members to the proportion of genetic materials they had in common. However, because maximum likelihood estimation was extremely time-consuming, measurements were most often compared using other statistical methods such as correlation and regression, limiting comparisons to one variable and requiring paired data sets, often from twins (Falconer 1989; Hopper 1993). Correlation and regression have dominated the quantitative genetic analysis of dermatoglyphics to the present day.

Chapter 2
INVESTIGATING THE GENETICS OF EPIDERMAL RIDGES

Ridge counting has become the primary technique of the genetic analysis of dermatoglyphics because it expresses more of the variation in finger patterns. Galton's results from analyzing the genetics of finger patterns were contradictory and questionable because he divided patterns into radial loops, ulnar loops, and whorls. These categories did not account for enough of the variation among patterns. There was a loss of information using three categories because, for example, all radial loops were not alike. Loops formed the vast majority of patterns, and Galton's division of loops into radial and ulnar did not differentiate them sufficiently. Henry (1900) precipitated the future use of fingerprints to aid in apprehending criminals and to identify their victims by specifying a method of counting loop ridges to distinguish differences between finger prints from different persons. These subdivisions were necessary for any data base of fingerprints, which would need to be sorted by measurement values, as Bertillon's anthropometric records were sorted, to allow an expedient matching of the values from evidence and the paper record by hand before electronic computers were available (Cummins 1965). No counts were recorded for whorls, which were classified into three groups based on the configuration of the ridges that coursed between the two triradii (Henry 1900).

Bonnevie (1924, 1929) extended and refined Henry's methods to enable better quantitative genetic comparisons for ridge counts among relatives. She added a ridge

counting technique for whorls, and her system forms the bulk of ridge counting methodology today. She also defined variables based on the measured shape of patterns, their tendency to twist, and calculated the "quantitative value", which evolved into the mean of the ridge counts across all 10 fingers. In her analysis of ridge counts from a small sample of twins, she concluded that differences between identical twin fingers were at the same level of asymmetry within an individual.

Holt and Heritability

Holt has made the greatest impact on the study of the genetics of finger prints. She was the first to analyze dermatoglyphic data using parent-child and various sibling-sibling correlations and regression. With the help and advice of Penrose, she "invented" and primarily analyzed one composite measurement, the Total Ridge Count (TRC), which has dominated dermatoglyphic genetic research to the present day (Holt 1949, 1951; Roberts 1979). TRC is the sum of the greater of the radial and ulnar counts for each finger, summed across all fingers. Holt was apparently unaware that by 1929, Bonnevie had already revised a new "individual quantitative value". TRC is simply Bonnevie's (1929) revised value multiplied by 10. In her studies, Holt used larger and larger data sets from siblings, parents and their children, and dizygotic and monozygotic twins. Holt's results convinced her and many others of nearly total genetic determination for epidermal ridges. These convincing results, however, are due to the excluding estimates of the repeatability of TRC, a methodological flaw with enormous consequences. However, Holt's work reveals remarkable consistency over a span of almost 30 years.

In 1952, Holt presented the results of an initial investigation into the genetics of TRC using 83 sets of full siblings, 50 parent-child pedigrees, and 100 pairs of twins. She used the intraclass correlation in her analysis, which compensates for different pedigree sizes. Her results indicated almost total genetic determination of TRC, with correlations for parent-child, siblings, and dizygotic twins close to .50. This would leave little or no variation for dominance. However, she did note that the correlations for the individual fingers were lower, as low as .31, and varied across fingers. However, the differences in correlations were not statistically significant. She, like Galton, noted a "proximity effect", with fingers that are close to each other showing higher correlations to each other. She believed the higher heritability of TRC compared to individual fingers was due to an overall "size effect"(positive and relatively high intercorrelations among all fingers). High counts on one finger would tend to be accompanied by high counts on other fingers. Midparent-child correlations were very close to 0.71, the theoretical correlation of a completely heritable trait, and the regression had no indication of nonlinearity, making dominance unlikely. Most convincingly, the monozygotic (MZ) twin correlation was 0.95, indicating a high heritability, though for MZ twins, dominance and additive variance are confounded (Falconer 1989). Because MZ twins share all of their allele pairs, the dominance effects of allele pairs cannot be separated from the additive effects of alleles. Mother-daughter regression showed a higher correlation, suggesting possible maternal effects, but none of the variously subdivided heritability estimates were significantly different from each other.

Holt's preliminary results indicating a high heritability for TRC were repeatedly confirmed by her. Holt (1956) calculated parent-child TRC correlations based on 149 families. She recognized that the intraclass correlation could not be used when employing parent-child data, and that the data must be balanced in pairs. As a result, she paired parental values for each child, so the same parent was used multiple times, but she did caution that it would affect the standard errors. She concluded, however, that the parent-child and midparent-child correlations confirmed her earlier results indicating nearly total genetic determination. In a 1957 analysis of sib-sib correlations using 523 siblings from 210 sibships, Holt's results once again indicated a near-complete heritability. All of Holt's estimates except those for MZ twins involve large standard errors, with the lower bound of the regression coefficient near .40, meaning a lower bound for heritability of about .80. In an examination of the distribution of TRC in a large sample, Holt (1955), like many others, found it to be negatively skewed, the distribution showing a long left tail. She interpreted the skewness to mean that a few genes with major effects determine TRC, because a larger number of genes would produce a more normal distribution than a small number of genes, and she had found no indications of dominance in any of her work. However, a significant factor in the negatively skewed distribution of TRC may be the negatively skewed Pattern Intensity Index, (PII, the total number of triradii across all fingers) distribution, which influences ridge counts, as pointed out by Froehlich (1970, citing Luu-Mau-Thanh 1965).

Holt (1954) examined the heritability of dermatoglyphic asymmetry as well. In an analysis of bilateral asymmetry among over 100 pairs of MZ and DZ twins, Holt compared the summed finger ridge counts from each hand (Holt 1954). She found that right hand counts were greater than left counts, but the heritability of each hand was close to 1. Her results also indicated that the absolute asymmetry (right minus left hand count) was not heritable, with no significant correlations, though she acknowledged that Grüneberg's (1928) results contradicted hers. Grüneberg (1928) had compared MZ and dizygotic (DZ) twins and concluded that asymmetry was heritable, either as a trait itself or as a result of separate right-left genetic factors. MZ twins showed greater within-individual left-right asymmetries in pattern type, orientation, and total pattern configuration than differences between the homologous fingers (the same finger on each hand) of each MZ pair (Grüneberg 1928). However, Holt (1954) did not examine ridge counts at the level of the individual finger, preferring summed variables from the left and right hands, so her results are not directly comparable to those of Grüneberg (1928).

Holt (1959) further investigated the proximity effect that Galton had observed and that she had noted in 1952. She found the highest phenotypic correlations between greater ridge counts from homologous fingers, and the next highest correlations among adjacent fingers. Correlations among nonhomologous fingers were highest for digits 3-4-5. The thumb had the lowest and least variable correlations, showing little or no proximity effects. In 1960, Holt examined the heritability of the Finger Diversity Index (the standard deviation of all 10 fingers, FDI). She found that the FDI was less heritable than TRC, with

correlations near 0.25 for parent-child, full siblings, and DZ twins, but 0.75 for MZ twins, suggesting intrauterine environmental effects and/or various nonadditive genetic effects (dominance, epistasis, genetic-environmental interactions). Jantz (1974, 1976) has pointed out that Holt's diversity index is actually a combination of asymmetry and diversity, and concluded that it is a somewhat muddled "trait".

By the time Holt wrote *The Genetics of Dermal Ridges* in 1968, a clear picture had emerged: Epidermal ridges, at least TRC, were determined almost entirely by additive genes, with little or no room for dominance or environmental effects. All heritabilities from her studies were close to 1, and the data from MZ twins allowed only 5 percent for environmental effects. Individual fingers, especially digit 1, showed lower heritabilities, but the high heritability of TRC was due to an overall "size effect", with small and random finger-level fluctuations. Finger diversity was less heritable than TRC, and asymmetry was not heritable. Holt went so far as to state "In man the best example of agreement between observed and theoretical correlations for a metrical character is provided by the *total finger ridge-count*." (Holt 1968:39). She is not alone in this conclusion:

> "To illustrate the application of this theory, we shall discuss the inheritance of the fingerprint ridge count in man, which provides a good example of what happens in the absence of any complications... It can be concluded that 95 per cent of the variability is of genetic and 5 per cent of environmental origin, and that there is completely additive gene action with no evidence of either dominance or epistasis."
> *The Mathematical Theory of Quantitative Genetics* (Bulmer 1980:75-76)

> "The heritability of several such [dermatoglyphic] traits is also nearly complete (Holt 1961), making them candidates for mathematical modeling."
> Chakraborty (1990: 155)

"Total finger ridge count is a highly heritable trait for which an abundance of pedigree data exists... Thus, these data provide no evidence for a dominance component... it is clear that the two ridge counts [total ridge count for each hand] are highly heritable traits. More surprising is the very high additive genetic correlation and very low environmental correlation between the traits. If these data are credible, then ridge counts on the left and right hands are basically determined by the same set of genes."
Documentation for FISHER, Version 2.1 (Lange *et al.* 1988)

"This trait [TRC] has the highest heritability of any studied in man, ...indicating that in the populations studied - caucasians primarily of northern European origin - genetic factors play an overwhelming role."
Miller (1981: 69)

"Of all the quantitative traits that have been studied adequately, fingerprint ridge counts provide one of the best examples of agreement between the expected and observed results"
Human Genetics (McKusick 1969:157)

"I've always regarded the genetic analysis of ridge counts to be far and away the best model we have in human genetics, of a polygenic or multifactorial trait that really obeys the predictions of the model."
Nance, in *Congenital Defects* (Janerich *et al.* 1974:52)

Holt saw the potential value of dermatoglyphic comparisons for human population comparisons. For her, highly heritable traits were more valuable because they would respond to selection and illuminate relationships between differentiated populations: "The observed racial variation shows that there must be natural selection" (Holt 1968:64). This was the contemporary causal assumption when population differences were discovered for any trait. Genetic drift was not considered. But comparisons among populations are of limited value using only one trait.

Confirmation and Dissension

Many studies have confirmed Holt's conclusions, and nearly as many have contradicted them. However, in not estimating repeatability, most researchers made the same mistake as Holt, though they used more and more sophisticated methodologies. Other researchers analyzed dermatoglyphic data indirectly, without pedigrees.

Loesch performed a great many analyses which largely confirm Holt's results. Loesch's (1983) estimated heritabilities for TRC are generally somewhat lower than Holt's, which she attributed to population differences. The heritability of finger ridges were still believed to be much higher than the heritability of palm ridges. Loesch (1983) also found that heritabilities for individual fingers were lower than for TRC, and concluded that the lower heritabilities for individual fingers were due to negative environmental covariances. Loesch's (1979) regression analysis of PII showed MZ twins had a heritability of .90, siblings and parent-child heritabilities were near .80, but DZ twin heritability of PII was near .60, suggesting nonadditive effects for PII. Heath *et al.* (1984) reanalyzed Loesch's PII data using the *z*-transformed correlations between combinations of relatives and comparing likelihood ratios for various models. They rejected the additive-only model and the model with additive and epistatic effects with no dominance produced the best fit to the data.

The results of Martin *et al.* (1982b), including a negative regression of DZ twins and sib-pair variances on pair means, and the negative skewness of individual finger ridge

count distributions, suggested non-additive genetic effects, in contrast to Holt's results. They used greater ridge counts from 221 DZ and MZ twin pairs and 80 pairs of opposite sex twins. Using between-pair mean squares, they calculated a narrow-sense heritability of TRC of .64 ± .28 (s.e.) and broad sense heritability of .95 ± .01, suggesting significant dominance. Univariate estimates for individual fingers were lower and varied somewhat. They also concluded that the lower heritabilities for individual fingers compared to TRC implied positive genetic covariances and negative environmental covariances between individual fingers. Later analyses of the same data using factor analytical techniques and quite elaborate models confirmed this pattern and their conclusions (Martin *et al.* 1982c).

Some authors found indications that TRC was merely part of an even larger-scale overall genetic ridge configuration of the hands and feet. Mathew *et al.* (1987) analyzed composite traits such as total ridge counts in fingers, toes, palms, soles, and various agglomerations of them from 125 Indian families, some with MZ and DZ twins. Path models fitted to familial correlations indicated near total heritabilities for each combination, as in other studies (Malhotra *et al.* 1982). These path models did not include dominance, however, and no goodness of fit statistics or likelihoods were presented.

Dominance effects in TRC have been suggested in examining admixed populations, which show mean dermatoglyphic values above the mean value of the parent populations. This pattern was present in mixed populations composed of Australian Aborigines and Europeans, (Robson and Parsons 1967) and Rehobothers (Khoisan and Europeans)

(Hitzeroth *et al.* 1986). Dominance effects varied across fingers in each hybrid population (Hitzeroth *et al.* 1986; Singh 1979). None of these studies utilized pedigree data, but relied on population means.

Another approach in estimating dominance effects has been to use SEDA (Structured Exploratory Data Analysis), made up of the Major Gene Index, Offspring Between Parents function, and Midparent Correlation functions (Karlin *et al.* 1983a). These statistics measure the relative distance of a child to the midparent and to each parent because major (dominant) genes would tend to give the child a value closer to one or the other parent. SEDA statistics do not require data to be normally distributed, an advantage in dermatoglyphic analyses. However, SEDA statistics provide no significance tests, and no probabilities can be ascertained, so their value is limited. Young *et al.* (1981) used SEDA and found that an analysis of finger patterns produced a large MGI, indicating a major gene effect. However, they found that the somewhat discrete distribution of their pattern classifications (12 classes) inflated MGI, so the significance of their results is uncertain. Karlin *et al.* (1983a) analyzed ridge count data from 125 families using SEDA, and found that parent-child results for TRC and for each hand showed near total additive variance, but individual fingers showed lower heritabilities and dominance effects.

Spence *et al.* (1973) analyzed TRC using ML estimations for a mixture of three normal distributions in samples of Japanese and American Whites. They actually analyzed $\log_{10}(\text{TRC} + 100)$ because logging the variables removed significant skewness from the

distributions. A mixture of three normal distributions fit the transformed data, and the authors concluded that a major gene with two alleles would explain over half the variation in TRC in both groups. They compared a proband (a selected group in a sample) consisting of relatives of people with arches on every finger to relatives of persons with loops or whorls and found that the proband differed from Hardy-Weinberg expectations and the control groups did not. Spence *et al.* (1973) also concluded that the alleles at the major locus for TRC show no dominance deviations. However, log-transforming variables can change parameter estimates dramatically (Hopper 1993; results not shown), and removes information as to the nature of the traits examined. Also, in using TRC, a composite of at least 10 separate traits, it is not surprising that Spence *et al.* (1973) would find a mixture of several distributions.

One of the most statistically sophisticated studies of TRC has been that of Huggins and Loesch (1994) in an analysis of small samples of normal and fragile X families. They used robust ML estimation on TRC and the "ridge count" of digit 3 (whether ulnar, radial, the greater of the two, or sum of the two from the left, right, or both is not clear) and analyzed residuals in terms of mixtures of distributions. The best models for each trait were those with additive and environmental effects because the models with dominance resulted in negative environmental variances. The residuals represented several normal distributions. Huggins and Loesch (1994) interpreted their results as confirming the large genetic additivity for TRC, and the presence of a major gene effect, or a small number of genes contributing to TRC. As in the study of Spence *et al.* (1973), it should not be

surprising that the residuals from fitting ML estimates to TRC, a composite of separate finger counts, can represent several distributions of individual finger counts. If anything, the study of Huggins and Loesch (1994) highlights the artificial constitution of TRC.

Weninger did not investigate the genetic properties of TRC as others did. Instead, she attacked the definition, justification, and use of TRC. As she repeatedly pointed out, TRC masks underlying differences in variances, means, and distributions of each finger and inconsistent correlations among fingers (Weninger 1964; Weninger *et al.* 1976; Weninger 1983). The variances of Holt's (1968) samples of ridge counts do show differences across fingers, with greater differences among non-homologous fingers. For Weninger, Holt's analysis of TRC alone did not reveal the factors influencing finger ridge counts, but merely reduced them to a "fact" of overwhelmingly additive genes. In sum, "TRC is, in spite of its continuous variation, a heterogeneous combination of values (fingers with different means, standard deviations and frequency distributions) and cannot pass for a biologically meaningful character." (Weninger 1983: 6-7).

In estimating the heritability of finger ridge counts, Rostron (1977), like Weninger, recognized that "Studies on the inheritance of dermal ridges have been, for the most part, univariate", and "In taking the total ridgecount as a measure of the phenotypic expression of the genotype one is implicitly assuming that the effect on each finger is the same" (Rostron 1977:199). Yet Holt's (1953) analysis, maximizing the intraclass correlation among pedigrees using summed homologous finger greater ridge counts, produced very

different coefficients for each finger, suggesting unequal effects among fingers. Rostron's solution was to perform a multivariate analysis of 10 finger ridge counts from pedigrees to estimate heritability. Using principal components analysis, only seven positive eigenvalues were extracted from the variance-covariance matrix, so the matrix was not positive-definite. He resorted to a principal components analysis limited to two axes. Rostron (1977) found a high heritability (.97) for the first principal component, which represented a general size effect, and a low heritability (.24) for the second principal component.

Instead of merely calculating heritability, some researchers used multivariate techniques on data from individual digits and established that the individual digits show differing relationships to each other. Knussman (1967, 1969), employed factor analysis on phenotypic correlations using published matrices and additional area and family samples. He found that finger ridge counts and pattern types clustered together digit 1, digits 2, 3, and 4, and digits 4 and 5. Toe factors included a big toe factor, the middle three toes, and a digit 5 factor. Additionally, when ridge counts from fingers and toes were analyzed concurrently, digit 1 and the big toe were the basis of a common factor. Roberts and Coope (1975) performed principal component analysis on radial and ulnar finger ridge counts. They recognized that TRC was of limited use for population comparisons, because groups could have the same TRC but different configurations on individual fingers. The first principal component was a general size component (as Rostron (1977) found), representing about 41% of the total variance. They also found an ulnar vs. radial component, a thumb component, an index finger component, and larger-scale lateral vs.

mesial components. They interpreted their results in terms of Butler's (1963) field theory, that is, that the location of elements determines final morphology. By analyzing all variables simultaneously, Roberts and Coope revealed dermatoglyphic properties that TRC cannot. Thus, the relative position of pattern areas on the hand and whether radial or ulnar influence morphology. "Each digit is a modification of its neighbours for features such as ridge count and pattern type." (Roberts and Coope 1975: 186). This echoes the proximity effect that Galton (1892) and Holt (1952, 1959) noted for finger dermatoglyphics, and is similar to the "neighborhood effect" among metacarpal measurements (Lewenz and Whitely 1903; Whiteley and Pearson 1900). Many subsequent studies confirmed finger ridge count factors similar to those found by Roberts and Coope (Jantz and Owsley 1977; Jantz *et al.* 1984; Reed *et al.* 1978; Siervogel *et al.* 1978). All of these authors stressed that the use of TRC masked underlying finger-level variations.

Holt's (1954) conclusion of the nonsignificant heritability of asymmetry was challenged indirectly. Martin *et al.* (1982a) analyzed MZ and DZ twins and found, using the same summary variables as Holt, that total hand asymmetry (summed right hand counts minus summed left hand counts) had a heritability of .52 in males, but was not heritable in females. Using a different measure of asymmetry, Singh (1970) found asymmetry to be significantly heritable. Jantz (1974, 1976) concluded that asymmetry and diversity were under partial genetic control because different populations show different levels of asymmetry. Specifically, African populations showed lower levels of asymmetry than Europeans.

Few studies have used direct evidence to ascertain the genetic components of epidermal ridge variation. Froehlich (1976) found highly significant associations between TRC, digit 1, and digit 4 greater counts and variation at the haptoglobin locus in an analysis of variance in two populations. For both sexes, homozygotes with the 1,1 haptoglobin genotype showed higher mean ridge count values than persons with the 2,2 haptoglobin genotype. Females heterozygotic at the haptoglobin locus had ridge counts at the midpoint between values for each homozygote, but male heterozygotes had values near the 1,1 homozygotes or greater. Approximately 3 or 4% of the variation in the ridge counts was explained by variation in the haptoglobin genotype. Froehlich concluded that his results indicated pleiotropy. Within sibships, the strongest associations of the haptoglobin genotypes were with left digit 1, right digit 2, and digit 5 on both sides. Anderson et al. (1979) analyzed the "trait" of "arch on any digit" in a sample with pedigrees and determined that it was controlled by a dominant major gene linked to the haptoglobin locus. Whether or not "arch on any digit" is a legitimate trait *per se*, the association with the haptoglobin locus was confirmed once again.

Several studies used more direct approaches based on children of MZ twins to estimate intrauterine effects. Reed *et al.* (1979) calculated a high (80%) additive genetic basis for the inheritance of TRC, but found that children of female MZ twins were more similar in finger ridge counts than children of MZ male twins, indicating that the intrauterine environment influences ridge formation. Intrauterine effects lowered variation by 10% in children of MZ females compared to children of MZ males. The intrauterine

effects were strongest for digits 1 and 5, at the periphery of the hand, and were significantly higher in radial than ulnar counts, confirming that different finger ridge counts represent different developmental fields. Cantor (1983) found similar intrauterine effects on finger ridge counts, especially for digits 1 and 5, both in terms of the mother's general intrauterine environment as well as effects specific to each pregnancy. Her estimate of heritability for TRC was only .55 using a model of equal polygenic influences on every digit and finger-specific environmental effects. Estimated heritabilities for individual fingers were lower, and Cantor (1983) concluded that negative environmental covariances would explain why individual finger heritabilities are lower than the heritability of TRC.

The Problem of Repeatability

Holt and others analyzed TRC and other summed counts because of statistical expediency: The distribution of TRC was more normal than individual counts, and as one trait, it enabled simpler univariate statistics. But a statistically convenient *phenotypic* trait is not necessarily a legitimate *genetic* trait, due to the problem of repeatability.

Falconer (1981, 1989) clarified the concept of repeatability and its importance in estimating genetic parameters. Repeatability is a measure of intraindividual variation derived from spatially or temporally repeated measurements that are considered genetically equivalent traits. The intraindividual variation is due to the specialized environment of each trait occurrence. For instance, the number of abdominal bristles can be counted from separate abdominal segments in *Drosophila*, and milk yield or litter size

can be measured repeatedly in mammals. For spatially repeated traits, the intraindividual environmental variation "represents the 'developmental' variation arising from localized circumstances during development" (Falconer 1989: 139).

When estimating genetic parameters, the intraindividual environmental component must be compensated for: The heritability of a trait set cannot be greater than its repeatability. The repeatability is estimated by r, the intraclass correlation (the ratio of between-individual variation to total phenotypic variation) of all measurements (Falconer 1989). In analyzing a composite (summed or averaged) trait, the additive variance is inflated because the specialized environmental variance of each variable within individuals is reduced proportionally by the number of variables measured (Falconer 1989). In the case of TRC, the intraindividual variation is reduced by a minimum factor of 5, for each finger, and if asymmetry is significant, by a factor of 10. Also, because TRC is calculated using only the greater count of the radial and ulnar side of each finger, the within-individual variance may be reduced by an even higher factor. Estimating dominance effects using TRC is also problematic because any finger-level dominance effects will be obscured or nullified by the different additive and dominance effects of the other fingers.

