12-1978

Dermatoglyphic Variability and Asymmetry of Patients with Cleft Lip and Cleft Palate

Douglas William Owsley
University of Tennessee, Knoxville

To the Graduate Council:

I am submitting herewith a dissertation written by Douglas William Owsley entitled "Dermatoglyphic Variability and Asymmetry of Patients with Cleft Lip and Cleft Palate." I have examined the final electronic 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.

Richard L. Jantz, Major Professor

We have read this dissertation and recommend its acceptance:

William M. Bass, Fred H. Smith, Patrick J. Carney

Accepted for the Council:

Carolyn R. Hodges

Vice Provost and Dean of the Graduate School

(Original signatures are on file with official student records.)

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DERMATOGLYPHIC VARIABILITY AND ASYMMETRY OF PATIENTS WITH CLEFT LIP AND CLEFT PALATE

A Dissertation

Presented for the

Doctor of Philosophy

Degree

The University of Tennessee, Knoxville

Douglas William Owsley

December 1978

ACKNOWLEDGEMENTS

This dissertation was completed with the aid of several individuals to whom I am very grateful. Special thanks are given to my committee members, Drs. Richard L. Jantz, William M. Bass, Fred H. Smith and Patrick J. Carney, for initiating my interest in this research and continued support and guidance. Their office doors were always open for consultation and assistance. Many of the hypotheses tested were initially suggested by Dr. Jantz.

I thank Dr. John Philpot of the University of Tennessee Department of Statistics for helpful suggestions concerning the statistical methodology. Dr. John Gregg of the Department of Anthropology aided in the cleft lip and cleft palate literature search. Dr. Mike Logan of the Department of Anthropology reviewed this thesis and offered several valuable suggestions. Dr. Letitia Oliveira kindly made data available for the control sample.

Valuable aid was supplied by Carol Dittner, Fred Fohl, David Glassman, Mark Guagliardo, Cleon Hawkinson, Wayne Roberts and Karla Vollmar by helping collect the finger and palm prints. Fred Fohl helped record and analyze the data.

The clinic data was obtained with the help and facilities provided by the East Tennessee Crippled Children's Service, Norma Osborne, Chief Nurse and Original Director. I thank Drs. Edmund B. Andrews, James B. Cox, K. Allen Harper, Robert E. Knowling, William J. Schneider and James W. Taylor and their staffs, Helen Carroll,

Dorothy M. Cody and Janice E. Dockery, for clinical diagnostic information of their patients.

I am most grateful to those families and students who participated in this project. Their willingness to participate is sincerely appreciated. It is my hope that research of this nature will be to their benefit.

To my wife, Susanne, a personal acknowledgement of gratitude for her encouragement and assistance throughout this entire investigation. I also thank my parents, Mr. and Mrs. William Owsley, for their interest in my graduate and undergraduate programs.

This research was partially supported by a University of Tennessee Non-Service Fellowship, a Hilton A. Smith Pre-Doctoral Research Fellowship and the Department of Anthropology.

ABSTRACT

Quantitative dermatoglyphic data for patients with oral-facial clefts and their first degree relatives were compared with controls. The objectives were to define the nature of the differences between those samples and to interpret the differences in terms of developmental processes.

The clinic samples were composed of Caucasian cleft lip and/or cleft palate patients and normal first degree relatives from Knox and surrounding counties in East Tennessee. The control sample consisted of 102 male and 102 female University of Tennessee students and Knoxville children. Specific diagnoses as to cleft type and associated malformations were determined by consulting clinic records. Two diagnostic classifications were employed: (1) patients with cleft lip with or without cleft palate (CL±P; n=88), and (2) patients with cleft palate only (CP; n=29). Patients with numerous developmental defects or Pierre Robin's syndrome were not included. For certain tests, the CL±P sample was divided into two subsamples: (1) probands having additional relatives with any type of cleft (FH+), and (2) patients having no other relatives with clefts (FH-).

The majority of variables were ridge-counts of finger and palmar patterns or dermatoglyphic areas. Radial and ulnar ridge-counts were obtained for each digit. Also, patterns in the second, third and fourth interdigital areas were quantified by counting the number of ridges between the pattern centers and corresponding

triradii. Five variables on each palm defined size of the interdigital patterns, their location and the triradii or main lines essential to their formation. Interdigital counts, a-b, b-c and c-d, were obtained for both palms. Ridge breadth and maximal atd angle was measured on the palms.

The data for CL±P probands and controls were separately factor analyzed and the factors compared. The entire sample (control plus clinic samples) was analyzed to obtain factor scores for all individuals. Those scores were used in multivariate tests for determining whether the factor score means or dispersion matrices of the patient and control samples differed significantly. In separate tests, controls were compared with CL±P probands, CL±P siblings and CL±P parents. Like comparisons were repeated for CP patients and their family members. CL±P and CP probands were tested for differences from corresponding siblings using a multivariate paired samples test. The effect of a positive history of clefts was determined by comparing factor scores of controls, CL±P, FH+ and CL±P, FH- patients.

Ridge breadth and maximum atd angle were analyzed independently from the other variables. Males and females were analyzed separately and an analysis of covariance technique allowed adjusting means for effects of age.

Fluctuating asymmetry of the control and patient samples were compared by testing for variance-covariance homogeneity. The tests measured sample differences in the patterning of the relationships among the asymmetry measures as well as magnitude differences in the within-pair variances.

Ten factors were extracted for controls, CL±P probands and the total sample. Five general types of factors are represented in each group: (1) finger radial count factors; (2) finger ulnar count factors; (3) thumb factors; (4) palmar interdigital count factors; and (5) palmar pattern factors. Controls and CL±P probands contrast in the relative contributions of certain variables to specific factors.

Scores for five factors were retained for further comparisons. Few differences in means are discernible between clinic samples and controls. The data provides no evidence that a positive or negative history of clefts has any affect on mean values. Proband and non-cleft sibling means are similar. CL±P females have significantly wider ridges than noted for control females.

The clinic samples are unusual in the dispersion matrices of factor scores and asymmetry measures. Factor score variance-covariance matrices of CL±P probands, siblings and parents significantly differ from controls. Probands are the extreme in this regard. Only a few variances, when tested individually, display heterogeneity suggesting that the interrelationships among the variables (covariances) differ from controls. The presence of covariance differences seems noteworthy in relation to cleft formation. Facial development requires a highly coordinated or correlated interaction of several embryological structures. Deviation from normal developmental pathways, as involved in the formation of cleft lip and cleft palate, would seem a likely possibility when normal relationships are not maintained. The variables examined bear no

direct relationship with morphogenesis of the face. Therefore, the types of covariance differences indicated in dermatoglyphics may be fairly generalized throughout the individual and somehow related to the cause of oral-facial clefts.

Variance-covariance asymmetry matrices for CL±P and CP probands and their siblings differ from controls although not the parent samples. The asymmetry matrices of CL±P, FH- probands significantly differ from controls. However, in contrast with some previous research, there does not appear to be a consistent tendency for the CL±P samples to show increased fluctuating asymmetry.

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CHAPTER I

DERMATOGLYPHICS IN MEDICINE--AN APPLICATION TO CLEFT LIP AND CLEFT PALATE

I. INTRODUCTION

Clefts of the lip and palate are the most prevalent type of facial malformation and are among the most common of birth defects. The factors responsible for cleft formation are operative in early intrauterine development and result in the failure of epithelial tissues to meet and fuse. In many respects the malformation reflects developmental asynchronies in morphogenesis in which certain structures fail to be in the right place at the right time to interact with other structures (Trasler and Fraser 1977). Coordination in the rates of growth of these structures is extremely important. Rates of growth and the proper coordination of prenatal developmental sequences are also important in the formation of dermatoglyphics--dermal ridges on the hands and feet. Environmental or genetic factors causing growth disturbances leading to cleft formation may affect the development of other structures such as dermal ridges. The present investigation examines this possibility. Furthermore, developmentally relevant dermatoglyphic variables are used to examine certain questions pertaining to the formation of oral clefts.