Summing measured traits ***can*** bring about more accurate estimates than single measurements for the additive genetic and general environmental components of variation, but three explicit requirements must be met: 1. The variances of each measurement must

be equal; 2. The variance components of each measurement must be equal; 3. The measurements must represent the same genetic character (Falconer 1989).

The first two requirements for legitimately summing traits are not likely met by finger ridge counts. Weninger (Weninger 1964; Weninger *et al.* 1976; Weninger 1983) has pointed out that the variances of individual finger ridge counts are quite dissimilar. There are also differences between fingers in the components of variation, with broad-sense heritabilities of paired homologous fingers (ignoring asymmetry) ranging from .62 to .94 between sibs and from .80 to 1.0 using doubled parent-child correlations (Holt 1968). Holt's (1953) maximization of the intraclass correlation of finger ridges produced individual finger loadings that differed in magnitude and sign. These very different loadings stand in opposition to treating fingers as genetically equivalent (Rostron 1977). The presence of different dermatoglyphic developmental fields (Cantor 1983; Reed *et al.* 1979; Roberts and Coope 1975; Siervogel *et al.* 1979) suggests that summing finger ridge counts obscures the unique genetic contributions of each finger. Additionally, combining the greater of radial and ulnar counts in TRC can include both ulnar and radial variables, which are influenced by different factors (Roberts and Coope 1975).

Whether or not the first two requirements for analyzing summed traits have been met, it is debatable whether the same finger count represents the same genetic trait. de Wilde (1967; de Wilde and Amesz-Voorhoeve 1979) pointed out that the same greater ridge count for a loop and a whorl are treated equivalently when calculating TRC when

they actually represent different phenomena. Ridge counts for whorls with bicentric cores (Figure 1a) discard information when only greater counts are included, and it has been suggested that the distance between the whorl cores also be recorded (Weninger *et al.* 1976). Minimally, ARC (Absolute Ridge Count, the sum of all radial and ulnar counts from all fingers) is thought to contain more relevant information than TRC. Both Bonnevie (1924) and Cummins and Midlo (1943) suggested that one should record additional shape measurements from finger patterns to account for pattern variations. Bonnevie (1924) discovered that other variables excluded in finger ridge counting, measurements of the relative breadth, height, and tortuosity of finger patterns, also have a genetic basis.

If the requirements for combining measurements have been met, there can be greater precision in estimating genetic parameters, but the difference in precision diminishes as the number of measurements is increased. Falconer (1981, 1989) suggests that it is not worthwhile to combine more than 2 or 3 measurements, and in fact, used TRC as an example of the misuse of repeatability. "The high value [of heritability] for the total count results from the multiple measurement which eliminates all but one-tenth of the environmental component V_{ES} affecting each finger separately" (Falconer 1989: 175). For TRC, the repeatability, the upper bound for heritability if all ten finger ridge counts represented the same genetic trait, is near .50 (see Results). This heritability estimate is much lower than Holt's estimates. The conclusion that finger asymmetry is not heritable (Holt 1954; Martin *et al.* 1982a), based on composite variables, is also questionable. The

problem of repeatability also invalidates composite variables such as "total hand count" (the sum of all finger and palm counts), "total foot count", and other sums of even more variables (Borecki *et al.* 1985; Gilligan *et al.* 1985; Mathew *et al.* 1987; Malhotra *et al.* 1982; Malhotra and Rao 1982). Repeatability also has implications for the traditional mid-parent value, in that the difference between parents is not taken into account, as Galton (1889) recognized.

In using TRC, Holt analyzed a composite trait that was much more likely to show a high heritability because it eliminated at least 90% of the individual variation from finger to finger. As a result, TRC was considered a "better" trait, and in so doing, Holt called a great deal of attention to dermatoglyphics by anthropologists and medical researchers (Roberts 1979). However, analyzing TRC is an indirect and biased approach to examining the genetics of epidermal ridges. The assumption of high heritability for dermatoglyphic traits (Froehlich and Giles 1981a,1981b; Meier 1991; Rogers and Harpending 1983) was based on Holt's results using TRC, rather than on heritabilities using 10 dermatoglyphic variables, which were consistently lower (Holt 1968). There is no evidence that phenotypic dermatoglyphic data will accurately reflect the phylogenetic relationships among populations. What was needed, rather than estimating a *heritability*, is an estimation of the components of variation for finger ridge counts and their relationships to each other. The relationships among variables will be important in assessing population relationships, which must be based on more than one variable to be most useful.

Relationships among fingers may also reveal more about the developmental factors influencing dermatoglyphics.

Chapter 3
DERMATOGENESIS

The genetic and phenotypic characteristics of dermatoglyphics are influenced by development. A well-supported hypothesis for the developmental determination of finger print patterns represents the outcome of complex interrelationships among many regulatory processes during development. As a result, the genetic properties of dermatoglyphics represent the genetic characteristics of regulatory agents and developmental processes.

Epidermal ridges appear on the surface of the skin near the final stage of limb and digit development *in utero* (Babler 1979, 1991; Kimura 1991). At approximately 6-7 weeks postfertilization, the hand plate is paddle shaped, with interdigital notches between thickened areas that are beginning to form the fingers. Volar pads, slightly swollen areas of mesenchymal tissue, appear on the palm (Figure 1c), and the cartilage precursors of hand and finger bones begin forming. The thumb begins to separate from the rest of the fingers. At about 8 weeks, the cartilage precursors begin ossifying, the other fingers begin to separate from each other, and finger pads appear on the ventral apical area of fingers in a radioulnar sequence. The volar pads expand and differentiate until approximately 10 to 11 weeks, when they begin to regress. At this point, primary epidermal ridges appear in the basal layer of the epidermis as intrusions into the outer layer of the dermis. The primary epidermal ridges, with sweat gland precursors at their apices, proliferate until

about the 15th week, when secondary ridges start forming between the primary ridges, and epidermal ridges begin to appear on the volar surface of the fingers. The subsurface ridges appear first on the distal fingers and proceed proximally to the palm. Ridges develop in a radioulnar gradient across the hand as shown by qualitative observations (Hale 1952) and by a gradient across fingers in the Ridge Maturity Index, the ratio of secondary ridges to primary ridges (Babler 1979). By the 17th week, the deepest layer of the epidermis has the adult configuration. Surface ridges, mirroring the primary ridges of the basal epidermis, continue forming until the 24th or 25th week, when ridge formation ceases and the finger prints manifest their lifelong configuration. Ridge development in the feet and toes lags behind that in the hand by one-half to one week, and proceeds in a tibiofibular direction (Babler 1979, 1991; Hale 1952; Kimura 1991).

A Developmental Hypothesis

Specific factors in the development of triradii and curvilinear patterns, which define finger ridge counts, have been proposed. The hypothesis of Mulvihill and Smith (1969) is a more refined version of earlier hypotheses (Bonnevie 1924; Cummins 1926) which proposed that ridge patterns are determined by volar pad topography at the time of ridge formation. According to the hypothesis, the relative timing of ridge formation between pad expansion and regression influences the finger patterns and ridge counts. Specifically, whorls develop earliest, on a swollen and higher pad (which has a larger surface area and volume), loops are formed on an intermediately sized and asymmetrically swollen pad, and arches are formed the latest in development, on a low pad that has

already regressed. Whorls have higher mean ridge counts than loops because they are formed while the pad is larger (Mulvihill and Smith 1969).

Cummins (1926), Penrose (1965), and Mulvihill and Smith (1969) noted that even the most malformed hands have a tendency to form parallel ridges, and interpreted the pattern of epidermal ridges as a consequence of local topographies at the time of ridge formation. Ridge development is seen as a response to areas of growth stress in the hand, and ridges align themselves transversely to the stress to provide the most economical epidermal covering (Penrose 1965). Curvilinear ridges and patterns result from ridges forming on a large convex surface, for instance, on swollen apical finger pads that have not fully regressed. Finger patterns and ridge counts therefore represent the relative degree of finger pad distention at the time of ridge formation (Mulvihill and Smith 1969).

Babler (1987) explicitly tested the Mulvihill and Smith (1969) hypothesis using fetal data. He analyzed the relationship of pad measurements to pattern types and concluded that pad width, rather than pad height, is more highly correlated with pattern type. The pad heights for loops and whorls were approximately equal, but the height for arches was lower, though not significantly. Pad widths differed significantly among all pattern types at $p < .05$. The widest pads were associated with arches, intermediate pads with loops, and whorls with the narrowest pads. Because absolutely narrower pads represent younger individuals, his results confirm that whorls are formed earlier than loops and arches. Once the covariance of pad size and crown-rump length (CRL) was removed,

the correlation of pad width and pattern was more highly significant. Additionally, radial loops, though rare, showed significantly different pad asymmetry compared to ulnar loops, arches, and whorls. Babler concluded that all loops have different pad shapes compared to arches and whorls, and that the shape of the distal phalanx influences pad shape. In sum, Babler's (1987) results support the hypothesis that pad shape influences pattern type, and skeletal factors influence the shape of the pad.

Some researchers have tested the Mulvihill and Smith (1969) hypothesis using data from adults. Siervogel *et al.* (1978, 1979) merged the hypothesis with that of developmental fields, the observation that specific structures are formed during development out of cells according to their position in the body and their relationship to adjacent cells (Butler 1963; Wolpert 1969). Siervogel *et al.* (1978, 1979) found very similar developmental dermatoglyphic fields in all the populations they analyzed. They emphasized differences among radial and ulnar ridge counts, even in whorls of the same individual. Suter and Harvey (1981) and Suter (1982) analyzed pattern distributions and ulnar and radial distributions of ridge counts, and concluded that their results also supported both the Mulvihill and Smith (1969) hypothesis and the presence of developmental fields in the formation of finger patterns and ridge counts.

An opposing hypothesis, that finger patterns influence pad shape rather than vice-versa, has also been presented. de Wilde (1979, 1982) argued convincingly that at least some epidermal ridges, for instance, the proximal and distal interphalangeal creases

(Figure 1c), are established at the hand plate stage of development, and are subsequently divided after the digits separate and grow differentially. Using examples from syndactylism (fused fingers) as well as normal fingers of children, he showed that "bundles" of ridges and creases run continuously across all digits. Thus, the fingers can be seen as adjacent developmental fields, with ridges "invading" one finger and then proceeding to the next before the fingers have separated. The continuous ridge bundles would be present as early as 6 weeks, before the fingers have separated and long before the volar pads have developed.

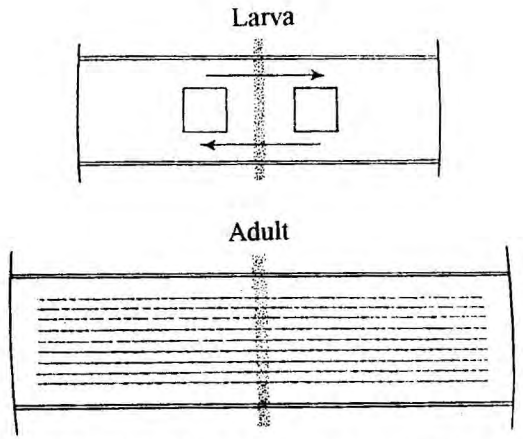
However, de Wilde's (1979, 1982) observations confirming very early continuous ridges do not disprove that finger pad patterns develop later independently. One observes that all finger patterns, in any combination, are connected by the continuous ridge bundles. Actually, the finger pattern areas probably reflect distinct areas of differential growth because the apical ridges of an arch, loop, or whorl reflect the contour of the fingertips, while the more proximal ridges, especially the proximal and distal interphalangeal creases, are more transverse and linear. Finger ridges on the interdigital (radial and ulnar) borders of the fingers are also more transversely oriented and are fewer in number. Thus, when placed side by side, the finger patterns form five interconnected waves, with their swells defined by zones of greater numbers of ridges between the distal interphalangeal crease and the apical ridges of the pattern. These zones probably reflect areas of greater growth, given the greater number of ridges present, but also represent distortions of an earlier ridge pattern.

While the cause and affect relationships of the Mulvihill and Smith (1969) hypothesis have not been established, it is clear that finger osteology, pad shape, pad regression, pattern type, and ridge counts are related statistically. The statistical relationships between these features involve relationships between different developmental systems.

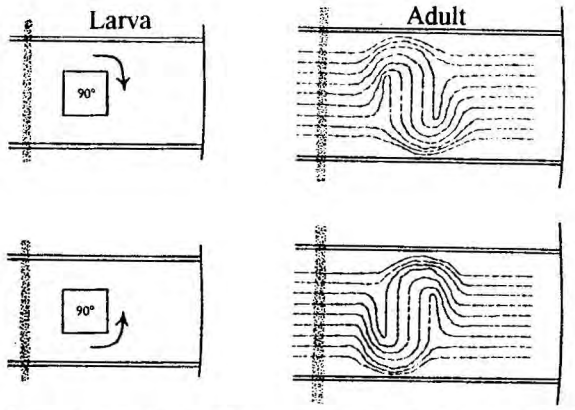
Interactions, Hormones, and Developmental Integration

The finger pad distortions that are triradii, arches, loops, and whorls have usually been described in mathematical rather than biological terms (Mulvihill and Smith 1969; Penrose 1965). However, recent research indicates that pattern types and ridge counts are the product of many biological interactions and influences including cellular behaviors, hormones, natural selection, and developmental regulation.

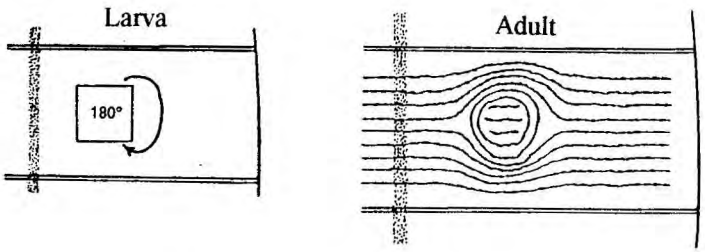
Loops and whorls are found on the finger tips and in several specific areas on the palm associated with dermal pads (Kimura 1991; Penrose 1965). But why these dermatoglyphic patterns develop, and why there are three basic types of finger patterns have remained mysteries. Locke (1959, 1960) has performed transplantation experiments on the abdominal cuticle of the larva of the assassin bug, *Rhodnius prolixus*, which is marked by epidermal ridges in the adult. When sections of larval epidermis from the abdomen were excised and transplanted in a different orientation, they produced ridge patterns in the adult remarkably similar to human finger patterns (Figure 2). The type of pattern produced depended on the anatomical orientation of the areas contributing to and



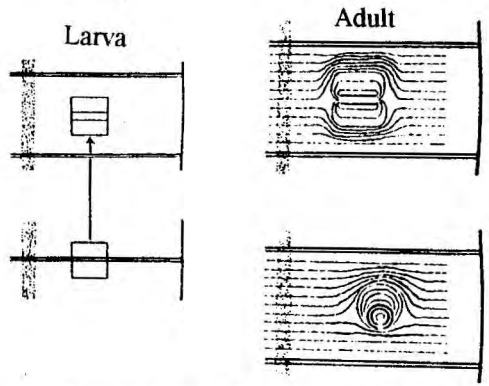
a. Transplants from the same anterior-posterior level produced no change in epidermal ridges.



b. Transplants from the same anterior-posterior level with 90° rotation produced patterns resembling loops.



c. Transplants from the same anterior-posterior level with 180° rotation produced a whorl-like pattern.



d. Transplants from an intersegmental boundary produced patterns resembling whorls.

Figure 2. Results of Epidermal Transplantations in the Abdomen of *Rhodnius prolixus*. Figures 2a,2b, and 2c adapted from Locke, 1959: The cuticular pattern in an insect, *Rhodnius prolixus* Stål. *Journal of Expermental Biology* 36: 459-477. Figure 2d adapted from Locke, 1960: The cuticular pattern in an insect-the intersegmental membranes. *Journal of Expermental Biology* 37: 398-406.

receiving the transplant. The adult insect epidermal ridge configuration resembles an arch, with fairly straight and parallel lines. Bilateral transplants with the same anterior-posterior orientation also resembled an arch, but epidermal sections transplanted bilaterally and rotated 90 degrees produced loops, and transplants from different anterior-posterior locations, intersegmental borders, or rotated 180 degrees produced whorls of various kinds (Locke 1959, 1960). Considering results from experimental transplants in insect larval epidermis, finger patterns may represent areas of relatively greater cellular damage and/or disorientation during pad expansion, with subsequent repair or compensatory growth.

The development of distinctive patterns in the epidermis of insect larva abdomen and human fingers are best explained by tissue interactions. Numerous experiments have shown that cells are "aware" of their position and role during development, and cell "behavior" (proliferation rate, differentiation, migration) is a function of their position relative to other cells (Wolpert 1969). The position and behavior of cells are foundations of the concepts of developmental fields and pattern regulation. These concepts are most dramatically illustrated by experiments involving insects and amphibians including severing a limb, rejoining part of it, and observing the regeneration of missing areas, or grafting severed limbs in different orientations and observing abnormal healing (Bryant *et al.* 1977; Bryant and Muneoka 1986; Holder 1983). In humans, the presence of developmental fields are illustrated by the anomalous development of an extra femur (Bodhurta *et al.* 1989) or the congenital absence of the fibula (Lewin and Opitz 1986). Also, the behaviors

of epidermal cells depend on interactions with other cells. Cells of ectodermal derivatives, such as the cornea, conjunctiva, and mammary glands, which do not form an epidermis, produce epidermal cells when grafted onto dermis which is usually covered by epidermis (Briggaman 1981). The epidermal tissue interactions are not unilateral during development: The presence of the ectoderm is necessary for the proper cartilaginous and osteological development of the limb (Hall 1983; Saunders 1948; Tschumi 1956, 1957). Cells are best understood as interactive elements of the whole organism.

Epidermal cells can clearly be regulated by hormones, and Epidermal Growth Factor (EGF) is one such hormone that also regulates other tissues. EGF induces cell proliferation, migration, adhesion, and/or differentiation, essential for fetal development and wound healing (Chen *et al.* 1994; Wiley *et al.* 1995). There is a high concentration of EGF receptors in sweat duct cells, which are located at the apices of the primary ridges of the basal layer of the epidermis (Hale 1952; Wiley *et al.* 1995). As mentioned, the primary ridges mirror the epidermal surface ridges. Green and Thomas (1978) showed that human epidermal cells *in vitro* tend to form patterns in the presence of EGF. After 30-40 days the cells formed triradii and patterns very similar to arches, loops, and whorls. They extracted epidermal cells from areas that do not normally form dermatoglyphic patterns when exposed to EGF, but were not in contact with dermal cells, with which they usually interact. Green and Thomas (1978) observed that the epidermal cell cultures formed patterns through migrations of cell groups, and whorls took longest to develop. In contrast, cell migrations have not been observed (or looked for) in human finger patterns

in vivo, and whorls are the earliest forming patterns (Babler 1979; Meier 1980). However, in another insect experiment involving the larva of *Rhodnius prolixus*, small epidermal wounds produced cell migrations into the center of the wound area. These cell migrations brought with them specific cellular properties based on their original positions and produced a different epidermal pattern in the adult (Wigglesworth 1937). Cell migrations are also believed to play a role in the formation of "dermatoglyphs" in *Rhodnius* (Locke 1959, 1960). Based on experiments involving human epidermal cells and transplants of larval epidermis, the development of human finger patterns is likely coordinated by EGF and may be due to the repair and compensatory growth of tissues after cellular damage and/or cellular disorientation during finger pad distention.

EGF is not only important in inducing epidermal cells to form patterns, but is also crucial for many other processes during mammalian development. It is important in blastocyst development and retention, and increases fetal viability (Morita *et al.* 1994). EGF is also required for the proper development and differentiation of the fetal digestive tract, lungs, and nervous system, and induces the production of other hormones necessary for development (Ladines-Llave *et al.* 1991; Price *et al.* 1993; Warburton *et al.* 1992; Wiley *et al.* 1995). EGF also influences the eruption of teeth, growth of the incisors, and affects tissues of the palate (Misumi and Ayoshi 1990). Limb development and chondral ossification are also regulated by EGF (Canoun *et al.* 1993), so a correlated response of both ectoderm and phalangeal cartilage precursors to EGF could be a biological

explanation for Babler's (1989) observed correlations between finger patterns and phalangeal ossification.

The role of EGF during development may also explain Babler's (1978) finding that epidermal ridges are indicators of prenatal fitness. He found evidence for directional selection *in utero* for more finger loops and whorls and higher ridge counts in comparing different classes of grossly normal aborted fetuses. Spontaneously aborted fetuses had greater frequencies of arches, but electively aborted fetuses showed significantly lower frequencies of arches that were close to adult frequencies. Early spontaneously aborted fetuses with a lower CRL and unfinished primary ridge formation showed a higher proportion of whorls, and late maturers showed greater frequencies of arches. Also, greater ridge depths were more common with whorls, and arches were associated with shallower ridge depths (Babler 1978). Thus, whorls represent relatively advanced epidermal development, and arches are more common with retarded epidermal development. The effect of EGF, in the development of the epidermis and many other tissue systems, and in fetal implantation and retention, is one likely factor in the relationship between ridge counts and prenatal fitness.

The process of development is a "regulatory, interactive, sequential, hierarchical, and epigenetic process." (Atchley *et al.* 1994: 93), and EGF is only one of many regulating and interacting factors that produce a viable fetus. The extent to which

epidermal ridge counts are developmentally integrated and regulated can be assessed by how interdependent ridge counts are genetically, known as the level of genetic integration:

> "Genetic integration is due to the common effects of genes acting in epigenetic processes, such as those controlling the level of circulating growth hormone and the sensitivity of its target tissues. Thus, developmental integration structures genetic integration" (Cheverud 1996: 45).

The degree of genetic integration can be estimated by examining the genetic covariances among measurements (Cheverud 1984, 1996). Higher genetic covariances in relation to variances represent higher levels of integration and greater developmental regulation, because changes in one variable must be accompanied by changes in other variables (Atchley 1987; Atchley *et al.* 1994; Cheverud 1984, 1996). The opposite of the integration of traits would be the independence of traits (Roth 1996). Developmental integration comes about through epigenetic control, the degree to which regulating genes that control one set of cells controls other sets of cells, such as heritable hormone levels, tissue sensitivities, and tissue interactions, and genetic integration is a result of pleiotropy (Atchley *et al.* 1996; Cowley and Atchley 1992). The pattern of genetic integration may reflect the pattern of stabilizing selection, if deviations among variables influence (or reflect, through pleiotropy) individual fitness (Cheverud 1984; 1996).

Functional constraints can also contribute to genetic integration (Cheverud 1996), but developmental integration probably influences the genetic variance-covariance matrix for dermatoglyphic traits much more than functional integration based on other developmental associations with dermatoglyphics. The concept of morphological integration (Miller and Olson 1958) came about as a result of observations that most phenotypic characters show high correlations among themselves, especially when divided into functional groupings. The greater tactile sensitivity of certain finger configurations (Loesch and Martin 1984) would contribute to the genetic integration of dermatoglyphic traits if an equal level of sensitivity among fingers has a selectional advantage. This advantage has never been demonstrated. However, it has been demonstrated that dermatoglyphics reflect an integrated blueprint for several components of human development and morphology. Meier *et al.* (1987) found correlations between finger pattern intensities and measures of maturation in adolescents. In adults, Loesch and LaFranchi (1990) and Sorenson Jamison *et al.* (1990) found high correlations between dermatoglyphic measures and many body anthropometrics in several populations, especially between hand, wrist and arm measurements, and ridge breadths and thumb variables.