Quantitative dermatoglyphic data for patients with cleft lip and cleft palate and for first degree relatives have been collected and compared with an appropriate control series. The overall objectives

are to define the exact nature of the differences between the clinic and control samples and to interpret these differences in terms of developmental processes. The approach is different than that generally followed in dermatoglyphic studies of clinical disorders. Frequently the tendency is to search for dermatoglyphic abnormalities which are possibly useful in clinical diagnosis of specific problems. In the case of an overt pathology as cleft lip and cleft palate, such information is unnecessary. Instead the contribution of dermatoglyphics may lie in its ability to increase our understanding of the developmental factors and growth rates involved in cleft formation.

The present chapter focuses on previous investigations of the dermatoglyphics of various clinical samples. The objective is to exemplify the kinds and degree of differences which have been reported and to illustrate the potential usefulness of dermatoglyphics in clinical diagnosis or possibly genetic counseling. This discussion also serves to introduce some of the terminology involved in dermatoglyphic analyses. Previous dermatoglyphic studies of cleft lip and cleft palate patients are important in this discussion and are considered as to research design and results. Modifications in methodology will be suggested and followed in the present analysis. The second chapter reviews the embryology of facial development and considers epidemiological and etiological factors important to the formation of oral clefts. The embryology and genetics of dermal ridges are similarly examined. The remainder of the chapter and Chapter III explores possible interrelationships between dermal ridges and cleft lip and cleft palate. This discussion considers possible reasons why dermatoglyphic differences

are expected in cleft lip and cleft palate patients and possible ways in which dermatoglyphics, may help improve our understanding of oral clefts. The fifth and sixth chapters present the results of the analysis and evaluate their significance.

II. PATIENT-CONTROL DIFFERENCES IN CLINICAL DISORDERS

Dermatoglyphic abnormalities have been identified in a large variety of medical disorders. Review summaries by Alter (1966), Holt (1968a), Preus and Fraser (1972), Schaumann and Alter (1976), and Uchida and Soltan (1963) help systematize and synthesize the very large number of reports describing dermatoglyphic variation associated with disorders caused by autosomal trisomy, monosomy or polysomy of the sex chromosomes, chromosomal structural aberration, single gene defects, disorders or uncertain genetic transmission, of disorders induced by exogenous (environmental) influences. The general emphasis in most research is the examination of affected individuals, only a few researchers have considered non-affected family members.

Dermatoglyphic abnormalities are clearly documented for patients with additional autosomes, particularly Down's syndrome (trisomy 21), trisomy 13 (14 or 15) and trisomy 17 (or 18). Down's syndrome or mongolism has received the most attention in qualitative and quantitative dermatoglyphic studies and was the topic of even the earliest studies investigating relationships between dermatoglyphics and congenital disorders (Cummins 1939). This syndrome generally results from trisomy of a small acrocentric chromosome, the twenty-first. A small percentage of cases are due to trisomic mosaicism or translocation

of the long arm of the twenty-first onto another chromosome of the G (21-22) or D groups (13-15) (Thompson and Thompson 1966:102). Dermatoglyphic features of Down's syndrome tend to be highly characteristic regardless of sex, racial composition or ethnic background (Schaumann and Alter 1966:147). Reference to the dermatoglyphic differences between Down's patients and normal controls illustrates the nature and magnitude of changes possible in severe congenital defects.