In sum, "The configurations of epidermal ridges that comprise dermatoglyphic traits are, in many respects, a history of the developmental period during which the ridges form." (Babler 1991:95). It is clear that the genetic parameters of dermatoglyphic traits will be influenced by heritable factors that regulate the developmental relationships

between dermatoglyphic traits. In terms of the Mulvihill and Smith (1969) hypothesis, the genetic properties of dermatoglyphics will thus reflect the heritabilities of the timing of pad swelling and regression, and ridge growth initiation and termination. At a deeper level, the genetic properties of dermatoglyphics reflect epigenetic control, and perhaps epidermal wound repair as well. The control of all developmental factors will be measured by the level of integration of the genetic variance-covariance matrix. The inheritance of developmental factors will thus determine whether dermatoglyphic phenotypes (the developmental products) can be used as proxies for dermatoglyphic genotypes (the development plans) in estimating the phylogenetic relationships among populations.

Chapter 4
MATERIALS AND METHODS

Samples

Data for this analysis come from the Heinz Brehme dermatoglyphic data base at the University of Tennessee, Knoxville. This collection includes pattern types and ridge counts from fingers, palms, soles and toes from over 60,000 persons from around the world collected by Brehme and other researchers. All finger ridges were counted by Brehme following the methods of Brodhage and Wendt (1951) and Baitsch and Schwarzfischer (1959), so interobserver error is not present. Ridge counts were not transformed before analysis.

The dermatoglyphic data with pedigrees come from families from Berlin, Freiburg, and Hesse, in Germany, and Vienna, Austria (Table 1). Nearly all pedigrees represent a complete family. In the finger and palm data, only seven families had data from only one parent, but some individuals had one or several measurements missing and were not included in every analysis. Families with triplets were not included. The families with toe data are a subset of the other families.

Table 1. Dermatoglyphic Data from German Families for Estimating Genetic Parameters.

Data Set	Total individuals	Males	Females	Pedigree n	Mean n per Pedigree	Twin Pairs MZ	Twin Pairs DZ
Fingers and Palms	4091	2032	2059	812	5.0	121	154
Toes	2597	1252	1345	575	4.5	121	151

The population samples are shown in Table 2. All but one of the population samples are from central European cities where German is spoken. The sample from Hungary consists of German-speaking populations and is presumably of German extraction. Means for the family and population samples are presented in Appendix 1.

Table 2. Dermatoglyphic Samples Used in Population Comparisons.

Location	N	Males	Females
Berlin	556	209	347
Freiburg	161	72	89
Hungary	236	105	131
Kiel	216	109	107
East Friesland	104	61	43
Vienna	1038	519	519

Variance Components and Maximum Likelihood

The components of phenotypic variance, with the explicit assumption of no epistasis or interactions between components of variation, can be decomposed into additive, dominance, and environmental variances:

$$V_P = V_A + V_D + V_E$$

where V_P is the phenotypic variance, V_A is the additive variance, V_D is the dominance variance, and V_E is the environmental variance (Falconer 1989). Additive genetic variance is important because it is the amount of genetic information that can consistently be passed from parent to offspring. The ratio of additive genetic to phenotypic variance is known as the narrow-sense heritability (Wright 1921). If dominance effects are included, the ratio of additive plus dominance variance divided by the phenotypic variance equals the broad-sense heritability, which is also known as the degree of genetic determination for a trait.

Put another way, the narrow-sense heritability expresses the extent to which a child resembles both of its parents, and the broad-sense heritability also incorporates similarities among siblings and similarities to one parent to the exclusion of the other. Heritability is a concern especially in animal breeding and evolution, where the response to selection can be predicted by the amount of additive variance present. Traits with little or no additive genetic variance are not expected to respond to selection, whether natural or artificial.

Most methods of estimation of genetic parameters require normality or multivariate normality of the trait distribution, no epistasis, and no genotype-environmental interaction. In estimating variance components in humans as opposed to in animals, there are several additional concerns. First, the most basic problem is a small sample size, because human data are usually collected from individuals, not families. Second, the data are usually unbalanced, with various numbers of children in each pedigree. Third, forced matings are not used between selected strains or individuals. This generally leads to inconsistent data collection, with a variety of pedigree relationships available for analysis. Thus, human data usually consist of small numbers of pedigrees with varying numbers of children in each family, and estimates of genetic parameters can be based on different combinations of relatives. As a result, data from human monozygotic and dizygotic twins are especially appealing.

In the Biometric school, statisticians such as Karl Pearson tried to explain family resemblance in continuous characters in terms of correlation and regression (Hopper

1993). This methodology fragments the data into relationship categories and derives separate estimates based on each. Twins naturally pair up in an analysis, but information from their parents is left out. Parameter estimation involving twins alone is biased because of dominance. For non-twins, data from individuals must be separated and then paired up in various combinations, leading to a loss of information. For instance, in parent-child regression, each parent can be paired either with only one child, with the mean of child values, or each parent can be used more than once. The researcher must either discard information and obtain larger standard errors, or include redundant data and seriously underestimate standard errors. Also, the standard errors of heritabilities based on doubling the regression coefficient must be doubled, and more than one variable cannot be analyzed reliably using multiple regression (Hopper 1993). Ultimately, regression and correlation methods are too limiting, and likely led to Holt's use of a summary variable, TRC, and likewise her results, expressed in one heritability. A genetic analysis of traits should provide much more: "Rather than presenting results in term of heritabilities, however, the conclusions should address the strength and limitations of the design, and emphasize the statistical precision of the results and the statistical power of the data to resolve the issues." (Hopper 1993:219)

In using fixed-effects ANOVA, the Birmingham School, which includes Mather, Jinks, Eaves, and Martin, among others, is hampered by problems similar to the Biometric school (Hopper 1993). Any data sets must be consistent, either from siblings, twins, or parent-child, and these cannot be mixed together. This can render goodness of fit tests

invalid due to a reduction in degrees of freedom. Additionally, many different models can fit the data equally well, and there is no way of objectively evaluating one model versus another (Wilson 1982). In an analysis of variance using balanced data, deviations from the grand mean can be modeled by the influences of various factors, and a unique solution exists. However, unbalanced designs produce no unique solution to partition the sum of squares (Hopper 1993).

Maximum likelihood is by far the best method for the multivariate estimation of genetic components, and deals quite well with the peculiarities of human data. Unbalanced data are handled much better, and more reliable estimates are possible because all relationships are included in an analysis, including twins and extragenerational relationships (Hopper 1993; Lange *et al.* 1976; Shaw 1987). In comparison to ANOVA, the degrees of freedom are greatly increased using ML, so there is more power for parameter tests (Hopper 1993). Maximum likelihood is model-bound, but allows flexible modeling, and a model's goodness of fit can be calculated. Different models can be specified and the improvement in fit can be tested by the likelihood ratio criterion. Epistasis and genetic-environmental interaction effects can be specified. Residuals can be calculated and examined, and their distribution can be informative. Deviations in residuals can be attributed to a major gene effect, a small number of genes influencing the trait, or an undetected covariate. The statistical significance of specific parameters can also be evaluated by examining the standard errors. Additionally, in multivariate analyses, the

covariances are part of the iterated model and are dealt with far better than in path analysis or ANOVA.

There are costs to using ML methods, among which is time. ML estimates require much more processing time than other methods, but this time differential is diminishing with faster and faster computers. Maximum likelihood is also sensitive to nonnormality, especially kurtosis, but this affects the estimated parameters little, influencing significance testing to a greater degree. Significant skewness affects the estimated means (Hopper 1993). Traits within a pedigree are assumed to be multivariate normal, and the central limit theorem would help large samples meet this criterion. The more important requirement of approximate multivariate normality must be satisfied by the residuals of the fitted model, which depend on the original data, any scale transformations, and the effects in the model. Additionally, ML estimates can be biased, but the bias can be minimized by the use of relatively small pedigrees compared to the total number of pedigrees (Hopper and Mathews 1994; Shaw 1987). When large samples are available, asymptotic estimates are much better than estimates from smaller samples (Hopper 1993).

Maximum likelihood estimation depends on an explicit model, but is very flexible as to what can go into the model. A basic model for covariance matrices based on independent genetic and random environmental factors is

$$\Omega = 2V_A \Phi + V_D \Delta_7 + V_E I$$

where Ω is the phenotypic variance-covariance matrix, V_A, V_D, and V_E are as above, Φ is a matrix of kinship coefficients, Δ_7 is a matrix of Jacquard's condensed coefficient of identity, and I is the identity matrix. The kinship matrix represents the proportion of alleles that relatives have in common with each other. For example, a parent and child will have 1/2 of their alleles in common, as will full siblings on average. Jacquard's coefficient of identity Δ_7 is the proportion of allele pairs that relatives have in common, and is 0 for a parent and child, 1/4 for full siblings, and 1 for monozygotic twins (Jacquard 1974). In a multivariate analysis, estimates of variance components are based on

$$\Omega = V_A \otimes 2\Phi + V_D \otimes \Delta + V_E \otimes I$$

where $\otimes$ represents the Kronecker products and other symbols are as above (Konigsberg and Ousley 1995). For testing goodness of fit, the log likelihood of the parameters is based on the data (X) across all individuals in a pedigree and is

$$\ln L\,(\mu, V_A, V_D, V_E \mid X) = -\tfrac{1}{2} \ln |\Omega| - \tfrac{1}{2} [\mathrm{vec}(X-\mu_j)'\, \Omega^{-1}\, \mathrm{vec}(X-\mu_j)] - \tfrac{p}{2} n \ln (2\pi)$$

where μ_j is a vector of phenotypic means for each variable, n is the number of individuals in the pedigree, p is the number of variables, vec converts the columns of a matrix into a column vector, and other symbols are as above (Konigsberg and Ousley 1995). The log likelihoods are summed across all pedigrees in a sample and through iteration, parameter estimates converge on values that produce an increasingly better fit to the data (as measured by greater likelihoods) until no more improvement can be made. The maximum likelihood solution represents the best estimation of parameters among all individuals in every pedigree (Hopper 1993). MYFISH, a computer program written in FORTRAN by

Lyle Konigsberg, was used to calculate maximum likelihood parameters. MYFISH does not produce predicted values, residuals, and outlier statistics for data sets as does FISHER (Lange 1988), but a model with additive, dominance, and environmental effects was already part of MYFISH and it is easier to change models with few modifications. Calculations using MYFISH were performed on several University of Tennessee mainframes and a personal computer. A MYFISH analysis of 5 variables ran for roughly 3 hours, and 10 variables took approximately one week. At least 100 separate analyses were run using various parts of the data set.

Other Multivariate Methods

Several techniques were applied to analyze estimated variance-covariance matrices. Principal Components Analysis (PCA) maximizes interindividual multivariate differences in all variables through linear combinations of variables along orthogonal variance-maximizing axes, called the principal components or eigenvectors. The multivariate relationships between variables can be examined through the principal component weights for each variable and the amount of variation explained by each axis. PCA can utilize original data, the correlation matrix, or the variance-covariance matrix. Matrix correlations were calculated using NT-SYS pc (Rohlf 1993). DIST, a FORTRAN program that performs permutation tests for comparing matrices, was provided by E. Jacquelin Dietz (Dietz 1983). DIST calculates Mantel's Z, Spearman's rho (R), and Kendall's tau (K_c) statistics from lower triangle similarity or distance matrices. Z is sensitive to changes of scale, R and K_c are scale-independent nonparametric procedures,

and K_c incorporates both within- and between-matrix comparisons. The program provides probabilities for the hypothesis of no matrix association.

Heritabilities were calculated by dividing the additive genetic variance by the phenotypic variance for univariate analyses, and by multiplying the inverse of **P**, the phenotypic variance-covariance matrix by **G**, the additive genetic variance-covariance matrix for multivariate analyses ($\mathbf{P}^{-1}\mathbf{G}$). For a set of completely heritable traits, $\mathbf{P}^{-1}\mathbf{G}$ would equal the identity matrix, with ones on the diagonals and zeros on the off-diagonals. If **G** were perfectly proportional to **P**, the diagonals (heritabilities) would have the same values and the off-diagonals would be zero. If the diagonals of $\mathbf{P}^{-1}\mathbf{G}$ are very different and the off-diagonals are not zero, the product $\mathbf{P}^{-1}\mathbf{G}$ merely indicates that the phenotypic variance-covariance matrix is not proportional to the genetic variance-covariance matrix.

Other techniques were used to analyze the population data. A PCA of the genetic variance-covariance matrix was used to calculate additive genetic values for a comparison of genetic and phenotypic ridge counts from populations. Canonical discriminant analysis was also used to compare genetic and phenotypic values. It is similar to PCA but it separates groups, rather than individuals, along multiple variance maximizing axes using a linear combination of group means and the pooled variance-covariance matrix. It also produces a distance matrix between all groups using all canonical axes (Blackith and Reyment 1971; Tatsuoka 1988). Both PCA and canonical discrimination were performed on a personal computer using SAS (SAS Institute 1985). Version 3 of the program RMET (Relethford and Blangero 1990;

Relethford *et al.* 1997) was used to perform PCA, compute the **R** matrix (a matrix of the relative divergence of each group), and calculate F_{ST} (Rogers and Harpending 1986; Wright 1951), using quantitative traits. RMET also provides a distance matrix and standard errors of distances. The methods are based on those utilized in the analysis of allele frequencies among populations (Harpending and Jenkins 1973).

A clustering program in NT-SYS pc (Rohlf 1993) was used to elucidate relationships between digits (using the variance-covariance and correlation matrices) and populations (using the distance matrix). Clustering output incorporates all the variation from multidimensional axes in a 2 dimensional plot or dendrogram. The UPGMA (unweighted pair-group method, arithmetic average) algorithm was used in all comparisons. UPGMA first joins the two most similar groups in a sample, treats them as one group, calculates the mean distance to other sample groups, and then finds the closest pair of groups once again. This procedure is repeated until all groups are connected.

Chapter 5
RESULTS

Dermatoglyphic data have been described as some of the most intractable data available due to their unusual distributions. To some degree this is illustrated in the present sample. First, the distribution of TRC in the German family data is significantly negatively skewed (Figure 3), as has been found in many other population samples. Ulnar count distributions are dominated by a great many zeros, and it is difficult to imagine any transformation that would make them useable (Figure 4). No ulnar counts from fingers could be analyzed using the maximum likelihood model. Greater counts will be used only to investigate the properties of TRC. As mentioned, TRC combines both ulnar and radial variables, which are influenced by different factors (Roberts and Coope 1975). In the family sample, between 92% and 99% of the finger greater ridge counts are radial counts, except for digit 2, where only 47% are radial counts. Radial ridge counts will be more

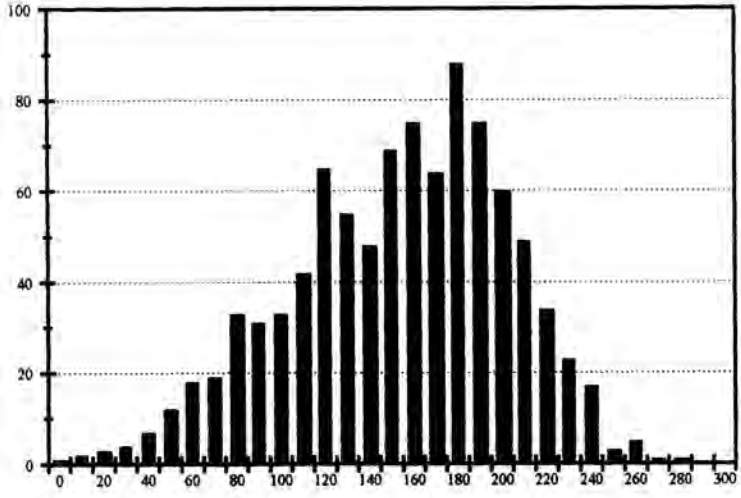


Figure 3. Distribution of TRC for Males.

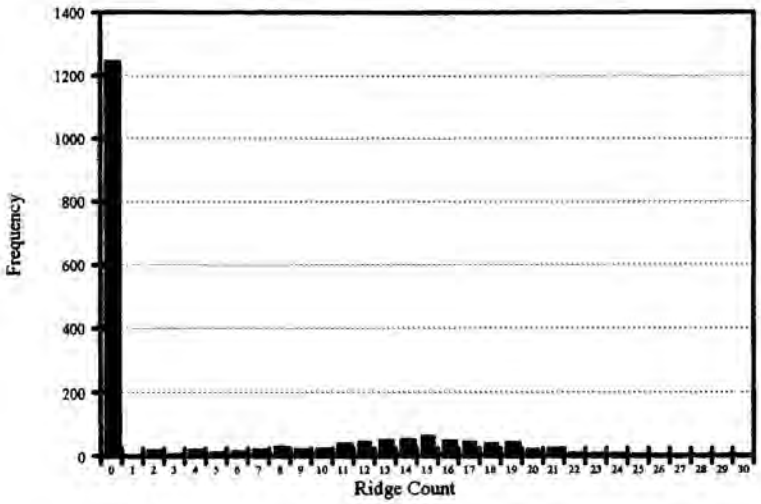


Figure 4. Distribution of Right Digit 4 Ulnar Ridge Counts for Males.

thoroughly analyzed because they are more normally distributed than ulnar counts and more likely represent a biologically meaningful grouping of variables.

Any analysis of ridge counts will also involve factors relating to the relationship between pattern types on ridge counts. Individuals with a higher PII (the total number of triradii across all fingers) have greater ridge counts per pattern (Figure 5). Individuals with low PIIs have more arches (which have ridge counts of zero), as well as fewer ridges per pattern (overwhelmingly loops). Additionally, the PII distribution is negatively skewed (Figure 6). Similar results have been found in other populations (Suter and Harvey 1981). Another complicating observation is that the distribution of ARC (the sum of radial and ulnar counts from all fingers) for the German family sample is *positively* skewed.

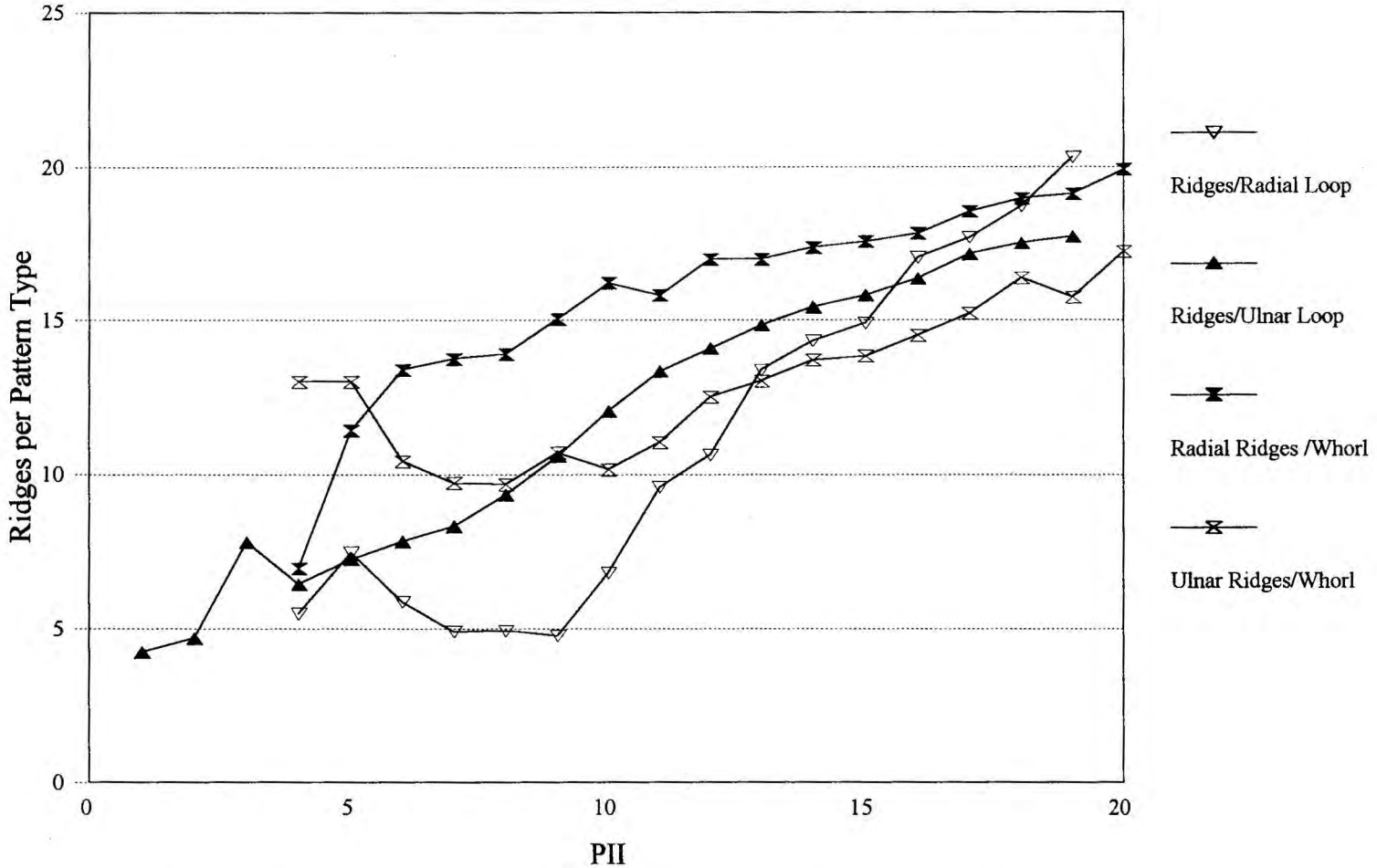


Figure 5. Pattern Intensity Index (PII) and the Number of Ridges per Pattern.

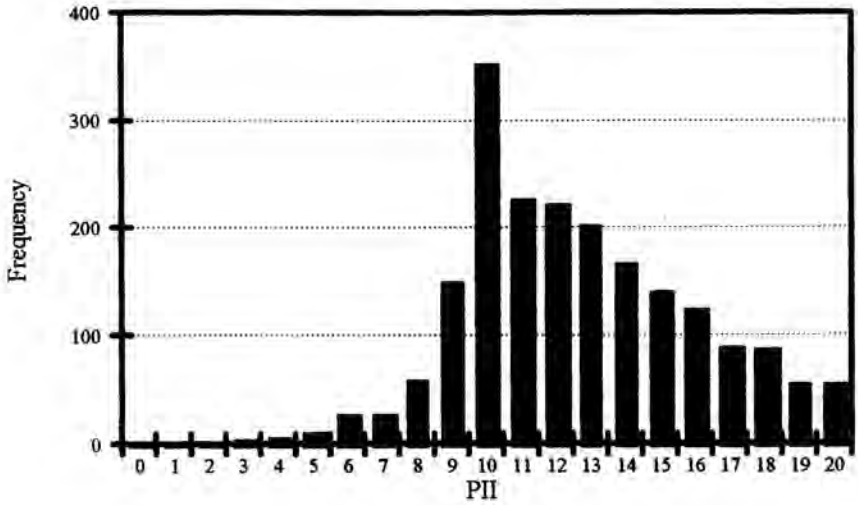


Figure 6. PII Distribution for Males.