Differences between finger patterns of Down's patients and normal controls include an increased frequency of high L-shaped ulnar loops with an associated reduction in mean total finger ridge-count (Beckman et al. 1962; Bryant et al. 1970; Fujita 1969; Giovannucci and Bartolozzi 1968); Holt 1964; Matsui et al. 1966; Shiono et al. 1969; Walker 1957; Zajaczjowska 1969). [The simplest classification of patterns on the fingers recognizes three major types of patterns: arches, loops and whorls (Galton 1892). Loops are further classified by the direction they face, ulnar loops open to the ulnar side of the finger, radial loops to the radial side. The size of a pattern may be quantified by counting the number of ridges between the pattern core and a deltoid shaped structure--the triradius--which defines a point where three parallel ridge systems meet. Each digit has two counts from the radial and ulnar side. The total ridge-count is the largest count, either radial or ulnar, summed over all ten digits.] In fact, 30 to 35 percent of Down's patients have ulnar loops on all digits (Holt 1964; Shiono et al 1969). Down's patients exhibit a shift in the predominance of radial loops to the fourth and fifth fingers whereas the second digit has the highest percentage of radial loops in normal samples (Bryant et al. 1970;

Fujita 1969; Giovannucci and Bartolozzi 1968; Holt 1964; Saksena et al. 1966; Shiono et al. 1969; Walker 1957). In Down's syndrome, loops in the palmar third interdigital area occur in increased frequency and large hypothenal patterns are common. Patterns of the thenar/first area have decreased frequency, size and complexity (Beckman et al. 1962; Berg 1968; Bryant et al. 1970; Dallapiccola and Ricci 1967; Fang 1950; Giovannucci and Bartolozzi 1968; Matsui et al. 1966; Plato et al. 1973; Saksena et al. 1966; Shiono et al. 1969; Walker and Johnson 1965). [Palmar patterns, generally loops or whorls, are found in five areas: in the interdigital areas at the base of the digits between the second and third digits (the second interdigital area), the third and fourth digits (the third interdigital area), the fourth and fifth digits (the fourth interdigital area), and in the thenar (thenar/first interdigital) and hypothenar areas.] In North American whites, for example, Down's patients have an incidence on the left hand of 60.3 percent for patterns in the third interdigital area, 55.6 percent for patterns in the hypothenar area and 2.6 percent for thenar patterns. These values markedly contrast with 25.7 percent, 26.0 percent and 13.4 percent for normal controls (Plato et al. 1973). Mongolism is characterized by distal displacement of the axial triradius t toward the center of the palm, a transition reflected in an increased maximal atd angle (Beckman et al. 1962; Bryant et al. 1970; Cummins 1939; Giovannucci and Bartolozzi 1968; Matsui et al. 1966; Penrose 1954; Saksena et al. 1966; Shiono et al. 1969; Soltan and Clearwater 1965; Walker et al. 1963). [The t or axial triradius generally occurs near the base of the palm near the

axis of the fourth metacarpal bone. This triradius may be displaced distally. A common means of measuring the amount of displacement is with the atd angle (Penrose 1954). This angle is formed by straight lines drawn from the t triradius to triradii positioned beneath the second and fifth digits. The larger the angle, the greater the distal displacement of the axial triradius.] In Penrose's (1954) initial study, the adult mean sum of atd angles for both palms was 137.5 degrees in mongols as contrasted with 85.5 degrees for the control sample.

Dermatoglyphic anomalies are documented for other full or mosaic autosomal trisomies or chromosomal structural aberrations: trisomy 8 mosaicism (Penrose 1972; Rodewald et al. 1977; Tuncbilek et al. 1972); trisomy 13 (Penrose 1966; Penrose and Loesch 1970; Uchida et al. 1962); trisomy 18 (Hecht et al. 1963; Penrose 1969a; Penrose and Loesch 1970; Uchida et al. 1962); deletions of the long or short arms of chromosome 18 (Mavalwala et al. 1970; Parker et al. 1972; Plato et al. 1971; Shaumann and Alter 1976; Passarge et al. 1970; Wolf et al. 1965); short arm deletion of chromosome 5--the cri du chat syndrome (Breg et al. 1970; Penrose and Loesch 1970; Schaumann and Alter 1976; Warburton and Miller 1967); and short arm deletion of chromosome 4--the Wolf-Hirschhorn syndrome (Miller et al. 1970; Mastroiacovo et al. 1976). Patients with full trisomy 18, for example, are unusual in their tendency to have arch patterns on nine or ten fingers.

Finger and palmar dermatoglyphics differ from the general population in sex chromosomal aberrations. Qualitative differences noted for Turner's syndrome, including cases resulting from mosaicism or structural aberration, include slight reductions in the frequencies of

arches and radial loops and a corresponding increase in the frequency of large ulnar loops. Large whorl patterns are also common on the fingers. Patterns in the third and fourth interdigital areas and large hypothenar patterns are elevated in frequency while thenar patterns occur less often than normal (Dallapiccola et al. 1972; Holt 1969; Holt and Lindsten 1964; Lindsten et al. 1963). Sex chromatin positive males (Klinefelters syndrome: 47XXY, 48XXXY, 48XXYY, 49XXXXY, 49XXXYY) show slight differences from controls as reflected primarily in a reduction in average total finger ridge-count (Alter et al. 1966; Cushman and Soltan 1969; Forbes 1964; Hubbell et al. 1973; Hunter 1968; Penrose 1963, 1967; Penrose and Loesch 1967; Saldãna-Garcia 1973; Uchida et al. 1964; Vormittag and Weninger 1972). Penrose (1963) attributes this reduction to decreasing pattern size although an increased frequency of arches is a related factor (Alter 1965; Schaumann and Alter 1976).

Parents and normal siblings of mongol children indicate tendencies of resembling Down's offspring dermatoglyphically (Holt 1970). The maximal atd angle of mothers and siblings of mongols show small but significant increases toward the mongoloid type (Penrose 1954). Priest et al. (1973) reported both fathers and mothers of Down's as showing "dermal microsymptoms" of mongolism using the Walker Dermal Index Score, a composite index based on digital patterns, position of the axial triradius, palmar interdigital patterns and the plantar hallucal areas.

Unusual dermatoglyphics have been reported in several disorders, as the de Lange syndrome (Ptacek et al. 1963; Smith 1966), the Ellis-van Creveld syndrome (Goor et al. 1965), and the Holt-Oram syndrome (Gall

et al. 1966), which are probably caused by single abnormal genes. Certain clinical disorders of unknown genetic or multifactorial causation also appear to have dermatoglyphic abnormalities. The list includes anencephaly (Hilman 1953), congenital heart disease (Sánchez Cascos 1964; Hale et al. 1961), idiopathic mental retardation (Fang 1950; Hirsch and Geipel 1960), schizophrenia (Beckman and Norring 1963; Rothhammer et al. 1971), the Smith-Lemli-Opitz syndrome (Schaumann and Alter 1976), the Rubinstein-Taybi syndrome (Berg et al. 1966; Robinson et al. 1966; Smith et al. 1970) and cerebral gigantism (Schaumann and Alter 1976).

III. DERMATOGLYPHICS IN CLINICAL DIAGNOSIS

The presence of unusual dermatoglyphics in a variety of clinical disorders was incentive for determining whether dermal ridges provide an effective diagnostic tool. Dermatoglyphics have certain advantages in this regard in being easily inspected (even for newborns) without patient trauma and the analysis requires no elaborate equipment (Alter 1966). Unfortunately abnormal dermatoglyphic patterns tend not to be pathognomonic of specific disorders. Patient-control differences involve only differences in the frequencies of specific traits. The differences are often fairly general representing some nonspecific effect produced by defective embryological growth (Mastroiacovo et al. 1976). Clinic samples frequently display considerable variation: "Conclusions derived from analysis of the dermatoglyphics of a group with a particular clinical abnormality may not apply to a given individual with the same clinical abnormality" (Alter 1966:39). Nevertheless

dermal configurations have diagnostic value for certain syndromes allowing preliminary determination of the condition or as support for a particular diagnosis.