Components of Variation in Finger Ridge Counts

First, TRC and its components of variation will be investigated. The repeatabilities of TRC using the intraclass correlation are .46 for females, .49 for males, and .57 for pooled sexes, with a 95% confidence interval from .50 to .65. These values are the maximum possible true heritability of TRC and indicate high intraindividual diversity that is hidden when the individual counts are summed. Table 3 shows the multivariate variance-covariance matrices, and Table 4 shows the narrow and broad $\mathbf{P}^{-1}\mathbf{G}$ matrices for the greater counts of all ten fingers and the univariate ML heritabilities for each finger. The covariance and correlation matrices will be examined in greater detail for the radial counts. For now, examination of the heritabilities for the greater counts are of main interest. The multivariate heritabilities, on the diagonal of each matrix, are much lower than the univariate heritabilities. The univariate narrow heritabilities are in the range of .62 to .70 with a mean of .66, and univariate broad sense heritabilities fall between .73 and .81 with a

Table 3. Variance-Covariance Matrices for Finger Greater Ridge Counts. LGC1-RGC5: Left Greater Ridge Count for digit 1 through Right Greater Ridge Count for digit 5.

	LGC1	LGC2	LGC3	LGC4	LGC5	RGC1	RGC2	RGC3	RGC4	RGC5
Additive										
LGC1	28.0									
LGC2	21.0	31.6								
LGC3	19.5	26.8	29.8							
LGC4	16.2	23.4	24.6	25.7						
LGC5	14.6	19.3	17.9	18.9	19.5					
RGC1	24.1	18.3	16.7	14.1	12.4	22.9				
RGC2	21.3	32.8	27.8	24.0	19.8	18.4	35.0			
RGC3	17.5	25.0	27.7	22.7	16.1	15.1	26.4	25.9		
RGC4	16.1	24.5	24.2	25.0	18.4	13.5	24.0	22.4	24.8	
RGC5	13.6	18.5	17.2	18.1	19.5	12.3	19.0	15.8	18.0	20.1
Dominance										
LGC1	3.7									
LGC2	1.7	8.9								
LGC3	-0.1	3.6	2.7							
LGC4	1.2	4.1	4.1	5.7						
LGC5	1.7	1.7	2.6	3.6	4.5					
RGC1	5.2	3.5	3.5	3.9	4.7	8.8				
RGC2	2.8	7.1	4.4	4.2	3.8	6.1	8.7			
RGC3	1.1	3.4	4.1	3.5	3.7	5.3	5.5	8.1		
RGC4	1.3	2.0	5.3	6.1	5.8	7.1	5.9	5.9	11.1	
RGC5	1.5	3.8	2.3	4.2	4.5	4.7	5.6	5.1	7.6	7.7
Environmental										
LGC1	8.4									
LGC2	-0.4	11.5								
LGC3	1.1	1.2	11.9							
LGC4	0.8	-0.3	0.6	9.8						
LGC5	-0.5	0.4	0.3	1.6	6.1					
RGC1	-0.1	-1.0	-0.2	1.7	0.2	10.3				
RGC2	-1.1	1.1	0.9	1.1	-0.2	0.2	15.1			
RGC3	0.1	1.0	0.4	0.3	-0.6	0.6	1.7	7.3		
RGC4	0.0	0.4	-1.0	3.1	0.2	1.0	1.4	0.7	9.1	
RGC5	-0.2	-0.7	1.0	1.4	1.1	1.6	1.0	0.7	1.9	6.1

Table 4. $\mathbf{P}^{-1}\mathbf{G}$ Matrices and Univariate Heritabilities for Finger Greater Ridge Counts. The multivariate heritabilities are on the diagonals of the heritability matrices. Abbreviations are the same as in Table 4.

	LGC1	LGC2	LGC3	LGC4	LGC5	RGC1	RGC2	RGC3	RGC4	RGC5
Narrow Sense										
LGC1	0.50	0.19	0.18	0.14	0.16	0.36	0.19	0.15	0.15	0.12
LGC2	0.07	0.24	0.07	0.07	0.07	0.08	0.23	0.06	0.13	0.07
LGC3	0.09	0.11	0.29	0.13	0.05	0.07	0.10	0.27	0.11	0.05
LGC4	-0.04	-0.02	0.10	0.24	0.08	0.00	0.02	0.09	0.21	0.04
LGC5	0.09	0.16	0.09	0.16	0.33	0.02	0.16	0.04	0.12	0.33
RGC1	0.14	-0.08	-0.11	-0.14	-0.12	0.24	-0.10	-0.11	-0.16	-0.10
RGC2	0.01	0.17	0.01	-0.02	0.00	-0.01	0.23	0.03	-0.03	-0.01
RGC3	0.01	0.07	0.27	0.12	-0.01	0.00	0.11	0.26	0.11	0.00
RGC4	-0.01	0.11	0.04	0.13	-0.05	-0.07	0.01	0.04	0.15	-0.06
RGC5	-0.04	-0.08	-0.08	-0.03	0.24	0.02	-0.06	-0.06	0.00	0.30
Broad Sense										
LGC1	0.53	0.08	0.03	0.03	0.08	0.33	0.08	0.01	-0.02	0.04
LGC2	0.07	0.46	0.10	0.10	0.01	0.03	0.30	0.02	0.00	0.07
LGC3	-0.01	0.07	0.22	0.14	0.02	0.01	0.06	0.20	0.16	-0.06
LGC4	-0.03	0.13	0.12	0.33	0.01	-0.13	-0.04	0.00	0.09	-0.04
LGC5	0.12	-0.04	0.13	0.15	0.45	0.12	0.10	0.07	0.15	0.27
RGC1	0.27	0.01	0.01	-0.04	-0.01	0.44	0.04	0.03	0.03	-0.03
RGC2	0.05	0.22	0.03	-0.03	0.02	0.03	0.32	0.04	0.00	0.00
RGC3	0.00	0.02	0.33	0.07	0.02	0.04	0.12	0.52	0.08	0.06
RGC4	-0.03	-0.07	0.16	0.20	0.07	0.11	0.05	0.08	0.46	0.09
RGC5	-0.06	0.10	-0.18	0.00	0.24	-0.05	0.03	-0.02	0.07	0.53
Univariate heritabilities										
Narrow	0.68	0.60	0.69	0.66	0.70	0.65	0.62	0.68	0.64	0.69
Broad	0.79	0.78	0.77	0.80	0.78	0.74	0.73	0.81	0.78	0.81

mean of .78. The univariate mean heritability for greater counts from all fingers is .28 and only one variable, the greater count of left digit 1, approaches the repeatability of TRC with a heritability of .50.

Any biases in heritability estimates of a composite trait such as TRC would be illustrated by an ML estimation, but a solution for TRC would not converge. However, an analysis of total radial count (the sum of all radial counts on all fingers) did converge. The ML estimate of narrow-sense heritability for total radial count is .84, and for broad sense heritability, .93. Table 5 shows the multivariate heritabilities for radial counts, which range from .23 to .48, with a mean of .31. The multivariate heritabilities of the radial counts are slightly higher than the multivariate heritabilities of the greater counts. The heritability matrices in Table 5 will be discussed in greater detail below. In the best case, the true "heritability" of TRC would be estimated as .84 x .57 (the pooled-sex repeatability) = .48, near Cantor's (1983) heritability estimate of .55 for TRC. In the worst (multivariate) case, however, the estimated heritability is near .31 based on the mean heritability of every finger. Greater values can be obtained using the proportion of phenotypic variance in the additive genetic matrix (.63) or the proportion of summed variances and covariances (.81). These are near Cantor's mean estimated finger heritability of .68 using her full genetic and environmental model.

Due to the heritability bias of TRC, and the extremely nonnormal distributions of ulnar counts (Figure 4), a multivariate examination of all radial counts was performed. The

Table 5. $\mathbf{P}^{-1}\mathbf{G}$ Matrices, Differences, and Univariate Heritabilities for Radial Counts. The multivariate heritabilities are on the diagonals of the heritability matrices. LRC1-RRC5: Left radial ridge count for digit 1 through right radial ridge count for digit 5.

Narrow Sense

	LRC1	LRC2	LRC3	LRC4	LRC5	RRC1	RRC2	RRC3	RRC4	RRC5
LRC1	0.48	0.05	0.09	0.07	0.08	0.33	0.07	0.05	0.06	0.03
LRC2	0.01	0.30	0.08	0.03	0.03	0.01	0.13	0.03	0.01	0.03
LRC3	0.10	0.17	0.27	0.11	0.05	0.08	0.10	0.25	0.09	0.04
LRC4	-0.03	-0.01	0.06	0.25	0.05	-0.06	-0.12	0.03	0.16	0.00
LRC5	0.08	0.14	0.09	0.18	0.35	0.02	0.19	0.03	0.13	0.32
RRC1	0.17	0.01	0.00	-0.06	-0.04	0.27	0.01	0.03	-0.05	0.00
RRC2	0.02	0.13	0.02	-0.05	0.03	0.01	0.31	0.06	-0.01	-0.01
RRC3	0.03	0.05	0.28	0.14	0.01	0.06	0.14	0.26	0.17	0.02
RRC4	0.01	0.00	0.07	0.16	0.00	0.00	0.04	0.11	0.23	0.03
RRC5	-0.01	0.00	-0.03	0.02	0.26	0.07	-0.07	-0.03	0.08	0.35

Broad Sense

	LRC1	LRC2	LRC3	LRC4	LRC5	RRC1	RRC2	RRC3	RRC4	RRC5
LRC1	0.44	0.09	0.03	0.03	0.08	0.37	0.05	-0.04	-0.02	0.07
LRC2	0.03	0.35	0.03	0.00	-0.01	-0.01	0.17	0.02	0.05	0.00
LRC3	0.03	0.02	0.24	0.18	0.01	0.00	0.05	0.23	0.17	-0.07
LRC4	0.02	-0.06	0.14	0.35	0.02	-0.03	-0.16	0.04	0.14	0.07
LRC5	0.11	0.06	0.14	0.10	0.44	0.07	0.05	0.11	0.14	0.28
RRC1	0.34	0.01	0.02	-0.02	-0.02	0.42	0.09	0.06	0.02	-0.05
RRC2	0.06	0.15	0.04	-0.05	0.00	0.07	0.31	0.06	0.00	0.03
RRC3	-0.03	0.12	0.34	0.09	0.07	0.01	0.18	0.44	0.03	0.06
RRC4	-0.02	0.19	0.19	0.20	0.06	0.06	0.13	0.06	0.43	0.02
RRC5	-0.01	-0.03	-0.19	0.09	0.27	-0.06	0.02	-0.06	0.02	0.50

Multivariate
Broad - Narrow

-0.05	0.05	-0.03	0.09	0.09	0.15	0.00	0.18	0.20	0.16

Univariate Narrow

0.69	0.55	0.67	0.66	0.70	0.63	0.52	0.65	0.62	0.69

variance-covariance matrices are shown in Table 6, and the correlation matrices are shown in Table 7. The standard errors could not be calculated for all 10 counts; significance figures are based on separate analyses of the left and right radial ridge counts. All values in the additive genetic matrix are much more than three times their standard errors ($p < .01$), which ranged between 1.0 and 2.3. Values from all other matrices are considered significant at $p < .05$ if they are at least two times greater than their estimated standard errors.

In the additive genetic variance-covariance matrix for radial ridge counts (Table 6), all covariances are high and positive, a typical pattern in many morphometric analyses involving size (Atchley 1984; Cheverud 1982; Olson and Miller 1958). This is the "size effect" of Holt (1952), and in developmental terms, indicates a high degree of genetic and developmental integration (Atchley 1984; Atchley et al. 1994; Cheverud 1984). Radial ridge counts also show the proximity effect that Galton (1892) and Holt (1952, 1959) noticed, with covariances highest for homologous fingers, high for adjacent fingers, and high for fingers adjacent to homologous fingers. The proximity effect is repeated in the additive genetic correlation matrix (Table 7), with homologous digits 3,4, and 5 showing coefficients of 1.0, .97, and .98, respectively. Digit 2 has the lowest homologous correlation at .86.

The dominance matrix for radial ridge counts (Table 6) shows a different pattern from the additive genetic matrix. Only digits 1, 3, 4, and 5 on the right hand and digit 4 on

Table 6. Variance - Covariance Matrices for Finger Radial Counts. Figures in Bold are Significant at $p < .05$. Abbreviations are the same as in Table 5.

	LRC1	LRC2	LRC3	LRC4	LRC5	RRC1	RRC2	RRC3	RRC4	RRC5
Additive Genetic										
LRC1	**28.38**									
LRC2	**16.36**	**27.66**								
LRC3	**19.36**	**23.77**	**29.21**							
LRC4	**16.16**	**18.57**	**24.22**	**27.05**						
LRC5	**14.43**	**16.00**	**17.97**	**19.73**	**20.26**					
RRC1	**24.69**	**14.82**	**17.25**	**14.02**	**12.71**	**23.83**				
RRC2	**15.60**	**23.16**	**20.91**	**14.73**	**14.21**	**14.10**	**26.44**			
RRC3	**16.91**	**20.24**	**26.29**	**21.36**	**15.31**	**15.66**	**19.75**	**23.77**		
RRC4	**16.17**	**18.50**	**24.06**	**26.27**	**19.56**	**14.37**	**16.13**	**21.95**	**27.06**	
RRC5	**13.62**	**15.37**	**17.34**	**18.95**	**20.20**	**12.92**	**12.87**	**15.03**	**19.60**	**21.02**
Dominance Genetic										
LRC1	2.44									
LRC2	1.70	2.57								
LRC3	-0.29	-0.32	1.89							
LRC4	1.42	1.27	**3.73**	**4.71**						
LRC5	1.55	0.17	1.20	**2.04**	**2.67**					
RRC1	**4.86**	1.56	0.58	2.22	1.79	**6.07**				
RRC2	1.98	2.51	1.20	1.35	0.22	**3.09**	1.90			
RRC3	-0.00	2.07	2.56	1.99	2.01	1.05	2.18	**4.45**		
RRC4	0.92	**3.60**	**4.14**	**4.42**	2.81	2.62	**2.64**	1.47	**6.40**	
RRC5	1.44	0.66	-0.08	2.95	2.37	0.68	1.78	1.45	**2.61**	**3.65**
Environmental										
LRC1	**9.65**									
LRC2	-0.24	**19.26**								
LRC3	0.89	**2.94**	**12.58**							
LRC4	0.44	0.86	-0.20	**9.97**						
LRC5	-0.48	0.61	0.17	1.27	**5.99**					
RRC1	0.09	0.17	0.18	0.82	0.34	**10.46**				
RRC2	-0.83	3.12	1.40	1.19	0.03	-1.64	**21.31**			
RRC3	1.34	1.11	0.20	0.17	-0.80	0.23	1.00	**8.56**		
RRC4	0.27	-1.64	-1.33	**3.03**	0.55	0.50	-0.49	0.13	**9.67**	
RRC5	-0.23	1.22	1.70	0.82	1.11	1.36	0.08	0. 38	**1.82**	**6.12**

Table 7. Correlation Matrices for Finger Radial Counts. Abbreviations are the same as in Table 5.

	LRC1	LRC2	LRC3	LRC4	LRC5	RRC1	RRC2	RRC3	RRC4	RRC5
Additive Genetic										
LRC1	-									
LRC2	0.58	-								
LRC3	0.67	0.84	-							
LRC4	0.58	0.68	0.86	-						
LRC5	0.60	0.68	0.74	0.84	-					
RRC1	0.95	0.58	0.65	0.55	0.58	-				
RRC2	0.57	0.86	0.75	0.55	0.61	0.56	-			
RRC3	0.65	0.79	1.00	0.84	0.70	0.66	0.79	-		
RRC4	0.58	0.68	0.86	0.97	0.84	0.57	0.60	0.87	-	
RRC5	0.56	0.64	0.70	0.79	0.98	0.58	0.55	0.67	0.82	-
Dominance Genetic										
LRC1	-									
LRC2	0.68	-								
LRC3	-0.13	-0.15	-							
LRC4	0.42	0.37	1.25	-						
LRC5	0.61	0.07	0.53	0.57	-					
RRC1	1.26	0.39	0.17	0.42	0.44	-				
RRC2	0.92	1.14	0.63	0.45	0.10	0.91	-			
RRC3	-0.00	0.61	0.88	0.43	0.58	0.20	0.75	-		
RRC4	0.23	0.89	1.19	0.81	0.68	0.42	0.76	0.27	-	
RRC5	0.48	0.22	-0.03	0.71	0.76	0.14	0.68	0.36	0.54	-
Environmental										
LRC1	-									
LRC2	-0.02	-								
LRC3	0.08	0.19	-							
LRC4	0.04	0.06	-0.02	-						
LRC5	-0.06	0.06	0.02	0.16	-					
RRC1	0.01	0.01	0.02	0.08	0.04	-				
RRC2	-0.06	0.15	0.09	0.08	0.00	-0.11	-			
RRC3	0.15	0.09	0.02	0.02	-0.11	0.02	0.07	-		
RRC4	0.03	-0.12	-0.12	0.31	0.07	0.05	-0.03	0.01	-	
RRC5	-0.03	0.11	0.19	0.11	0.18	0.17	0.01	0.05	0.24	-

the left hand have significant dominance variance at $p < .05$. There are also relatively fewer significant dominance covariances than in the additive genetic matrix. The significant dominance covariances involve digit 4 with digits 2,3, and 5, and right digit 1 and 2. Right digit 4 shows strong dominance covariance with left digits 2 and 3, and these covariances with digit 4 are higher than their variances. Left digit 1, which does not show significant dominance variance, shows significant dominance covariance with right digit 1. Thus, every finger has significant dominance variance or covariance at $p < .05$. At $p < .01$, the significant variance pattern is simplified somewhat, with left digits 4 and 5 and right digits 3,4, and 5, and significant covariance between left digits 3 and 4 and right digit 4 significant. Generally, the right hand shows stronger dominance, with higher values and more of them significant. In Table 5, the left-right differences are shown more clearly. The differences between broad and narrow-sense heritabilities for each digit show higher values for the right digits.

The highly significant dominance variances for the greater counts of digit 2 (Table 3) disappear when the radial counts are examined, and further illustrate the perils of arbitrarily selecting data for their computational convenience. The "dominance" variance of the greater counts represents statistical artifact, because the greater count for digit 2 is composed of radial and ulnar counts, as mentioned. Using the greater counts from fingers not only results in a loss of information, as Jantz and Chopra (1983), Jantz *et al.* (1984), Roberts and Coope (1975), and Weninger (1964, 1983) have suggested, but also leads to misleading conclusions.

In terms of development, the dominance patterns probably reflect intrauterine effects, which act to make siblings more similar dermatoglyphically due to a common shared environment, as Cantor (1983) and Reed *et al.* (1979) detected in the children of MZ female twins. Dominance variance is estimated by greater sibling similarities or differences than would be expected given the values of the parents, but also through children that are more similar to one parent than the other. These different contributions to dominance variance cannot be separated, but the strongest dominance effects in the present analysis are at the periphery of the hand, on digits 1, 4, and 5, as Cantor (1983) and Reed *et al.* (1979) found. To get clarification of the sources of dominance variance, further ML estimations using the same experimental design as Cantor (1983) should be more informative. Whatever the source and extent of dominance, it has a highly significant influence on radial ridge counts. In a comparison of models with and without dominance effects, the likelihood ratio test with 55 degrees of freedom and $\Lambda = 120.28$ yields $p <$ .000001, meaning that the model with dominance effects produces a significantly better fit to the data. This high level of significance is all the more remarkable because every finger does not show significant dominance variance.

The values in the environmental variance-covariance matrix reveal yet another pattern. The variances are high and significant, and left and right digit 2 show the highest environmental variances by far. Left digits 2 and 3, left and right digit 4, and right digits 4 and 5 show low but significant ($p <.05$) covariance. Left and right digit 4 are the only homologous digits that show significant covariance. No covariances are significant at $p <$

.01. The rest of the covariances are much lower than values in the additive genetic and dominance matrices. The environmental matrix has no significant negative covariances, as was proposed by Loesch (1983) and Martin *et al.* (1982c) as the cause of lower heritabilities for individual fingers. The lack of negative covariances is consistent with the conclusion that the higher heritability for TRC is a statistical artifact.

The environmental variance-covariance matrix for radial counts (Table 6) indicates similar finger-specific tissue sensitivities of dermal and epidermal cells during development and significant local environmental variation. The environmental effects are very similar in magnitude for homologous fingers, yet no homologous fingers show significant covariance at $p < .01$. The local environmental variation is not merely hand specific, because fingers of the same hand also show little or no environmental covariation. The end result is an environmental response unique to each individual finger, as Cantor (1983) proposed, similar to digit-level sensitivities to environmental disturbances observed in the developing limb (Saunders 1948; Tschumi 1953). The pattern of different tissue sensitivities, recognizable in examining covariances, would not be seen in an analysis using correlations alone.

An examination of the components of variation for finger radial counts has revealed different patterns for the additive genetic, dominance, and environmental variance-covariance matrices. While the additive genetic variance-covariance matrix shows a high level of integration, the dominance and environmental variance-covariance matrices

reveal largely independent finger-specific effects. The dominance and environmental effects vary across fingers. For example, more dominance variances are significant for the right hand, and digit 2 has the highest environmental variances among fingers. The environmental covariances are consistently smaller than the dominance or additive genetic covariances. The covariances will be examined in further detail following a genetic analysis of palm and toe ridge counts.

Palms, Toes, and Overall Relationships

First, results from analyses of palm and toe ridge counts will be briefly reviewed, then results from analyses combining different dermatoglyphic areas. Table 8 shows the variance-covariance matrices for palm counts. Standard errors could not be calculated for all 6 variables; standard errors from mean counts were used for significance tests.

All figures in the additive genetic variance-covariance matrix for palm counts (Table 8) are significant at $p < .05$. As in finger radial counts, homologous palmar areas have higher covariances and the homologous midline traits are essentially the same genetic trait: The counts for Lbc and Rbc (Left and right bc counts) have a correlation of 1. The pattern of additive genetic covariances suggests only a mild "proximity effect' between Lbc and Lcd, while Lab and the right hand variables show roughly equivalent relationships between each of them and the other two palmar variables.

The dominance and environmental variance-covariance matrices (Table 8) reveal topological factors in the development of palm ridges absent for finger ridges. All counts

Table 8. Variance - Covariance Matrices for Palm Counts. Figures in bold are significant at $p < .05$. Lab - Rcd: Left palm ab ridge count through right palm cd count.