As early as 1946, Cummins and Platou (1946) were able to diagnose Down's syndrome using palm prints with an accuracy approaching 90 percent. Since then, several diagnostic indices or discriminant functions have been proposed such as the Total Dermal Index, the Hopkins Composite Score, the Dermatoglyphic Nomogram or the Radboud Score (Bolling et al. 1971; Borgaonkar et al. 1971; Deckers et al. 1973a, b, c; Reed et al. 1970; Walker 1957, 1958). Using maximal atd angle alone, Penrose (1954) was able to classify correctly 88 percent of his sample of Down's cases. The Hopkins Composite Score provides accurate classifications regardless of sex or racial background (Borgaonkar et al. 1973). This method misclassified only 2.3 percent of a full trisomy 21 mongoloid and normal Dutch sample examined by Oorthuys and Doesburg (1974).

Penrose and Loesch (1971) have developed several two group discriminate functions aiding differential diagnosis of patients trisomic for D, E or G group chromosomes or having a 4-5 short arm (B_p). deletion chromosome. Separate discriminant functions were developed using finger, palmar or sole characters and one using variables from each of these sources. The discriminant for trisomy E has a very low probability of misclassification (1.5 percent). A high frequency of finger arches is so characteristic of trisomy E, that less than six arches or more than two whorls are evidence against its diagnosis (Preus and Fraser 1972). Of the original samples, only 4.8 percent

of the Down's cases, 8.9 percent of trisomy D and 14.5 percent of the cri du chat patients were misclassified by the Penrose and Loesch (1971) method.

Clinical diagnosis of sex chromosomal anomalies using dermatoglyphics seems less practical since differences in pattern frequencies are small. Penrose and Loesch (1971) estimated the probability of misclassification for Turners and XYY syndromes to be at least 15 percent. Dallapiccola et al. (1972) have, however, developed a scoring system for diagnosis of Turners syndrome which is based on skeletal morphology of the hand and five dermatoglyphic traits. Other researchers have suggested that the presence of patterns in the palmar hypothenar area is potentially useful in the differential diagnosis of XXY and XXYY chromosomal states (Uchida et al. 1964; Alter et al. 1966).

IV. DERMATOGLYPHIC STUDIES OF CLEFT LIP AND CLEFT PALATE

The dermatoglyphics associated with cleft lip and cleft palate have been examined in several studies. Researchers have hoped to determine whether the disturbances which cause clefts also affect the formation of dermal ridges. The presence of unusual dermal patterns in cleft patients would imply a fairly generalized growth disturbance rather than factors affecting only craniofacial development.

One of the earliest studies was Silver's (1966) comparison of pattern frequencies on the fingers, third interdigital area and hallucal area of 71 Boston white cleft lip and cleft palate patients with controls from the same locality. Chi square tests for sample differences were not significant. Achs et al. (1966) examined seven patients with

cleft palate and found unusual patterns in three. Two individuals had distally placed axial triradii, one also had a simian line, while the third patient had radial loops on fingers other than the second digit. These observations were considered significant since they are common in various syndromes. The frequency of a distal axial triradius in 50 cleft lip and cleft palate Russian subjects was 25.5 percent as compared with 13 percent for controls (Usoev 1972). Wittwer (1967) similarly noted distal displacement of the t triradius in 15 patients with clefts. The palmar interdigital areas also seemed somewhat unusual in showing duplication and atypical positioning of triradii. Dzuiba's (1972) study of 152 Polish cleft patients indicated reduced pattern intensity on the fingers of males. This reduction was attributed to an increased frequency of ulnar loops accompanied by fewer whorls. Females showed a decreased frequency of fourth interdigital patterns while the entire series (sexes combined) had higher frequencies of thenar patterns on the left palm and hypothenar ulnar loops on the right palm.

A comprehensive study of dermatoglyphics and oral clefts is provided in a dissertation by Potrzebowski (1974). Qualitative and quantitative dermatoglyphic data for 52 Mexican families (including parents, normal siblings and cleft probands) were systematically examined. Multivariate analysis was used to simultaneously compare these groups (cleft probands, normal siblings, mothers and fathers) with an unrelated control sample. A significant difference was present with two, total ridge-count (TRC) and average main line index, of the five variables considered contributing most to group differences.

Means for TRC were higher in cleft cases and family members than in controls. In another test, controls were excluded while probands, siblings and parents were simultaneously compared. Differences within families and especially between normal siblings and cleft cases could reflect the presence of environmental factors affecting normal development of the lip and palate. The test statistics from this comparison was not significant. In tests of qualitative variables, differences between controls and the families were noted in finger and palmar pattern frequencies, palmar main line terminations and abnormal flexion creases. Third interdigital patterns were more common in the clinic sample. Cleft probands differed from their relatives in the frequencies of simian creases, extra digital triraddi and main line C terminations.

A few researchers have analyzed data for cleft palate (CP) and cleft lip with or without palate (CL±P) separately (Adams and Niswander 1967; De Bie et al. 1977; Gall et al. 1973; Piatkowska and Sokolowski 1972, 1973; Potrzebowski 1974; Woolf and Gianas 1976, 1977). This approach is necessary since etiological differences have been suggested for these types of clefts (Fraser 1970).

De Bie et al. (1977) were unable to detect any significant differences between controls and patients having CL±P or CP. A large number of dermatoglyphic features were examined including finger pattern frequencies, TRC, palmar pattern frequencies (hypothenar, thenar and interdigital areas), a-b ridge-count and main line terminations. The statistical comparisons were performed using chi square tests for significance of the differences.

Potrzebowski (1974) repeated her multivariate analysis excluding CP patients and family members. The values for TRC are higher in CL±P patients than those with CP. A study (Piatkowska and Sokolowski 1972, 1973), comparing the dermatoglyphics of cleft primary palate patients with cleft secondary palate patients using a large number of variables (including finger pattern intensity, TRC, palmar pattern intensity, interdigital ridge counts, and atd angle) revealed only two differences. For example, females with clefts of the primary palate have fewer patterns in the fourth interdigital area. Because many variables were considered, Piatkowska and Sokolowski attributed the results as due primarily to chance. They concluded that dermatoglyphics are largely invariant with respect to cleft of the primary or secondary palate. Gall et al. (1973) did report increased frequencies of unusual dermatoglyphics in children having oral clefts plus additional anomalies relative to those without associated malformations. Frequencies of unusual palmar flexion creases, missing digital or axial triraddi, imperfect ridge formation and increased wrinkling almost doubled in children having additional dysmorphogenetic features. The effect was most pronounced in children with CL±P. Fluctuating asymmetry, a form of bilateral asymmetry measured as the within-pair variance between hands of a single individual, is increased for clef ipropositi relative to normal controls. Adams and Niswander (1967) and later Woolf and Gianas (1976, 1977) found increased asymmetry for atd angle, a-b interdigital ridge-counts and finger patterns of CL±P propositi with a family history of oral clefts (FH+). Patients with CP or CL±P with no history of clefts (FH-) exhibited little differences from normal. First degree relatives

of CL±P propositi with positive family history also show increased asymmetry, although this effect was missing in normal siblings and parents of cleft cases lacking a positive history (Woolf and Gianas 1977).

Only Gall et al. (1973) have classified cleft patients according to presence or absence of additional craniofacial or clinical anomalies. The dermatoglyphics of patients with Pierre Robin syndrome, a syndrome including cleft palate as a characteristic feature, has been studied although no differences were noted (Brehme and Härle 1971).