	Lcd	Lbc	Lab	Rab	Rbc	Rcd
Additive Genetic						
Lcd	**28.8**					
Lbc	**14.8**	**25.2**				
Lab	**11.7**	**11.3**	**22.6**			
Rab	**12.2**	**11.9**	**22.0**	**22.2**		
Rbc	**16.1**	**26.0**	**10.7**	**11.4**	**26.8**	
Rcd	**23.5**	**8.2**	**9.5**	**10.5**	**9.2**	**21.7**
Dominance Genetic						
Lcd	**4.5**					
Lbc	1.4	**3.7**				
Lab	0.9	0.4	2.0			
Rab	1.3	1.1	**3.2**	**4.5**		
Rbc	1.8	**2.5**	1.1	**2.2**	1.7	
Rcd	**5.3**	**3.1**	1.4	**2.4**	**3.6**	**5.1**
Environmental						
Lcd	**13.3**					
Lbc	**-1.4**	**8.8**				
Lab	1.3	-0.1	**11.7**			
Rab	0.7	-0.8	0.4	**9.1**		
Rbc	0.1	**1.8**	0.1	**-1.8**	**10.0**	
Rcd	**2.3**	**-1.4**	1.3	0.6	**-3.1**	**13.7**
Phenotypic						
Lcd	**46.5**					
Lbc	**14.8**	**37.7**				
Lab	**14.0**	**11.6**	**36.3**			
Rab	**14.2**	**12.2**	**25.7**	**35.8**		
Rbc	**18.1**	**30.3**	**11.9**	**11.7**	**38.6**	
Rcd	**31.1**	**9.9**	**12.2**	**13.4**	**9.7**	**40.6**

except Lab and Rbc show significant dominance variance, and the right palmar areas show greater dominance effects, as in fingers. In contrast to finger radial counts, all homologous palmar areas show significant and high dominance covariance. All right hand palm counts covary significantly even though Rbc shows no significant dominance variance. The environmental variances are highest in the Lcd, Rab, and Rcd areas, none of which are adjacent to each other. Additionally, adjacent areas except for Lab-Lbc show significant negative environmental covariances, especially on the right hand, and probably reflect competition between adjacent areas for ridges during formation. Palm count areas abut each other physically without separations, unlike fingers. A greater number of ridges between two palmar triradii implies less room and less ridges for an adjacent area. Thus, palm ridges are more influenced by topological factors than finger ridges.

Table 9 shows the heritability matrices for the palm counts. As in fingers, the off-diagonals are not zeros, meaning that the additive genetic variance-covariance matrix is not proportional to the phenotypic variance-covariance matrix. The mean multivariate narrow-sense heritability is .35, and the mean broad-sense heritability is .39. The narrow-sense heritability is slightly higher than the mean multivariate heritability of finger radial ridge counts (.31), in contrast to earlier conclusions that the heritability of finger ridges was higher than that of palm ridges (Holt 1968; Loesch 1983). Dominance in the palms seems to have a smaller overall effect than in the fingers, but its relative effects in certain palmar areas are greater than in other areas, as seen in the broad minus narrow figures. Three nonadjacent areas, Lbc, Rab, and Rcd, show greater relative dominance effects. The

Table 9. $\mathbf{P}^{-1}\mathbf{G}$ Matrices for Palm Counts. Abbreviations are the same as in Table 8

	Lcd	Lbc	Lab	Rab	Rbc	Rcd
Narrow						
Lcd	0.39	0.08	0.04	0.03	0.11	0.28
Lbc	0.09	0.33	0.08	0.09	0.35	-0.01
Lab	0.02	0.02	0.37	0.32	0.00	0.01
Rab	0.03	0.08	0.32	0.35	0.05	0.06
Rbc	0.09	0.37	-0.01	0.01	0.37	0.02
Rcd	0.22	-0.06	-0.03	0.00	-0.04	0.29
Broad						
Lcd	0.41	0.02	0.02	-0.04	0.05	0.32
Lbc	0.08	0.44	0.03	0.01	0.41	-0.00
Lab	-0.01	-0.02	0.35	0.32	-0.04	-0.04
Rab	0.02	0.09	0.43	0.47	0.11	0.09
Rbc	0.12	0.35	0.04	0.10	0.36	0.07
Rcd	0.34	0.05	-0.01	0.07	0.07	0.38
Broad -Narrow Heritabilities						
	0.02	0.12	-0.02	0.12	-0.01	0.09

other palmar areas show little or no dominance variance relative to the additive genetic and environmental variances.

The variance-covariance matrices for the tibial ridge counts from toes are shown in Table 10. Standard errors could not be calculated for individual toes, and are based on an analysis of mean left plus right values. Left toe 4 was excluded because it showed no dominance variance. There are strong similarities between the toe and finger matrices, including a high level of integration and the proximity effect: Homologous toes show the highest correlations, then adjacent toes, then toes adjacent to homologous toes. Additionally, as in finger radial counts and palm counts, homologous counts at the midline, in this case toe 3, are essentially the same genetic trait, with a correlation of 1.

As with palms and fingers, there are several nonhomologous dominance and environmental covariances that are significant. Left toe 1 shows significant dominance covariance with right toes 1-4. There are also differences in the toe matrices compared to the finger matrices. Toes 3 and 4 show very high additive genetic and environmental variance compared to the other toes, and left toe 5 shows the lowest variance, due to a very low mean ridge count. Additionally, one toe environmental covariance, between right toes 1 and 3, is negative and significant.

The $\mathbf{P}^{-1}\mathbf{G}$ matrices for toes are shown in Table 11. As in the radial counts and palm count heritability matrices (Tables 5 and 9), the off-diagonals are not zeros, and in several

Table 10. Variance - Covariance Matrices for Toe Tibial Counts. Figures in bold are significant at $p < .05$. LTC1 - RTC5: Left tibial ridge count for toe 1 through right tibial ridge count for toe 5.

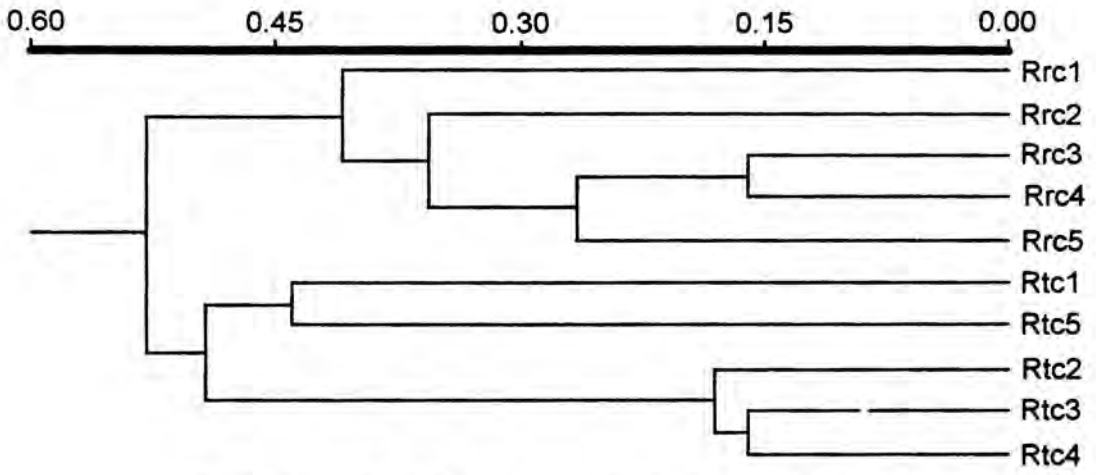
	LTC1	LTC2	LTC3	LTC5	RTC1	RTC2	RTC3	RTC4	RTC5
Additive Genetic									
LTC1	**37.6**								
LTC2	**24.9**	**43.5**							
LTC3	**24.6**	**43.8**	**67.6**						
LTC5	**10.2**	**10.9**	**10.2**	**7.7**					
RTC1	**34.3**	**20.0**	**19.5**	**9.1**	**33.1**				
RTC2	**24.0**	**40.5**	**42.2**	**11.0**	**19.0**	**40.7**			
RTC3	**22.9**	**41.9**	**64.1**	**10.2**	**18.0**	**41.7**	**61.3**		
RTC4	**22.4**	**40.4**	**56.0**	**14.9**	**19.3**	**41.0**	**53.2**	**62.8**	
RTC5	**12.5**	**12.0**	**10.9**	**8.7**	**11.2**	**12.5**	**11.5**	**17.2**	**11.6**
Dominance Genetic									
LTC1	**13.0**								
LTC2	1.2	**5.3**							
LTC3	0.2	0.1	**9.4**						
LTC5	-0.5	0.5	3.1	**6.4**					
RTC1	**11.4**	2.2	2.2	-1.1	**13.0**				
RTC2	1.2	**4.4**	-0.8	0.3	**3.3**	3.7			
RTC3	-0.1	1.3	**4.6**	-0.2	**4.4**	-0.8	**8.5**		
RTC4	1.8	1.9	**4.5**	-0.8	**4.1**	-1.1	**7.5**	**7.9**	
RTC5	-0.1	0.1	2.8	**7.0**	-0.6	1.5	1.3	1.8	**9.0**
Environmental									
LTC1	**12.6**								
LTC2	1.5	**15.7**							
LTC3	0.1	**3.4**	**21.5**						
LTC5	0.6	0.8	-1.4	**6.8**					
RTC1	0.7	1.1	-1.1	1.1	**6.4**				
RTC2	0.7	**4.1**	1.0	1.2	0.9	**14.3**			
RTC3	0.4	0.6	**5.0**	1.2	**-2.5**	0.8	**20.5**		
RTC4	0.6	0.4	-0.9	**2.9**	-1.2	1.7	-0.9	**24.7**	
RTC5	0.1	0.4	-1.9	0.8	0.7	0.3	-0.4	0.4	**5.1**

Table 11. $\mathbf{P^{-1}G}$ Matrices for Toe Tibial Counts. Abbreviations are the same as in Table 10.

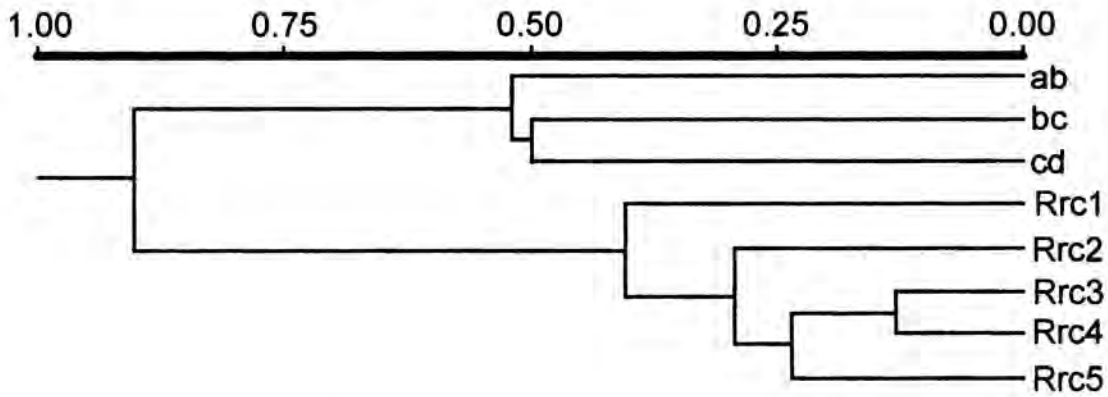
	LTC1	LTC2	LTC3	LTC5	RTC1	RTC2	RTC3	RTC4	RTC5
Narrow Sense									
LTC1	0.27	0.11	0.09	0.04	0.19	0.12	0.08	0.01	0.05
LTC2	0.06	0.30	0.05	0.03	0.04	0.16	0.02	0.02	0.02
LTC3	0.05	0.08	0.30	-0.01	0.04	0.05	0.28	0.17	-0.03
LTC5	0.04	0.01	-0.05	0.14	0.05	0.01	-0.06	0.05	0.06
RTC1	0.32	-0.05	-0.07	0.04	0.41	-0.08	-0.09	-0.03	0.05
RTC2	0.05	0.25	0.18	0.03	-0.01	0.36	0.24	0.21	0.03
RTC3	0.02	0.08	0.27	-0.01	0.00	0.10	0.26	0.08	0.00
RTC4	-0.04	0.06	0.14	0.07	-0.01	0.08	0.12	0.38	0.08
RTC5	0.13	0.03	-0.06	0.14	0.11	0.04	0.01	0.13	0.29
Broad Sense									
LTC1	0.44	0.06	-0.01	0.03	0.22	0.03	-0.11	-0.08	0.03
LTC2	0.02	0.41	0.01	0.03	-0.03	0.27	0.06	0.09	-0.04
LTC3	0.03	0.01	0.48	0.09	-0.02	0.02	0.23	0.13	0.04
LTC5	-0.03	0.03	0.07	0.37	-0.07	-0.07	-0.18	-0.17	0.22
RTC1	0.45	0.00	0.04	-0.03	0.67	0.04	0.15	0.12	-0.01
RTC2	0.02	0.29	0.06	-0.01	0.00	0.39	0.06	-0.00	0.03
RTC3	-0.01	0.08	0.23	-0.05	0.06	0.07	0.40	0.19	-0.01
RTC4	-0.01	0.06	0.14	-0.02	0.02	0.03	0.19	0.46	0.02
RTC5	0.06	-0.03	-0.00	0.37	0.03	0.14	0.09	0.29	0.63
Broad - Narrow Heritabilities									
	0.17	0.10	0.18	0.22	0.27	0.04	0.14	0.09	0.34

cases are as high as or higher than the diagonals. There is a cluster of nonhomologous nonzero values involving left digit 3 and right digits 2 and 4. The mean narrow-sense heritability is .29 and the mean broad sense heritability is .46, close to the heritabilities of the finger and palm ridge counts. As in the finger and palm counts, dominance appears to have a stronger effect on the right side tibial counts based on the broad minus narrow heritability estimates, especially for toes 1, 3, and 5.

Ideally, the overall relationships between finger, palm, and toe ridge counts would be estimated in an analysis of all variables simultaneously. Unfortunately, a ML analysis of finger, palm, and toe counts was not successful. However, finger and toe counts were compared together, as were finger and palm counts. Relationships among right side variables using correlations are shown in Figure 7. Fingers and toes are genetically distinct (Figure 7a), as shown by their division into finger and toe variables. Further, the relationships within the hand are different from those within the foot: Fingers 1 and 2 are separated genetically from each other and the other more ulnar fingers, but toes 1 and 5 are separated genetically from the toes in the middle. In Figure 7b, palm counts are quite distinct from fingers genetically. Additionally, palms showed no relationship to toes. Therefore, finger, palm, and toe ridge counts apparently represent largely independent epidermal systems genetically. Their genetic independence is a likely reason why population comparisons using palm ridge counts and finger ridge counts separately produce different population relationships (Jantz and Chopra 1983; Jantz *et al.* 1992; Yokota 1997).



a. Right Finger Radial Counts and Right Toe Tibial Counts.



b. Right Finger Radial Counts and Right Palm Counts.

Figure 7. Cluster Analysis of Genetic Correlations Among Different Epidermal Ridge Areas. Numbers represent the scale of genetic distances. RRC1 - RTC5: Right digit 1 radial ridge count through right toe 5 tibial ridge count. Rab- RRC5: Right palmar ab ridge count through right digit 5 radial ridge count.

Examination of the genetic components of variation for ridge systems has revealed differences within and among fingers, palms, and toes. Fingers, palms, and toes apparently represent separate genetic epidermal systems. The palmar dominance and environmental variance-covariance matrices reveal topological influences on palmar ridge development, because palmar areas are physically adjacent throughout development, unlike fingers and toes. The relationships among toe ridges are different from relationships among finger ridges. However, finger, palm and toe ridge counts show an integrated additive genetic variance-covariance matrix structure. Also, as in fingers, both palms and toes produce $\mathbf{P}^{-1}\mathbf{G}$ matrices with variable diagonals (heritabilities) and non-zero off-diagonals, meaning that minimally, the additive genetic variance-covariance matrix is not proportional to the phenotypic variance-covariance matrix. The relationships among and within variance-covariance matrices will now be explored to further elucidate the causes and consequences of nonproportionality.

Comparisons of Variance-Covariance Matrices

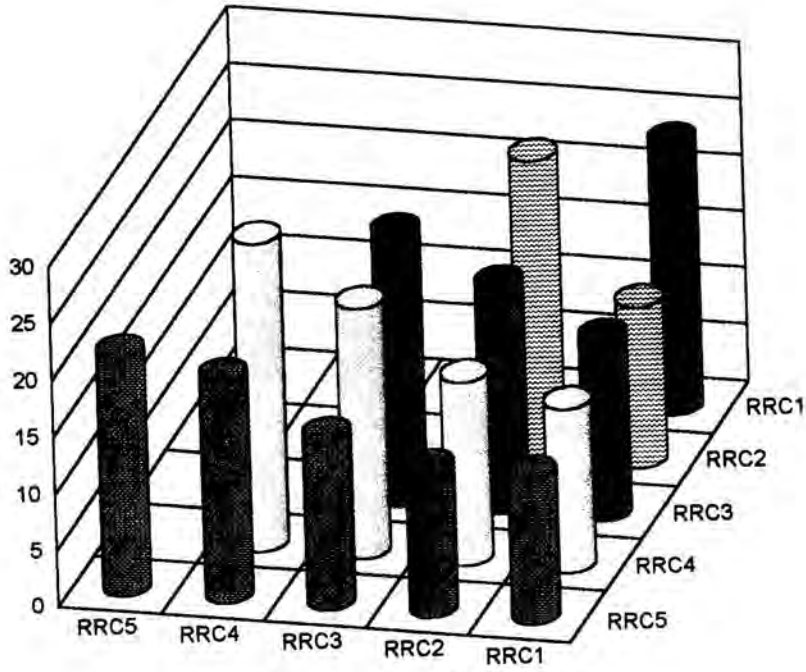
Comparisons among the additive genetic, dominance, environmental, and phenotypic variance-covariance matrices for finger radial counts reveals more about the components of variation within and among them. The phenotypic variance-covariance matrix and elementwise comparisons between the additive genetic and phenotypic variance-covariance matrices are shown in Table 12. The ratio of additive genetic to phenotypic matrix elements provides heritabilities as if all variances and covariances were independent. The additive genetic variances (diagonal elements) are on average 64% of

Table 12. Phenotypic Variance-Covariance Matrix and Additive Genetic Comparisons.
LRC1-RRC5:Left radial ridge count for digit 1 through right radial ridge count for digit 5

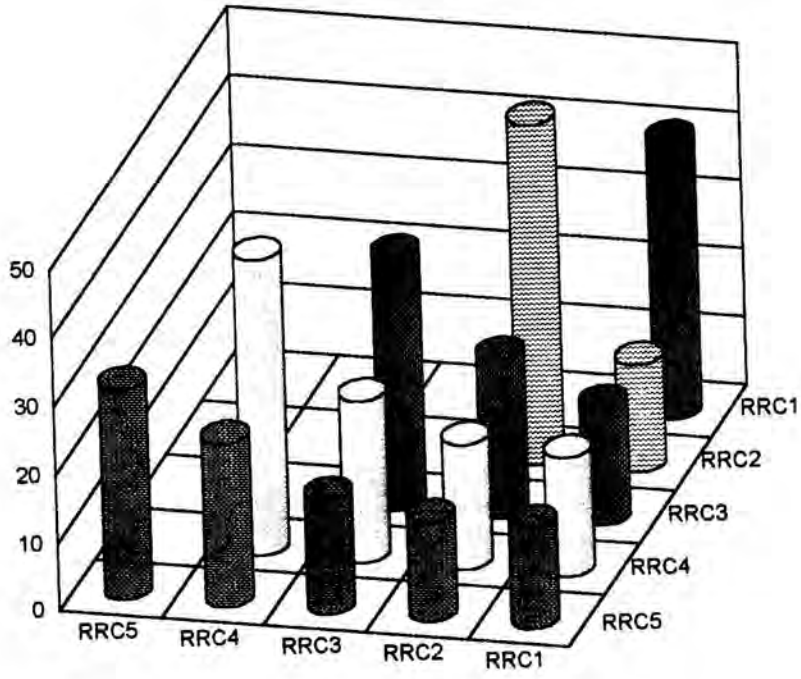
	LRC1	LRC2	LRC3	LRC4	LRC5	RRC1	RRC2	RRC3	RRC4	RRC5
Phenotypic Variance-Covariance Matrix										
LRC1	40.5									
LRC2	17.8	49.5								
LRC3	20.0	26.4	43.7							
LRC4	18.0	20.7	27.8	41.7						
LRC5	15.5	16.8	19.3	23.0	28.9					
RRC1	29.6	16.6	18.0	17.1	14.8	40.4				
RRC2	16.8	28.8	23.5	17.3	14.5	15.5	49.7			
RRC3	18.3	23.4	29.0	23.5	16.5	16.9	22.9	36.8		
RRC4	17.4	20.5	26.9	33.7	22.9	17.5	18.3	23.5	43.1	
RRC5	14.8	17.3	19.0	22.7	23.7	15.0	14.7	16.9	24.0	30.8
Elementwise Ratio: Additive Genetic/Phenotypic Covariances										
LRC1	0.70									
LRC2	0.92	0.56								
LRC3	0.97	0.90	0.67							
LRC4	0.90	0.90	0.87	0.65						
LRC5	0.93	0.95	0.93	0.86	0.70					
RRC1	0.83	0.90	0.96	0.82	0.86	0.59				
RRC2	0.93	0.80	0.89	0.85	0.98	0.91	0.53			
RRC3	0.93	0.86	0.91	0.91	0.93	0.92	0.86	0.65		
RRC4	0.93	0.90	0.90	0.78	0.85	0.82	0.88	0.93	0.63	
RRC5	0.92	0.89	0.91	0.83	0.85	0.86	0.87	0.89	0.82	0.68
Elementwise Subtraction: Phenotypic - Additive Genetic Covariances										
LRC1	12.1									
LRC2	1.5	21.8								
LRC3	0.6	2.6	14.5							
LRC4	1.9	2.1	3.5	14.7						
LRC5	1.1	0.8	1.4	3.3	8.7					
RRC1	5.0	1.7	0.8	3.0	2.1	16.5				
RRC2	1.2	5.6	2.6	2.5	0.2	1.5	23.2			
RRC3	1.3	3.2	2.8	2.2	1.2	1.3	3.2	13.0		
RRC4	1.2	2.0	2.8	7.5	3.4	3.1	2.2	1.6	16.1	
RRC5	1.2	1.9	1.6	3.8	3.5	2.0	1.9	1.8	4.4	9.8

the phenotypic variances, while the covariances (off diagonals) are 89% of the phenotypic variances. The difference in proportions imply that the covariances of variables are generally more "heritable" than the variances. In other words, the dominance and environmental variances, more so than their covariances, have more impact on the phenotypic variance-covariance matrix. The elementwise differences (Table 12) between the two matrices also highlight greater disparities in variances rather than covariances. These important discrepancies between variances and covariances would be obscured in an analysis of correlations, where all diagonals are 1. Figure 8 presents the different relationships within each matrix for the right hand graphically. As can be seen, the phenotypic variances are greater relative to the phenotypic covariances, and therefore the genetic integration of ridges is higher than the phenotypic integration of ridges. Also, the genetic variances of digits 1,2, and 4 are very similar, but the phenotypic variance of digit 2 is much higher, due to very high environmental variance (Table 6).

The matrix comparisons in Table 12 are not multivariate tests, however, and more rigorous comparisons are required to assess overall matrix similarity. PCA was performed on all variance-covariance matrices and the results are presented numerically in Table 13 and graphically in Figure 9. The additive genetic and phenotypic matrix plots (Figures 9a and 9d) show many similarities. In both plots, variables have positive and similar eigenvalues for PC1. For the additive genetic variation, PC1 is by far the most important component, explaining 74% of the total additive genetic variation. Digit 3 has the highest values for PC1, and scores taper off as one moves in a radial or ulnar direction. Left and



a. Genetic

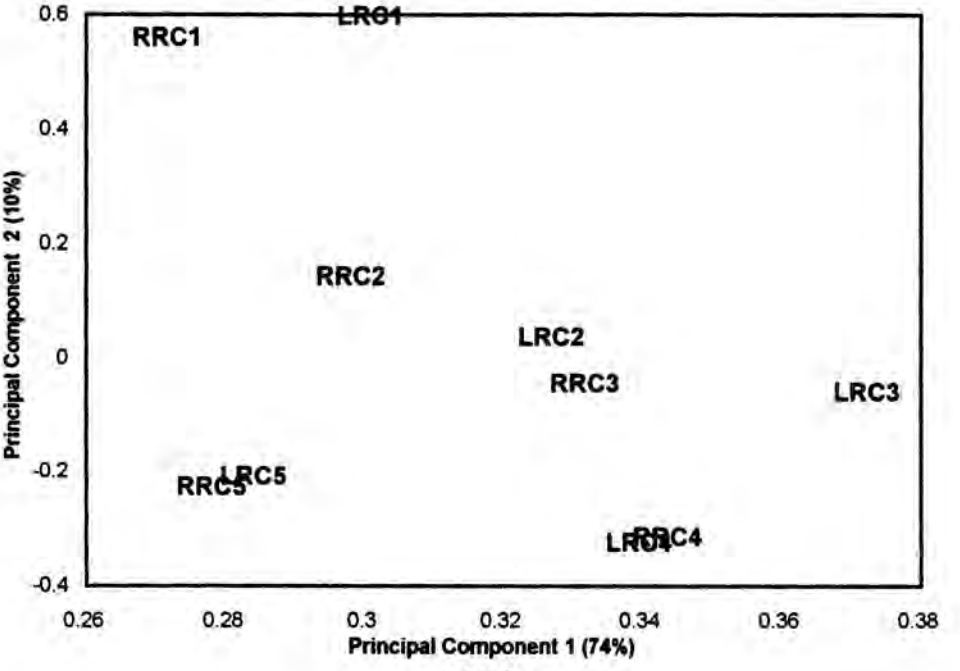


b. Phenotypic

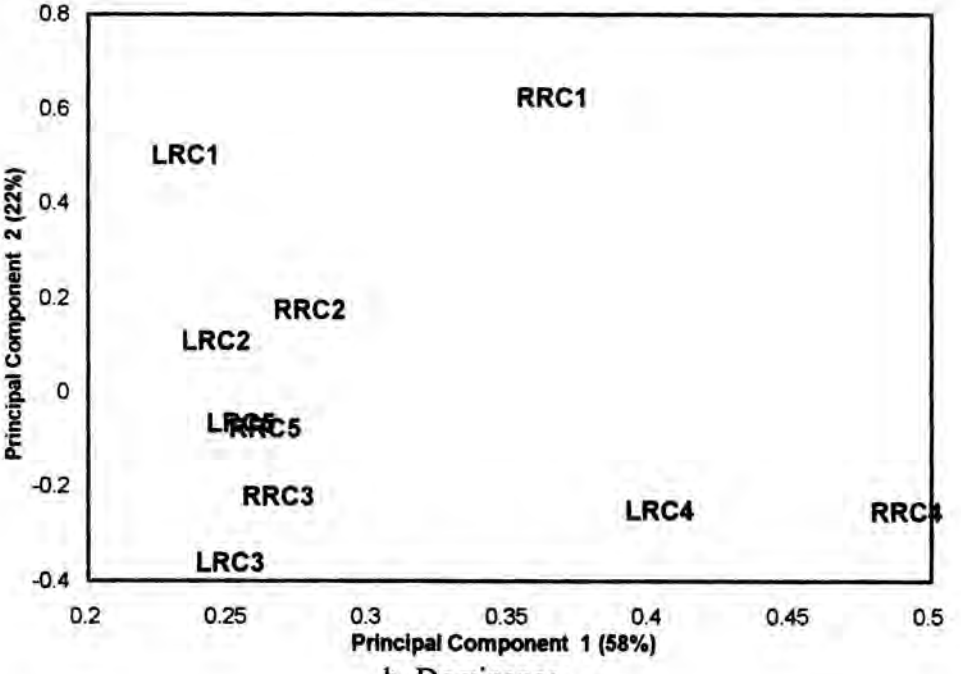
Figure 8. Relationships Within the Finger Radial Count Genetic and Phenotypic Variance-Covariance Matrices. The diagonal bars represent the variances and the off-diagonal bars represent the covariances.

Table 13. Principal Components of Finger Radial Count Variance-Covariance Matrices. Abbreviations are the same as in Table 12.

		LRC1	LRC2	LRC3	LRC4	LRC5	RRC1	RRC2	RRC3	RRC4	RRC5
	Variance explained (%)										
Additive Genetic											
PC1	74%	0.301	0.327	0.372	0.340	0.284	0.272	0.298	0.332	0.344	0.278
PC2	10%	0.596	0.035	-0.063	-0.329	-0.212	0.557	0.141	-0.048	-0.318	-0.230
PC3	8%	0.297	-0.469	-0.177	0.216	0.243	0.269	-0.570	-0.186	0.185	0.298
PC4	4%	-0.044	0.248	-0.361	-0.257	0.462	-0.010	0.296	-0.385	-0.212	0.496
PC5	2%	-0.057	-0.734	-0.085	-0.177	0.083	0.010	0.562	0.273	0.135	0.065
Dominance Genetic											
PC1	58%	0.235	0.246	0.251	0.404	0.255	0.365	0.279	0.268	0.492	0.263
PC2	22%	0.499	0.107	-0.365	-0.254	-0.069	0.621	0.173	-0.224	-0.256	-0.080
PC3	12%	-0.152	0.492	0.048	-0.295	-0.354	-0.048	0.326	0.501	0.019	-0.400
PC4	11%	0.055	-0.051	-0.251	-0.119	0.305	-0.129	0.067	0.591	-0.460	0.492
PC5	9%	0.019	-0.460	0.408	0.152	0.126	0.355	-0.130	0.349	-0.304	-0.474
Environmental											
PC1	22%	-0.029	0.609	0.245	0.074	0.025	-0.065	0.734	0.086	-0.090	0.058
PC2	16%	0.071	0.664	0.276	-0.034	0.038	0.175	-0.648	0.029	-0.110	0.107
PC3	13%	0.062	0.043	-0.115	0.630	0.179	0.329	0.011	0.060	0.608	0.264
PC4	11%	0.364	-0.390	0.804	-0.014	-0.023	0.154	0.058	0.110	-0.006	0.170
PC5	9%	0.706	0.100	-0.255	0.053	-0.204	-0.283	-0.034	0.519	0.054	-0.170
Phenotypic											
PC1	56%	0.287	0.337	0.362	0.349	0.272	0.276	0.313	0.323	0.352	0.276
PC2	11%	-0.006	-0.474	-0.040	0.349	0.255	0.028	-0.615	-0.094	0.360	0.265
PC3	10%	0.630	-0.160	-0.145	-0.196	-0.067	0.660	-0.129	-0.095	-0.211	-0.089
PC4	6%	-0.049	0.202	-0.557	-0.084	0.387	0.035	0.265	-0.451	0.005	0.454
PC5	5%	-0.013	-0.747	-0.055	0.036	-0.013	0.002	0.645	0.083	0.118	-0.014

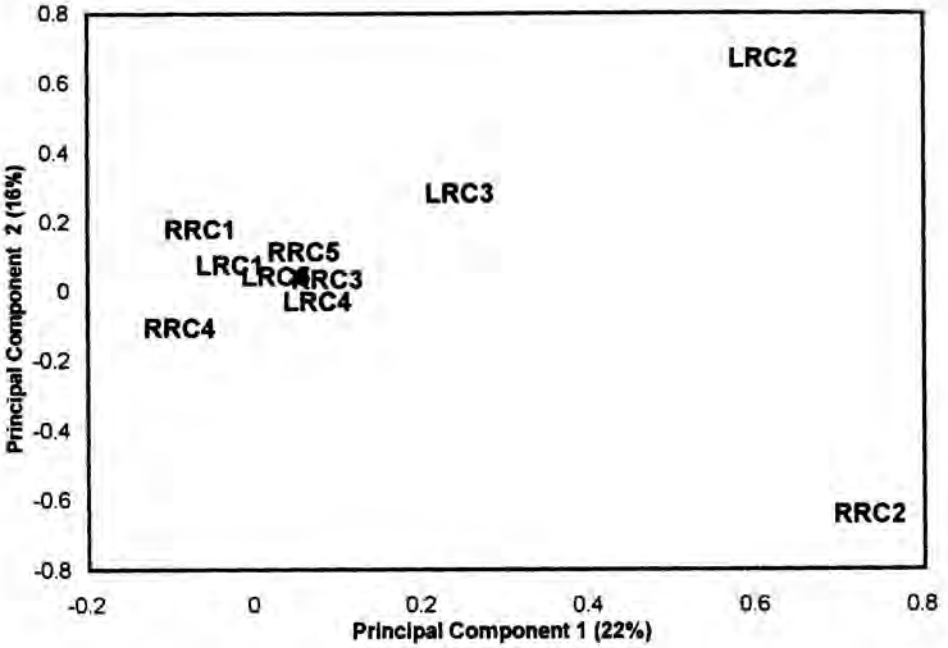


a. Additive

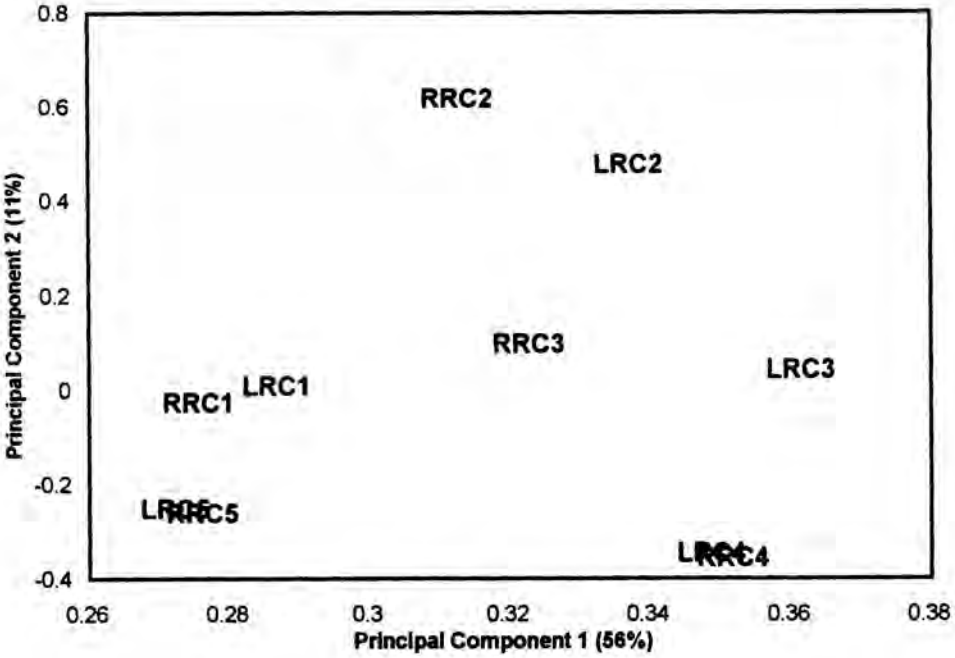


b. Dominance

Figure 9. Principal Components of Variance-Covariance Matrices for Radial Counts.



c. Environmental



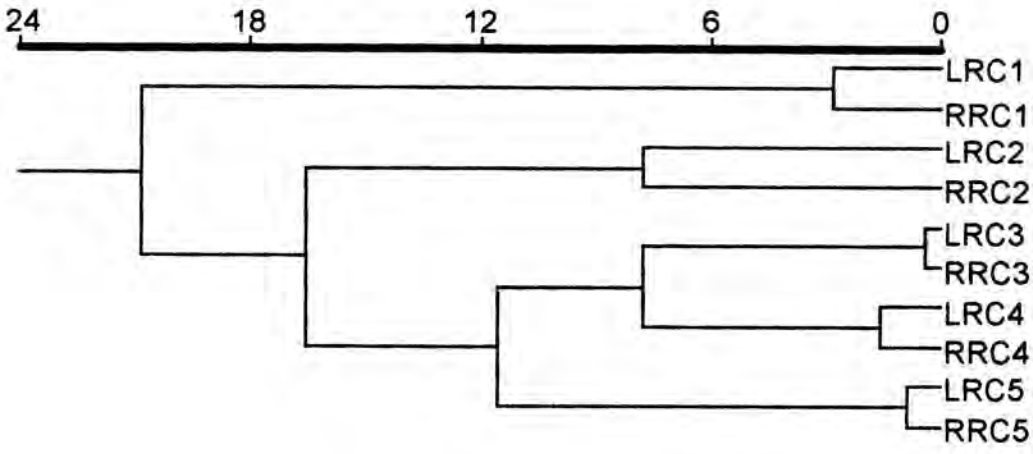
d. Phenotypic

Figure 9. (continued)

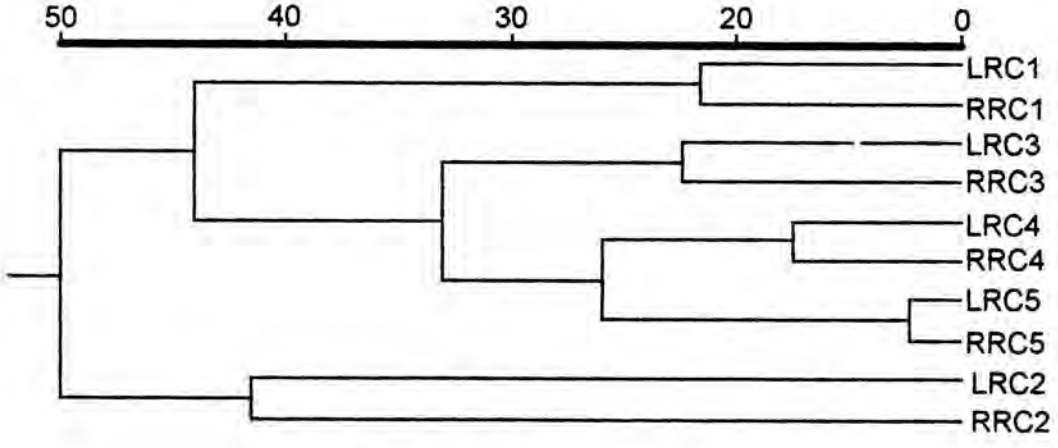
right digits 4 and 5 contribute equally to component scores, but digits 1,2, and 3 show asymmetry, with the right digits showing lower scores than the left for PC1. Greater differences between the additive genetic and phenotypic matrices are in PC2. Digits 3,4 and 5 have similar scores for each PC2, but digit 2 and especially digit 1 show higher additive genetic PC2 scores. The fingers show a radioulnar gradient for additive genetic PC2, with high scores for digit 1 and progressively lower scores for each finger as one moves in an ulnar direction. Digit 1 seems to be the most independent finger genetically, while digit 2 is probably the most independent finger phenotypically.

The strongest dominance effects (Figure 9b) shown in PC1 are on digit 4, especially right digit 4, which shows the highest dominance variance in Table 6. In fact, all of the right digits indicate stronger dominance effects than the left, especially for digits 4 and 1. PC2 separates digits 1 and 2, with positive scores, from digits 3,4, and 5 with negative scores. The PCA of the environmental matrix (Figure 9c) shows substantial and differing environmental effects for left and right digit 2. The other values cluster near the 0,0 point. The environmental effects are more difficult to asses visually because the effects are not as strong as the other matrices, with the first two principal components accounting for only 38% of the total environmental variance.

A cluster analysis was also performed on the additive genetic and phenotypic variance-covariance and correlation matrices of finger radial counts to visually assess similarities (Figure 10). The covariances were converted to distances and the correlations

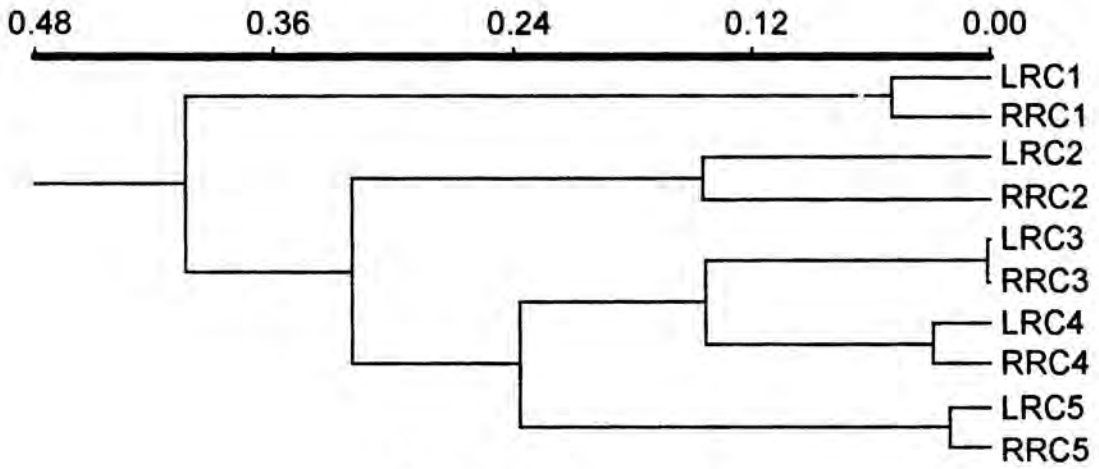


a. Additive Genetic Covariances

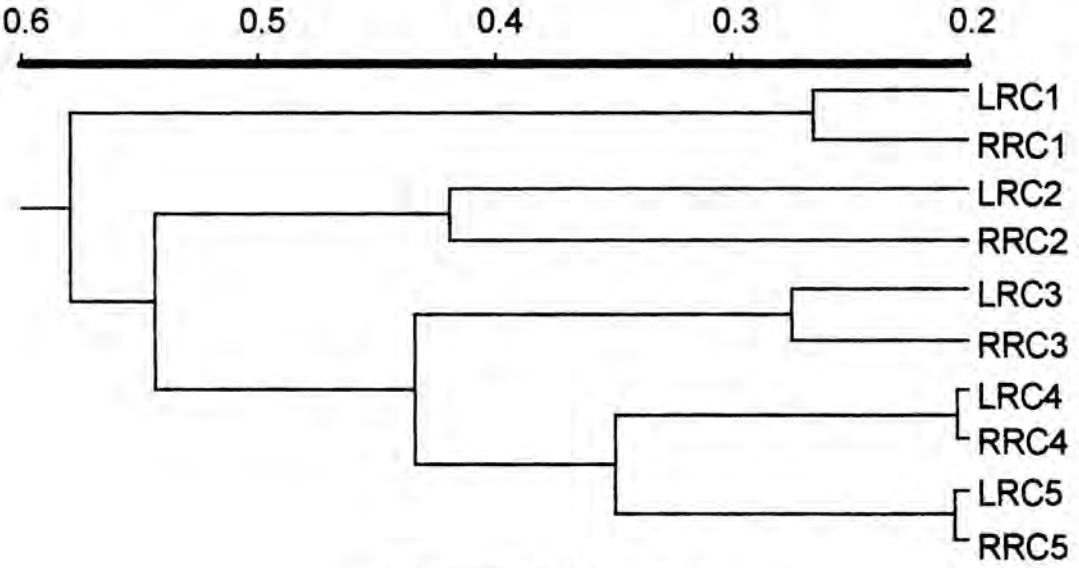


b. Phenotypic Covariances

Figure 10. Cluster analysis of the Additive Genetic and Phenotypic Variance-Covariance and Correlation Matrices. Numbers refer to genetic or phenotypic distances between variables. LGC1 - RGC5: Left Greater Ridge Count for digit 1 through Right Greater Ridge Count for digit 5.



c. Additive Genetic Correlations



d. Phenotypic Correlations

Figure 10. (continued)

were changed to (1-correlation). The additive genetic covariance relationships confirm that digit 1 is genetically isolated from the rest of the hand. Digit 5 is also relatively isolated, and digits 3 and 4 are the most genetically similar digits. Homologous digits cluster together, especially digit 3, but left and right digit 2 are the most disparate. The phenotypic clustering presents a different pattern. Digit 2 is a phenotypically outlying digit, and left and right digit 2 are even more distinct from each other, showing greater differences between them than among digits 3,4, and 5. On the whole, there are greater homologous phenotypic differences, and digit 5 shows the greatest homologous similarity. The cluster analysis indicates that the phenotypic relationships among variables differ from the additive genetic relationships between variables. The differences between the additive genetic and phenotypic variance-covariance matrices must be due to different dominance and/or environmental effects. The cluster analysis based on correlations points to the same conclusion.

Visual comparisons have illuminated aspects of the relationships between variables but can only confirm that the genetic and phenotypic variance-covariance matrices are different. More rigorous comparisons between matrices are needed to estimate the amount of disagreement and calculate similarity probabilities. Distance matrices among variables, calculated from variance-covariance matrices, were compared using procedures used by several researchers (Cheverud 1988; Dietz 1983; Kohn and Atchley 1988). The pairwise variance-covariance matrix comparison statistics are shown in Table 14, and for these statistics, the *p* values are the most important. Based on the *p* value of the K_c statistic, the

Table 14. Comparison Statistics for Variance-Covariance Matrices.

Matrix Comparison	Matrix Correlation (p)	Mantel's Z (p)	Spearman's R (p)	Kendall's K_c (p)
Additive and Phenotypic	.88 (.0001)	29773.1 (.0001)	30144 (.0001)	240 (.0001)
Dominance and Phenotypic	.22 (.103)	6963.34 (.098)	24947.5 (.202)	67 (.043)
Environmental and Phenotypic	.72 (.0002)	41648.1 (.0001)	29288.5 (.0003)	174 (.0003)

additive genetic and environmental matrices have highly significant similarities to the phenotypic variance-covariance matrix. These results contradict conclusions based on the $\mathbf{P}^{-1}\mathbf{G}$ matrix, which showed differing heritabilities and off-diagonals greater than zero. These matrix comparison procedures, however, exclusively utilize the off-diagonals in comparisons because they are frequently used for correlation or distance matrices, which have diagonals equal to either 1 or 0. As has been pointed out, matrix correlations (especially the Mantel test) represent overall tendencies in matrices rather than detailed comparisons (Dietz 1983). Unfortunately, comparisons of the diagonals and off-diagonals are not directly testable, because the variances are essentially independent vectors while the off-diagonals, as part of a matrix, should be treated differently (Dietz 1983). Nevertheless, several statistics were calculated in comparisons of the diagonal elements. The pairwise Pearson's r was .82 ($p < .01$), Spearman's coefficient was .72 ($p < .02$), and Kendall's tau b was .60 ($p < .02$) for the diagonals of the additive genetic and phenotypic

matrices. The lower significance figures for the diagonals can only suggest that the additive and phenotypic diagonals (variances) are not as similar as the off-diagonals (covariances) are. The results of comparing diagonals and the proportional differences between additive and phenotypic variances and covariances (Table 12) may illustrate the misleading nature of some matrix comparison procedures. Matrix comparison statistics that test the hypothesis of no association between matrices may have low power to detect important differences between phenotypic, genetic, linguistic, and other matrices employed in anthropological or genetic analyses.

There are other strong indications that the additive genetic variance-covariance matrix is more highly integrated than the phenotypic variance-covariance matrix, as suggested by Figure 8. The mean correlation of the additive genetic co relation matrix is .71 ± .020 (s.e.), higher than the mean phenotypic matrix correlation of .51 ± .018. Also, the additive genetic PC1 represents a higher percentage of the variance than the phenotypic PC1 (Table 13), reflecting higher intercorrelations among variables in a multivariate sense (Golub 1989). The $\mathbf{P}^{-1}\mathbf{G}$ matrix and the fact of greater additive genetic than phenotypic integration indicate that it is likely that the phenotypic variance-covariance matrix for ridge counts is not proportional to the additive variance-covariance matrix, despite matrix comparison statistics to the contrary. However, the implications of this unknown level of apparent nonproportionality for population comparisons are unknown, and a practical comparison of population relationships using each kind of data is needed.

Population Comparisons

A comparison of population relationships based on additive genetic and phenotypic values will illustrate the consequences of the violation of the assumption of proportionality for the additive genetic and phenotypic matrices. The phenotypic data for ten finger radial ridge counts were converted to additive genetic values based on the principal components of the additive genetic variance-covariance matrix. Only seven positive principal components could be extracted, as Rostron (1977) also found, and the tenth eigenvalue was negative. Therefore, the matrix is not positive-definite, and is in fact indefinite (Ortega 1987). Positive-definite matrices have variances that are much higher than covariances (Golub 1989), and therefore show a lower level of integration than indefinite matrices. A reduced number of axes were extracted from the additive genetic variance-covariance matrix, but little information was lost: The seven principal components represent 99.92% of the total variation present in all 10 variables. Additive genetic and phenotypic data from the population samples were analyzed using matrix comparisons, PCA, and canonical discrimination.

An RMET analysis (Relethford and Blangero 1990) was performed on the phenotypic and genetic values for finger radial ridge counts. Table 15 shows the results of comparing the population additive genetic and phenotypic distance matrices for the radial ridge counts of the fingers. All statistics indicate a highly significant relationship between the additive and phenotypic distance matrices, with no indications that there may be important differences between them.

Table 15. Additive Genetic and Phenotypic Population Distance Matrix Comparisons.

Matrix Correlation (p)	Mantel's Z (p)	Spearman's R (p)	Kendall's K_c (p)
.88 (.002)	.361 (.002)	1215 (.002)	40 (.003)

An examination of the population distances and their standard errors (Table 16) shows discrepancies that arise when populations relationships are estimated based on the phenotypic, rather than the additive genetic variance-covariance matrix. Although the genetic distances are generally greater than phenotypic distances, out of 15 unique distances, there are only 3 significant genetic distances, but 11 significant phenotypic distances at $p < .05$. The difference in the number of significant distances is not due to relatively greater among-group variation compared to within-group variation of the phenotypic data. The mean within-group variance is 1.022 for additive genetic and 1.012 for phenotypic data. The phenotypic F_{ST} is 0.0034 and the genetic F_{ST} is 0.0033, so the relative amount of within-group variation is virtually the same in each data set.

The difference in significances of genetic and phenotypic distances is due to differences in the standard errors, which are proportionally smaller for the phenotypic data. The smaller standard errors are apparently due to the relatively inflated variances compared to covariances in the phenotypic data. As mentioned, the inflated variances are due to greater finger-specific dominance and environmental effects. As a result, the

Table 16. Additive Genetic and Phenotypic Population Distance Matrices and Standard Errors (s.e.). Values are multiplied by 1000. Values shown in bold are significant at $p < .05$. EAST FRIE: East Friesland.

	BERLIN	FREIBURG	HUNGARY	KIEL	EAST FRIE
Genetic Distances (s.e.)					
FREIBURG	7.6 (4.2)				
HUNGARY	**8.0** (3.7)	4.5 (3.6)			
KIEL	5.2 (3.1)	**12.6** (6.2)	6.9 (4.2)		
EAST FRIE	8.3 (5.2)	17.1 (8.8)	12.0 (6.9)	9.2 (6.1)	
VIENNA	1.7 (1.2)	3.9 (2.8)	4.8 (2.7)	5.6 (3.0)	**11.4** (5.9)
Phenotypic Distances (s.e.)					
FREIBURG	**6.8** (3.3)				
HUNGARY	**5.7** (2.6)	4.2 (3.0)			
KIEL	**6.5** (2.9)	**13.0** (5.3)	**7.5** (3.6)		
EAST FRIE	8.9 (4.5)	**15.3** (6.9)	**12.1** (5.8)	**15.9** (6.7)	
VIENNA	1.3 (0.8)	3.9 (2.4)	3.5 (1.9)	**6.2** (2.6)	**11.0** (4.8)

phenotypic values are less integrated (more independent), with lower intercorrelations. Lower correlations among variables increases the significance of differences in variable means among groups in a multivariate analysis because the variables are less interdependent. Higher correlations among variables (showing greater integration) reduce the significance of differences in group means because the distance statistics (such as Mahalanobis D^2) compensate for intercorrelations among variables (Tatsuoka 1988). The relatively higher genetic correlations, showing a greater level of integration, produce relatively higher standard errors for a given distance between populations. Population distances based on dermatoglyphic phenotypes are likely to show higher significance levels than the underlying genetic values because of finger-specific dominance and environmental effects.

Other important considerations in the genetic and phenotypic relationships between populations are any differences in population relationships using each kind of data. A closer look at the distance matrices (Table 16) indicates that the population samples may show different genetic and phenotypic relationships. The results of the PCA of additive genetic and phenotypic values are shown in Figure 11. The percentage of total variance explained by each axis is 52% and 23% of the total genetic variance, and 45% and 33% of the total phenotypic variance for axes 1 and 2, respectively. The genetic relationships between all samples are fairly close except for East Friesland, which is a clear outlier. Freiburg and Hungary are less distant from the remaining groups. The phenotypic relationships are different from the genetic relationships, with three outlying groups, East

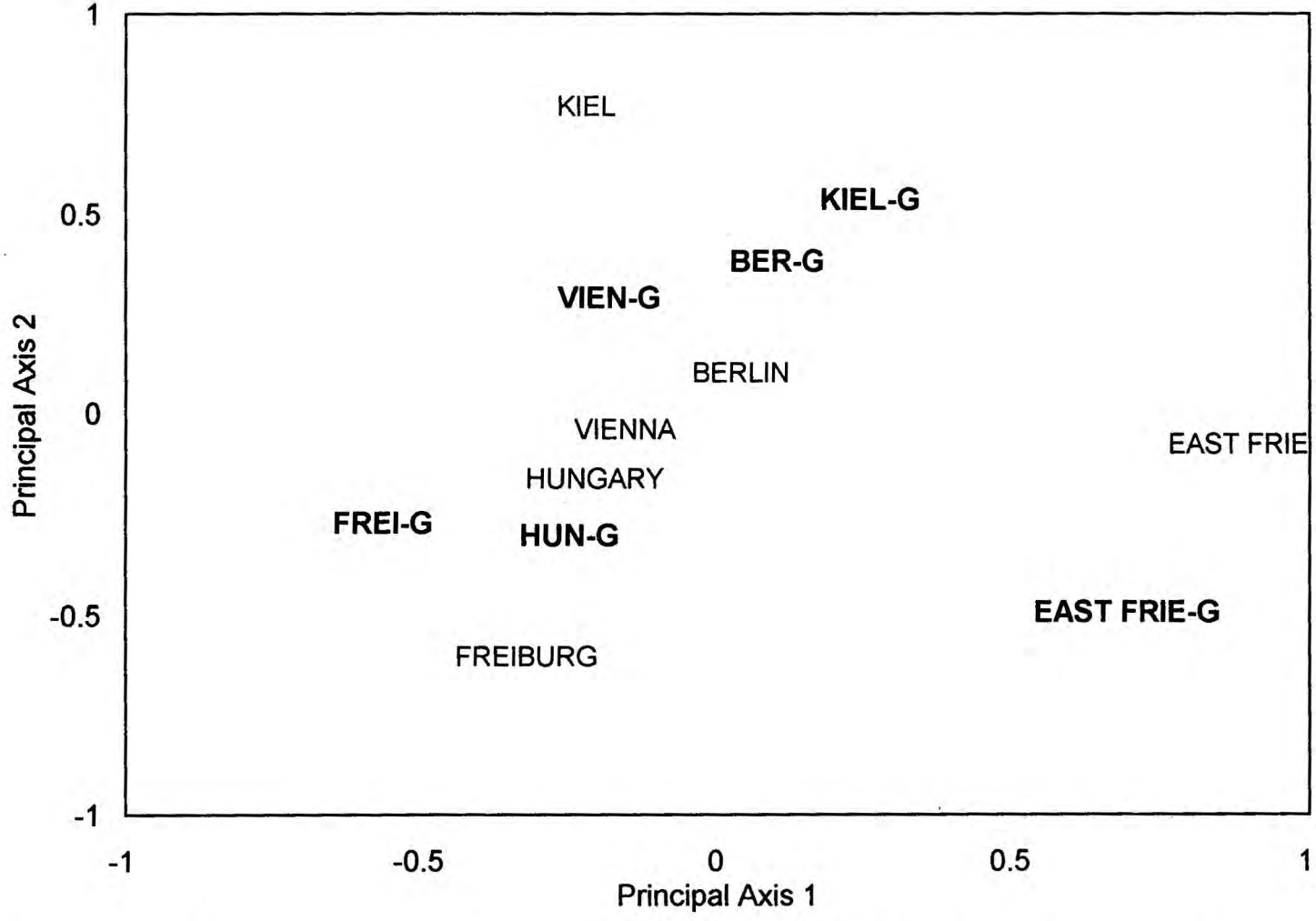


Figure 11. Population Relationships Based on Phenotypic and Genetic Values for Radial Ridge Counts of the Fingers (Two Dimensions). The abbreviated population labels with bold text and the -G suffix represent genetic population centroids. EAST FRIE: East Friesland.

Friesland, Freiburg, and Kiel dispersed around the other groups which form a tight core in the center. The differences between genetic and phenotypic centroids of the same groups are greatest for the three phenotypic outliers. These groups are displaced in three different directions (roughly counter-clockwise) along axes 1 and 2 from their genetic centroids. A three dimensional plot using canonical axis 3, which represents 20% of the total genetic and 14% of the total phenotypic variance, is shown in Figure 12. The Berlin, Vienna, and Hungary samples, which showed little genetic and phenotypic differences on axes 1 and 2, show much lower genetic than phenotypic scores on axis 3, as does the Kiel sample. Both the two-dimensional and three dimensional plots indicate that the genetic and phenotypic relationships between populations are different.

A cluster analysis was also performed on the distance matrices and results are shown in Figure 13. The clusters, like the matrices, show some similarities and some differences. The East Friesland sample is an outlier in each. The biggest difference between them is that the Kiel sample, which is genetically closer to the Berlin-Vienna cluster, is more distant phenotypically, displaced by the Freiburg-Hungary cluster.

The genetic variance-covariance matrix for fingers, palms, and toes shows a high level of integration, with very high genetic correlations among traits. The phenotypic variance-covariance matrices for these traits are quite different in structure. However, important differences between the additive genetic and phenotypic variance-covariance matrices are not detectable using current matrix comparison methods. These differences

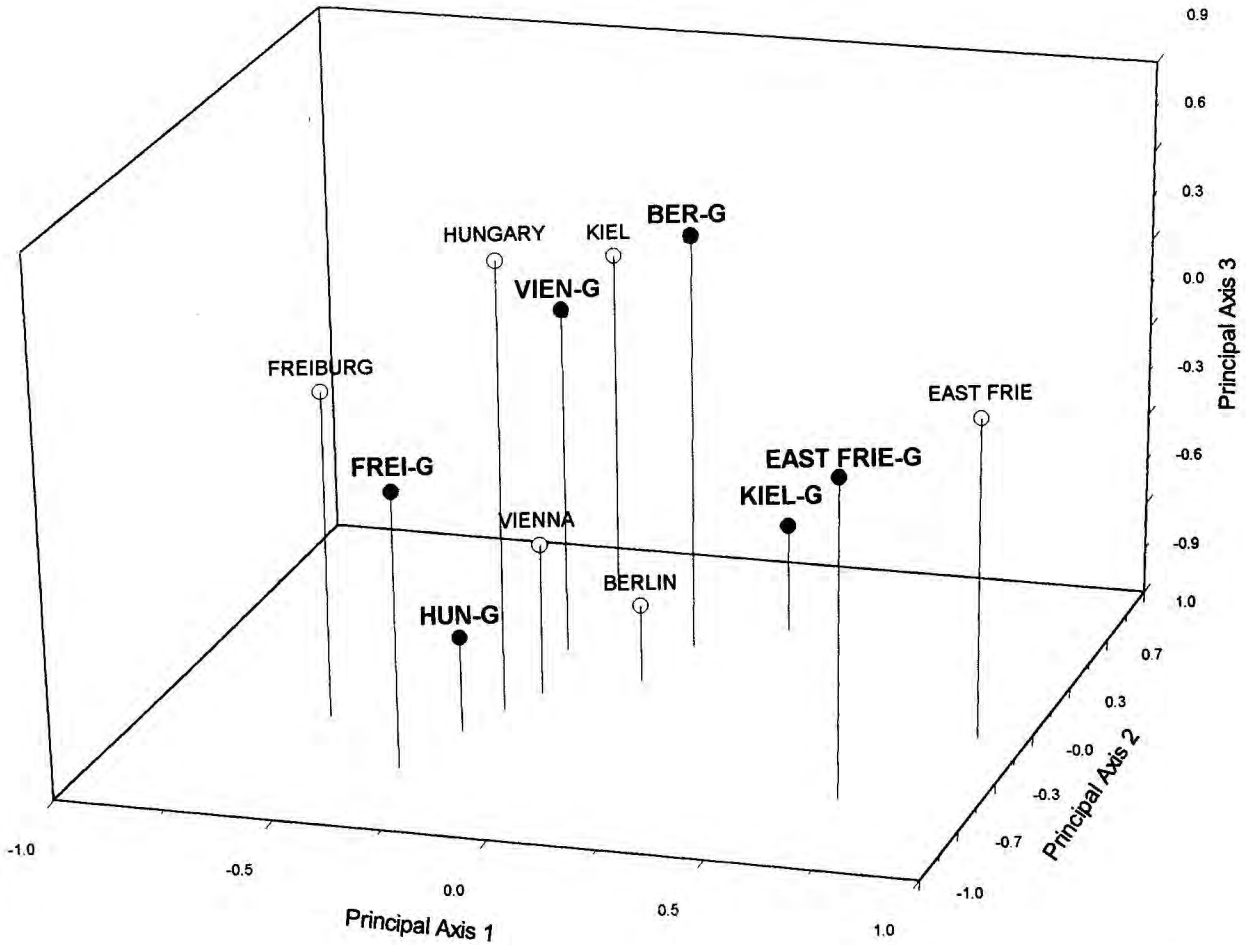


Figure 12. Population Relationships Based on Phenotypic and Genetic Values for Radial Ridge Counts of the Fingers (Three Dimensions). Hollow circles represent the phenotypic centroids. Filled circles with the abbreviated population labels with bold text. and the -G suffix represent genetic population centroids. EAST FRIE: East Friesland.

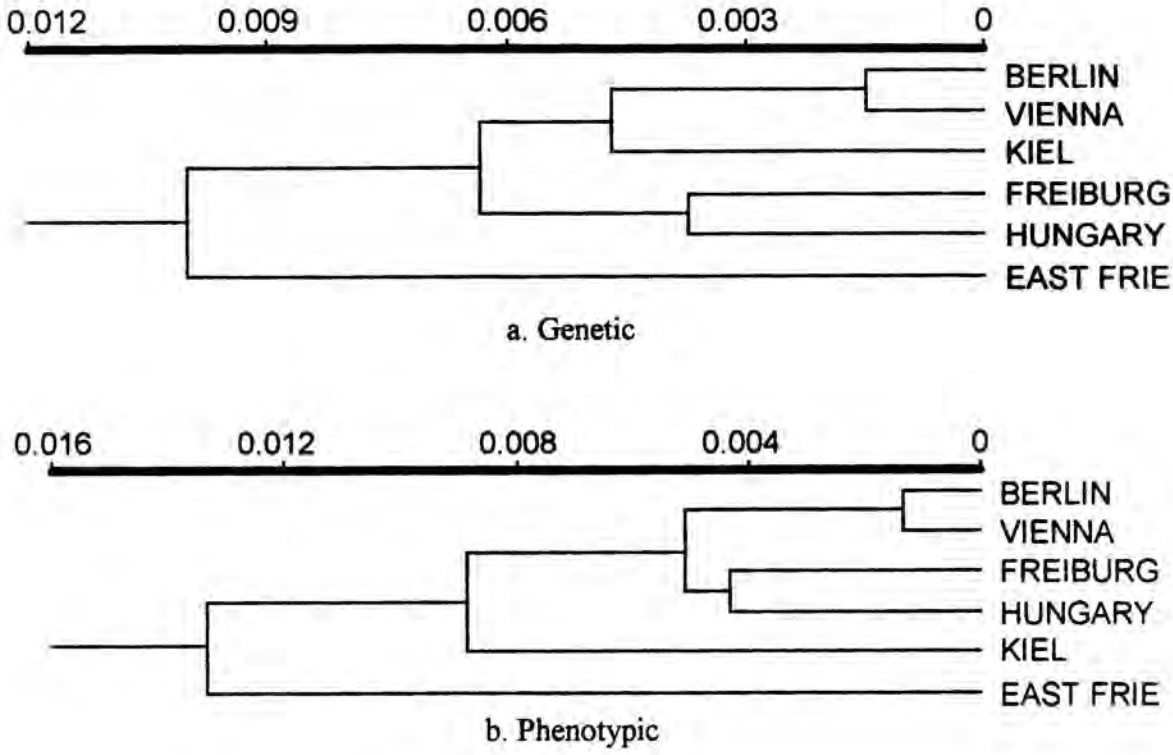


Figure 13. Cluster Analysis of Population Distances. Numbers represent distances. EAST FRIE: East Friesland.

are due to primarily finger-specific dominance and environmental influences. In this practical test of the assumption of proportionality of the additive genetic and phenotypic variance-covariance matrices, the violation of this assumption has been shown to have serious implications for assessing the interrelationships among populations and interpreting the statistical significance of distances among populations. Population relationships among these groups are very different using phenotypic and genetic data. With more distantly related populations, this distortion is likely to increase.

Chapter 6
DISCUSSION AND CONCLUSIONS

Variances, Covariances, Heritability, and Heritabilities

Anthropologists who have estimated population relationships based on ridge counts from each finger have overwhelmingly applied the higher heritability for TRC in their studies rather than the lower heritabilities for individual fingers (Jantz 1987). The exclusion of nonadditive genetic variation, environmental variation, and natural selection enabled a simpler interpretation of dermatoglyphic comparisons among populations. An examination of the genetic parameters of epidermal ridges in the present study has revealed important methodological mistakes which have implications for the genetic study of epidermal ridges and other quantitative traits.

Holt's predominant genetic analyses that indicated a high degree of genetic determination for dermatoglyphic traits were based solely on results from TRC. She analyzed TRC because the methods of quantitative genetics were initially restricted to correlation and regression, which are limited to analyzing one variable at a time. TRC was believed to represent overall tendencies in finger ridge counts, but relationships between variables were omitted. The predicted negative environmental covariances (Cantor 1983; Loesch 1983; Martin et al. 1982c), which were supposed to explain higher heritability for TRC and lower heritabilities for individual finger ridge counts did not appear in the present study.

The present study has revealed that the high heritability of TRC is a statistical artifact, due to the omission of estimates of repeatability. Holt's implicit assumption in using TRC was that finger pad configurations are metameric traits. TRC, however, clearly masks the different heritabilities and dominance effects for individual fingers, and due to epidermal variation within individuals, its heritability is far lower than 100%. The much lower multivariate heritabilities for individual finger ridge counts justify Falconer's (1981, 1989) and Weninger's (Weninger 1964; Weninger *et al.* 1976; Weninger 1983) repeated objections to combining ridge counts. Given the lower heritabilities of epidermal ridges, birth defects and other medical anomalies which have some association with certain dermatoglyphic peculiarities may involve environmental disturbances rather than the previously assumed genetic causes (Chakraborty 1991; Fañanas *et al.* 1996; Nance 1974; Roberts 1979).

Though the additive genetic variance of ridge counts is overestimated using TRC, as shown in the present study and others, significant dominance was also detected using TRC. On the level of individual fingers, part of the dominance is an artifact of using the greater counts from fingers. Digit 2, which showed dominance effects in greater ridge counts, showed no dominance effects in radial counts. Genetic dominance significantly contributes to variation in finger dermatoglyphics, and probably to variation in palm and toe dermatoglyphics as well. Some part of the dominance variance no doubt reflects intrauterine effects, which make siblings more alike than expected from the additive effects

of parental genes (Cantor 1983; Reed *et al.* 1979), with the result that some of the detectable dominance is "statistical" dominance, rather than genetic dominance.

Additionally, a high level of genetic integration (Holt's (1952) "size effect") for any quantitative traits complicates heritability estimates. All epidermal ridge systems showed significant and high correlations among all variables. High counts in one area tend to be accompanied by high counts in other areas, but the univariate and multivariate heritabilities for each variable are quite different, due to the different methods of calculating heritabilities. While the univariate heritability is simply a ratio of additive genetic to total phenotypic variance, the multivariate heritabilities are calculated using $\mathbf{P}^{-1}\mathbf{G}$: If there are substantial genetic covariates, they **must** by definition lower estimated heritabilities due to significant heritable **co**variance. In essence, the level of genetic integration and mean heritabilities are inversely related. As the level of correlation or number of interdependent traits increases, the mean heritability decreases. The decline in heritabilities as one adds covariates for finger ridge counts is shown in Table 17. Given that fingers clearly pair up into right and left homologous fingers, this should come as no surprise. When homologous fingers are included in the 10 variable multivariate analysis, the heritabilities decrease even more. However, the relationships between fingers are also revealed in separate comparisons: The thumb's greater independence from the rest of the hand is illustrated by minimal lowering of heritability from the univariate to the 5 variable comparisons, in contrast to the other fingers. A multivariate analysis of paired homologous

Table 17. Comparison of Narrow-Sense Heritability Estimates for Radial Counts.

	LRC1	LRC2	LRC3	LRC4	LRC5	RRC1	RRC2	RRC3	RRC4	RRC5
Univariate										
	0.69	0.55	0.67	0.66	0.70	0.63	0.52	0.65	0.62	0.69
Left Hand Multivariate (5 variables)										
	0.54	0.37	0.40	0.36	0.56					
Right Hand Multivariate (5 variables)										
						0.60	0.36	0.40	0.37	0.53
All Variables Multivariate (10 variables)										
	0.48	0.30	0.27	0.25	0.35	0.27	0.31	0.26	0.23	0.35
Homologous multivariate (L+R)		RC1		RC2		RC3		RC4		RC5
		0.64		0.43		0.50		0.38		0.64

digits (right + left) showed slightly higher heritabilities than the hands estimated separately, due to the removal of asymmetry.

Finger ridge counts have been described as the most heritable quantitative traits observed in man (Bulmer 1980; Chakraborty 1990; Holt 1968; Lange *et al.* 1988; Miller 1981; McKusick 1969). However, the multivariate heritabilities of finger ridge counts from the present study are much lower (.31) and approximate the multivariate heritabilities of palm and toe ridges. For comparative purposes, human body and head anthropometrics have a slightly higher mean multivariate heritability of .42 (Konigsberg and Ousley 1995), and morphological traits from a variety of animals have an average univariate heritability

near .40 (Mousseau and Roff 1987) or .35 (Cheverud 1988). The latter heritabilities are low compared to the univariate heritabilities for finger ridges, which average .65. Ridge counts may have higher heritabilities than many morphometric traits, but more multivariate comparisons are needed among trait sets.

As the present study has demonstrated, the genetic covariance pattern influences the calculation of heritabilities, and covariances are best estimated using simultaneous multivariate maximum likelihood estimation. One cannot estimate heritabilities without properly estimated components of variation. As is quite clear from the deflation of heritabilities, univariate estimates are best regarded as maximum estimates assuming no covariates. The presence of negative genetic covariances could raise some heritabilities, but overwhelmingly positive variance-covariance matrices are widespread among animals (Atchley 1984; Cheverud 1982; Olson and Miller 1958). Principal components and factor analysis have been used to combine multiple variables together for a regression and correlation analysis using pedigrees to enable easier multivariate calculations of heritabilities (Atchley and Rutledge 1980; Blangero 1988; Cheverud 1982; Chopra 1979; Leamy 1977; Relethford and Blangero 1990; Martin *et al.* 1982c; Rostron 1977). But results from the present study show that estimates of the "heritability" of a principal component are likely inflated because it is a composite trait, as is TRC. Estimating the genetic parameters of principal components may overestimate the additive component to the greatest degree because principal components are linear combinations that maximize

variances among individuals. A truly multivariate genetic analysis must involve the simultaneous estimation of all parameters.

As the present study illustrates, and as others have noted, heritability as traditionally defined does not explain enough about the behavior of quantitative traits (Falconer 1981, 1989; Feldman and Lewontin 1975; Houle 1992). The influences of additive genetic covariances are too important to be left out (Willis *et al.* 1991). Just as it no longer makes sense to talk about **the** heritability of dermatoglyphic ridge counts, it may make little sense to talk about **the** heritability of ***any*** trait without qualifications. The phrase is inherently ambiguous due to heritable covariance as shown in the off-diagonals of the $\mathbf{P}^{-1}\mathbf{G}$ matrix. The researcher now has at least five narrow-sense ' heritabilities" to consider: The univariate heritabilities, theoretically the highest estimates (V_G/V_P); the multivariate heritabilities, as part of a trait complex ($\mathbf{P}^{-1}\mathbf{G}$); the proportion of genetic variation in a trait complex (the summed diagonals of **G** divided by the summed diagonals of **P**); the total amount of covariance explained (ratio of summed off-diagonals); and the ratio of the last two (the sum of the diagonals divided by the sum of the off-diagonals). All of these "heritabilities" are potentially important in assessing morphological integration, predicting response to selection, and comparing populations (Bailey 1956; Barton and Turelli 1989). They may also aid in understanding selectional limits, stasis, spontaneous response, and other unpredictable selectional phenomena (Barton and Turelli 1989; Falconer 1989; Gromko *et al.* 1991; Houle 1992; Maynard Smith et al. 1985; Reeve and Robinson 1953; Willis *et al.* 1991; Yoo 1980).

Fisher objected to the great emphasis laid on one number, a heritability, expressing the genetic qualities of traits, preferring that measurements "must be adequate to evaluate the entire organism as a going concern, and not merely one aspect, or 'point', indicative of its value", emphasizing the "physiological [inter-] correlations of different characters", and expressed disdain for statistics such as "heritability, which I regard as one of those unfortunate short-cuts, which have often emerged in biometry for lack of a more thorough analysis of the data." (Fisher 1951:217). More specifically, regarding the calculation of heritability,

> "the numerator has a simple genetic meaning.. the amount of the relevant measurement directly available for utilization by selection, [but] the denominator is the total variance due to errors of measurement [as well as] those due to uncontrolled, but potentially controllable environmental variation. Obviously, the information contained in the numerator is largely jettisoned when its actual value is forgotten, and it is only reported as a ratio to this hotch-potch of a denominator."
> (Fisher 1951: 217-218)

Some of Fisher's contempt for the use of heritability probably comes from the fact that heritability was invented by Wright (1921), with whom Fisher quarreled for most of his academic life (Provine 1986). However, the point is well taken that in many studies, results have emphasized heritabilities alone, with little or no mention of means, variances, or other sources of variation. This single-mindedness in quantitative genetic studies is still a concern (Houle 1992). The present study highlights the importance of estimating all components of variation in quantitative genetic studies.

It has proven misleading to depend on one value, a heritability, or a composite variable, to estimate the genetic behaviors of traits. The present study has also illustrated the folly of relying on one number (or two, a correlation and significance figure) to assess matrix relationships. Matrix comparison statistics have been shown to produce erroneous indications of association. The present study uncovered patterns of variation inestimable in studies of univariate variables, composite variables, or principal components. It is more rewarding to analyze traits in a truly multivariate framework and then ascertain the relationships among and within the different components of variation. Investigators may have to use fewer traits, larger samples, and more complex procedures to estimate the components of variation in quantitative traits, but the outcome will be more reliable and meaningful results.

Dermatoglyphics and Developmental Integration

The genetic properties of epidermal ridges are best understood in a developmental framework. Some reduction in multivariate, as opposed to univariate, heritabilities for epidermal ridges must be due to higher genetic correlations among ridges, indicating more highly integrated developmental processes. The results of Hale (1952, 1956) and Babler (1979, 1987, 1991) strongly confirm the developmental hypothesis of Mulvihill and Smith (1969). In the context of the hypothesis, the highly integrated additive genetic variance-covariance matrix for epidermal ridges in the present study reflects a high level of integration for developmental processes such as pad expansion and regression, and ridge formation commencement and termination. Other developmental factors may include a

cellular capacity for repair. Based on results of epidermal transplants in insect larvae (Locke 1959, 1960), finger print patterns may represent a reparatory response to cellular damage or disorientation during the period of pad expansion.

EGF synthesis and cell sensitivities to EGF may provide a developmental explanation for Babler's (1978) demonstration of prenatal selection against lower ridge counts and arches. It is doubtful that dermatoglyphic traits are directly selected against. Instead, ridge counts probably reflect the developmental status of other crucial organ systems, or indicate how the organism is developing overall, reflecting the interactions of various epigenetic controls. EGF is a very important hormone for the proper development of many organ systems, as well as for the survival and retention of the fetus. EGF also influences the development of epidermal ridges. All surviving persons have survived prenatal selection, and given the high level of genetic integration, Babler's (1978) results may also represent prenatal selection for a high level of developmental integration. Alternatively, it may be difficult to produce independent epidermal relationships because of pleiotropic dermatoglyphic genes that direct many other interdependent developmental relationships as well.

Epigenetic control involving EGF may explain the observed correlation between fingerprint patterns and the ossification of phalanges (Babler 1989), and the "neighborhood" or "proximity" effect observed among fingers in the present study. These effects were observed in the process of cartilaginous/chondral ossification and its outcome represented by the adult bone morphology of the hand (Lewenz and Whitely 1903;

Whiteley and Pearson 1900). The proximity effect observed in each hand, with adjacent body parts more highly correlated with each other, has been found repeatedly in many other animals (Alpatov and Boschko-Stepanenko 1928; Cheverud 1982, 1996; Guttman and Guttman 1965; Leamy 1977; Olson and Miller 1958; Pearl and Clawson 1907; Roth 1996). The proximity effect is limited to radial counts, however. The extremely different distributions of radial and ulnar counts seen in the present study also support the concept of separate dermatoglyphic radial and ulnar developmental fields (Roberts and Coope 1975).

The differences between palmar and digital ridge configurations are also best understood in terms of development. Adjacent palmar ridge areas show different quantitative genetic relationships to each other than adjacent finger and toe ridge areas due to the different topological considerations of palm ridge development as opposed to finger and toe ridge development. The different relationships within ridge systems contradict de Wilde's (1979, 1982) hypothesis of the very early determination of finger patterns. If finger patterns were determined early in development, at the hand plate stage, when areas are physically adjacent and inseparable, one would expect the relationships within finger counts to be similar to that within palm counts, with negative environmental covariances between adjacent ridge forming areas and large dominance effects. Instead, finger pattern configurations show much lower dominance and environmental covariances, none of which are significant and negative. While some finger ridges are clearly determined much earlier in development (such as the interphalangeal creases), the

differences between finger and palmar ridge counts indicate that the finger patterns are formed when the fingers have separated, after (and physically between) the very early forming continuous ridges. This conclusion is confirmed by a reconsideration of finger print patterns as distortions of the earlier ridge bundles described by de Wilde (1979, 1982).

Given the many interdependent processes involved in epidermal ridge development, and evidence for environmental influences (Schaumann and Alter 1976; Sorenson Jamison *et al.* 1993, 1994), it may come as no surprise that ridge counts are not entirely additive traits. However, the additive genetic variance-covariance matrix shows a high level of integration, far higher than is present in the phenotypic variance-covariance matrix. High genetic and developmental integration may produce an evolutionary constraint because changes in one variable can only occur along with coordinated changes in other variables (Atchley et al. 1994). Evolutionary constraints would result in slower rates of evolution for dermatoglyphic traits as opposed to anthropometrics and blood markers, as has been proposed (Froehlich and Giles 1981a, 1981b). If genetically integrated traits are evolutionarily constrained, the source of evolutionary constraints is pleiotropy, rather than any inherent qualities of polygenic traits, as had been assumed (Birdsell 1950; Froehlich and Giles 1981a, 1981b; Relethford and Lees 1983).

If ridge counts are more constrained evolutionarily, ridge counts may reflect "ancient" population relationships better than anthropometrics (Froehlich and Giles 1981a,

1981b), even if epidermal ridge counts have lower heritabilities than anthropometric traits. However, for accurate population relationships, the genetic variance-covariance matrix would need to be proportional to the phenotypic variance-covariance matrix (Cheverud 1988), which has been shown to be an incorrect assumption for the populations in the present study. The $\mathbf{P}^{-1}\mathbf{G}$ matrices for fingers, palms, and toes indicate nonproportionality of the additive genetic and phenotypic variance-covariance matrices. Further, the observed high genetic integration of dermatoglyphic traits relative to phenotypic integration results in misleading significance figures for distances derived from phenotypic data. Overestimates of statistical significance for distances are due to greater finger-specific dominance and environmental effects, which inflate the phenotypic variances relative to the phenotypic covariances. The pattern of variance inflation is not limited to finger ridge counts, with fewer apparent interdependent spatial relationships than, say, mammalian craniometrics. The relationships within the finger and palmar ridge additive genetic variance-covariance matrices are very similar. Palms have proportionally higher phenotypic variances than covariances in ratios similar to finger ratios, though some of the higher variances are due to negative environmental covariances between adjacent variables. Toes show a similar pattern. Variance inflation is due to largely independent dominance and environmental effects in epidermal ridge systems.

The greater significance figures for phenotypic distances than for genetic distances is a recurrent pattern. Comparisons of morphological versus genetic traits consistently produce higher statistical significance for phenotypic distances among humans or animals

compared to genetic distances (Black 1982; Friedlaender 1976; Lewontin 1984; Neel and Ward 1970; Ousley 1993, 1995; Relethford 1983; Relethford 1988; Relethford and Lees 1982; Spielman and Smouse 1976; Szathmary 1976; Wright 1978). The greater statistical significance of phenotypic differences has previously been attributed to developmental constraints and integration (Cheverud 1982, 1984; Maynard Smith *et al.* 1985; Moss and Young 1960) or statistical artifact (Lewontin 1984). In contrast, dermatoglyphic results from the present study show that the higher phenotypic diversity and significances may be due to what may be more objectively termed *phenotypic disintegration*: The phenotypic variance-covariance matrix of all ridge systems is a diluted form of the more highly integrated genetic variance-covariance matrix. The different phenotypic variance-covariance matrix pattern is due to less integrated, more independent dominance and environmental effects on fingers. While some level of phenotypic integration (developmental fields) can be detected in dermatoglyphic traits (Jantz and Owsley 1977; Jantz *et al.* 1984; Knussman 1967, 1969; Reed *et al.* 1978; Roberts and Coope 1975; Siervogel *et al.* 1978), the additive genetic integration is higher. It is difficult to argue against phenotypic disintegration in the present study, which involved the same observer, the same traits, the same data sets, and the same subjects. Phenotypic disintegration is a factor inflating the statistical significance of estimated distances among populations using quantitative phenotypes.

Dermatoglyphic Traits and Population Relationships

Phenotypic disintegration produces inaccurate population relationships based on dermatoglyphics in addition to inflated significance figures. Froehlich and Giles (1981a, 1981b) relied on a high heritability and the selective neutrality of dermatoglyphic traits to claim that the phenotypic relationships between populations would mirror the genetic relationships between them. However, the heritabilities of dermatoglyphics are much lower than previously estimated, allowing room for dominance and environmental effects. The present study has shown that variation among dominance and environmental effects distorts the genetic relationships among variables, and by extension, distorts the relationships among populations. Cheverud's (1988) general rule of proportionality of additive genetic and phenotypic variance-covariance matrices apparently holds for anthropometrics (Konigsberg and Ousley 1995). The results of the present study indicate that the assumption of proportionality is not valid for dermatoglyphic traits, despite matrix comparison statistics to the contrary.

Diversity statistics such as F_{ST} can be quite different based on genetic and phenotypic values. In order to compare levels of variation among groups in the present study, the calculated F_{ST} of .0034 for the phenotypic radial count data was based on a heritability of 1, as was the genetic value (.0033) for radial counts. Genetic values naturally have a heritability of 1, but the calculation of F_{ST} using phenotypic values must use a more realistic heritability. A heritability of .6 produces an estimated F_{ST} of 0.0056 and a heritability of .3 yields an Fst of 0.011. These values indicate a much higher level of

among-group phenotypic variation, representing local phenotypic differentiation that is not reflected in the genetic values. Therefore, phenotypic traits may increase diversity statistics such as F_{ST} compared to the genetic values of the same trait. One may thus question the validity of comparing phenotypic and genetic F_{ST}s from different data sets such as craniometrics, anthropometrics, dermatoglyphics, and blood markers (Jantz 1996, 1997; Jorde 1985; Konigsberg and Blangero 1993; Ousley 1993, 1995; Relethford 1994). Thus, analyzing phenotypic dermatoglyphic data produces distorted phylogenetic relationships among populations, overestimated statistical significance for distance measures, and inflated diversity statistics such as F_{ST}.

Given differences in proportionality, dermatoglyphic and anthropometric comparisons among populations will produce different population relationships, though dermatoglyphics and anthropometrics are correlated with each other to some extent (Loesch and LaFranchi 1990; Sorenson Jamison *et al.* 1990). There may be discernible reasons why dermatoglyphics and anthropometrics do not indicate the same population relationships. The differences between each type of trait may lie in differences in dominance and environmental effects. The proportionality of anthropometric variance-covariance matrices may be due to equally proportional additive genetic, dominance, and environmental effects, or insignificant dominance effects and randomly distributed environmental effects. Alternatively, the quite different developmental histories of dermatoglyphic traits and anthropometrics could produce different developmental (genetic) relationships among variables. The level of genetic integration may be lower for

adult anthropometrics, due to a much longer period of development. Anthropometric traits may therefore show the effects of greater developmental plasticity, but may also require greater functional phenotypic integration than dermatoglyphic traits. The more flexible development and greater functional requirements of anthropometric phenotypes may result in greater proportionality of the additive genetic and phenotypic variance-covariance matrices for anthropometrics.

Different selection pressures acting on dermatoglyphics and anthropometrics may produce differences in proportionality. The directional selection for higher ridge counts *in utero* found by Babler (1978) would probably not misrepresent phylogenetic relationships among populations because all printed persons will have survived childbirth. Results from estimating selection using adult dermatoglyphics (Blangero 1988; Loesch and Wolanski 1985) are in disagreement. The results of Rogers and Harpending (1983) indicating dermatoglyphic neutrality depended on calculating the heritabilities of principal components, which have been shown to produce erroneous results in the present study. However, because Rogers and Harpending (1983) estimated population relationships based on anthropometrics, dermatoglyphics, and blood markers (though there were some differences in sample composition), their results are of interest for comparison. Anthropometrics showed a much higher diversity than dermatoglyphics and neutral blood markers, and a realistic heritability (.4) for anthropometrics would mean that anthropometrics are roughly 12 times more diverse in their sample than blood markers, strongly suggesting the influence of selection. The test for the neutrality of

dermatoglyphics was based on a heritability of 1 for all eigenvalues. Based on the estimated heritability of the second principal component (.25) from Rostron (1977) and the second eigenvalue (1.00) and standard error (.73) of finger greater counts from Rogers and Harpending (1983), there is evidence for selection on dermatoglyphic traits because the second dermatoglyphic principal component is over 4 times more diverse than blood marker principal components ($p < .01$). Anthropometrics and dermatoglyphics may be subject to different selective pressures, and selection may operate to change population relationships as well as diversity. A better test of variable sets would involve multivariate ML estimates of genetic parameters from the populations. In the present study, the phenotypic and genetic population dermatoglyphic F_{ST}s were roughly equivalent using a heritability of 1. However, when a heritability of .3 is used, the phenotypic (.011 ± .0015) and genetic (.0033 ± .0010) F_{ST}s are significantly different at $p < .01$, further challenging the value of diversity statistics for comparing genetic and phenotypic traits.

In the present study of dermatoglyphic traits, a large dermatoglyphic sample with pedigrees allowed the estimation of multivariate genetic parameters. Heritabilities of dermatoglyphic ridge counts were shown to be far lower than 1. The lower heritabilities, partially due to high levels of genetic integration, permit dominance and environmental effects to influence dermatoglyphic phenotypes. The dominance and environmental effects are largely independent, with the greatest influence on trait variances, resulting in variance inflation in dermatoglyphic phenotypes. Dominance and environmental effects also varied in magnitude, and dominance effects were stronger for the right hand, distorting the

genetic pattern among variables, a process termed phenotypic disintegration. Variance deflation and phenotypic disintegration produced significant changes in population relationships including different phenotypic relationships among populations compared to additive genetic relationships; exaggerated statistical significance for phenotypic distance measures; and overestimated diversity statistics such as F_{ST}. All of these discrepancies between phenotypic and additive genetic values reflect nonproportionality of the additive genetic and phenotypic variance-covariance matrices, despite matrix comparison statistics to the contrary. Further multivariate explorations of the variance components of dermatoglyphics and other quantitative traits are needed.

SUMMARY

This dissertation has investigated the genetic features of dermatoglyphic ridge counts in a multivariate analysis. Results of this study, utilizing maximum likelihood estimation and the largest data set with pedigrees so far, contradict many previous results and assumptions concerning dermatoglyphic heritabilities and neutrality, and proportionality of the additive genetic and phenotypic variance-covariance matrices. The components of dermatoglyphic variation are best understood developmentally.

Heritabilities of dermatoglyphic ridge counts in the fingers are much lower than has been estimated and assumed, and approximate the heritabilities of palm and toe ridge counts. Ridge count heritabilities are lower due to significant dominance and environmental effects, but also to a high level of genetic integration, as shown by high genetic correlations among variables. A high degree of genetic integration lowers multivariate heritabilities for each covariate. The high level of genetic integration may be evidence for stabilizing selection through rather rigid epigenetic control of development. EGF, with its widespread action on the developing fetus and the epidermis, may be an especially important hormone for developmental integration.

The level of integration is much lower in the phenotypic variance-covariance matrix than in the additive genetic variance-covariance matrix due to varying and independent dominance and environmental effects. These effects produce relatively larger variances in the phenotypic variance-covariance matrix compared to the additive genetic

variance-covariance matrix. As a result, the phenotypic variance-covariance matrix is not proportional to the additive genetic correlation, and phenotypic dermatoglyphic traits cannot be expected to reflect the phylogenetic relationships between populations when used in an analysis of population structure.

Unfortunately, the lower integration of the phenotypic variance-covariance matrix also produces overestimates of statistical significance for phenotypic distances among populations and inflated measures of overall diversity. Analyses of phenotypic data produce more significant distances and greater diversity among populations than analyses of genetic data, and the components of variation among variables examined in this analysis may have revealed a quantitative genetic basis for this pervasive pattern.

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APPENDIX

Finger Ridge Count Means for Sample with Pedigrees

Sex	Lrc1	Lrc2	Lrc3	Lrc4	Lrc5	Rrc1	Rrc2	Rrc3	Rrc4	Rrc5
F	14.80	7.50	11.05	15.39	12.43	17.10	8.24	11.48	15.63	12.30
M	17.05	8.28	12.37	16.54	13.85	19.64	7.44	11.81	16.42	13.67

Palm Ridge Count Means for Sample with Pedigrees

Sex	Lc-d	Lb-c	La-b	Ra-b	Rb-c	Rc-d
F	36.83	27.09	42.39	41.21	27.60	37.66
M	36.79	27.34	42.60	41.59	27.87	38.21

Toe Ridge Count Means for Sample with Pedigrees

Sex	Ltc1	Ltc2	Ltc3	Ltc4	Ltc5	Rtc1	Rtc2	Rtc3	Rtc4	Rtc5
F	11.25	9.11	13.85	9.12	1.93	11.98	10.22	15.29	10.14	2.92
M	11.87	10.22	15.80	11.88	3.34	12.76	11.42	16.96	12.67	4.56

Finger Ridge Count Means for Population Samples

SEX	GROUP	Lrc1	Lrc2	Lrc3	Lrc4	Lrc5	Rrc1	Rrc2	Rrc3	Rrc4	Rrc5
F	BERLIN	14.56	7.84	10.84	15.85	12.81	17.28	8.68	11.19	16.31	12.97
M	BERLIN	17.84	8.47	13.26	17.27	14.50	20.01	8.10	12.41	16.70	14.44
F	EAST FRIE	14.12	8.26	9.14	15.72	13.05	15.74	6.95	9.42	14.95	13.21
M	EAST FRIE	15.44	8.20	12.00	16.92	14.31	18.10	7.41	12.72	16.67	14.18
F	FREIBURG	15.45	7.73	10.76	15.46	12.58	17.27	8.28	10.81	14.51	11.72
M	FREIBURG	17.43	8.19	12.68	16.94	13.75	19.57	7.93	11.88	16.10	13.33
F	HUNGARY	13.79	6.91	10.06	14.12	11.10	15.69	6.76	9.87	14.11	11.16
M	HUNGARY	17.15	8.22	12.00	16.92	13.66	19.94	7.50	11.12	16.31	13.19
F	KIEL	14.21	6.64	10.42	15.34	12.50	16.62	6.78	10.12	15.54	11.82
M	KIEL	16.89	8.86	13.57	17.58	14.53	20.13	7.01	12.23	18.54	14.31
F	VIENNA	15.13	7.72	11.41	15.72	12.61	17.34	8.50	11.57	15.86	12.47
M	VIENNA	17.13	8.99	12.83	17.31	13.99	19.49	8.39	12.15	17.00	13.71

VITA

Stephen David Ousley was born on March 10, 1961, and eventually escaped from Seabrook, Maryland, where he grew up. He attended the University of Maryland, College Park, and was the physical anthropologist and hack zooarchaeologist for the 1982 Caesarea Maritima Summer Excavations. In 1985 he received a B.A. in Anthropology and nearly one in History. He then spent a year in Germany as an exchange student, studying German history, language, and beer. On his return, he worked at the Smithsonian Institution as a Museum Technician, then worked at the Armed Forces Institute of Pathology as a collections manager for the Blackburn-Neumann Neuropathology Collection.

Steve arrived at the University of Tennessee in 1989. Since then he has been the manager of the Forensic Data Bank and research assistant to Dr. Richard Jantz, with whom he co-authored FORDISC 1.0 and 2.0, computer programs that estimate the sex and race of an unknown skull using information from the Forensic Data Bank. His interests include all applications of the theory and methods of Biological Anthropology, especially human and primate variation and evolution, skeletal biology, quantitative genetics, and forensics.