Researchers have reported a few dermatoglyphic abnormalities in patients with oral clefts. Variables as TRC and atd angle are the frequencies of certain palmar patterns seem to show differences from normal. Increased fluctuating asymmetry in dermal traits of CL±P propositi was noted in three studies. First degree relatives of CL±P probands with a history of clefts similarly expressed this tendency. Nevertheless, it also seems apparent that the differences from normal are relatively small. The results across studies are by no means consistent with no differences being reported in some instances. Part of this inconsistency derives from variation in the variables selected for analysis. Part derives from the statistical methodology used for analysis. For example, in Silver's study (1966) the comparison of finger patterns was accomplished by combining whorls with arches and radial loops with ulnar loops to allow adequate chi square cell sizes. It was suggested that these combinations reflect groups having similarity in pattern formation. This justification is questionable if one considers pattern size or the radial/ulnar differentiation indicated by factor analytical studies. (Holt 1968a; Roberts and Coope 1975;

Jantz and Owsley 1978). For example, whorls have two triradii and ridge-counts with values greater than zero; arches lack triradii and have ridge-counts of zero.

In general, there is a tendency in the previous studies to examine large numbers of variables in a univariate context rather than handling the data simultaneously with a multivariate approach. When many variables are tested individually, it is difficult to assess the significance of the findings. A difference in only one or two variables may reflect chance. Multivariate procedures allow simultaneous confidence limits for individual variables. The analysis (Potrzebowski 1974) applying multivariate tests did find significant differences between controls and cleft families. Here, however, it is again difficult to evaluate the results. Controls were simultaneously compared with cleft cases, their normal siblings and parents. It is unclear which or how many of these groups significantly differ. Paired comparison tests provide a more efficient means of comparing family members since these samples are not random in the statistical sense. Only a few studies have examined the dermatoglyphics of parents or siblings of cleft propositi. The present study provides a multivariate analysis of finger and palmar quantitative ridge-count data of cleft lip and cleft palate patients and first degree relatives. CL$\pm$P and CP patients are compared with controls in separate analyses.

CHAPTER II

POSSIBLE INTERRELATIONSHIPS BETWEEN FACIAL CLEFTS AND DERMATOGLYPHICS

I. CLEFT LIP AND CLEFT PALATE

Combinations of four basic structures being affected completely or incompletely are involved in the formation of oral-facial clefts; the lip, alveolus, hard palate and soft palate (Santiago 1969). Nevertheless, the malformation should not be considered a localized defect as the impairment adversely affects development of related structures (Berndorfer 1970; Pruzansky 1975). Associated craniofacial anomalies involving organs contiguous to the maxilla are often reported for facial clefts (Bishara and Iverson 1974; Coccaro and Pruzansky 1965; Krogman et al. 1975; Ross 1965).

Several classifications have been proposed as a means of systematizing the various types of clefts. A taxonomy frequently employed was proposed by Veau (1931) and Fogh-Andersen (1943). It recognizes three basic cleft types: cleft lip (CL); cleft palate (CP); and cleft lip and cleft palate (CL+P). A more comprehensive system proposed by the American Cleft Palate Association distinguishes two major types: (1) prepalate clefts involving the lip and alveolar process, and (2) palate clefts affecting the palatal shelves of the maxillae, the horizontal processes of the palatine bones, the vomer and the soft palate (Harkins et al. 1962). The incisive foramen is considered the point of demarkation. Prepalate clefts have unilateral

or bilateral occurrence. When unilateral, the left side tends to be involved about twice as frequently as the right (Fraser 1970). Prepalate clefts may have an associated palate cleft (CL±P); this is the case in 68 percent of patients with unilateral and 86 percent with bilateral cleft lip (Fraser 1970). Observed frequencies to prepalate clefts (with or without involvement of the palate) to palate clefts follow an approximate ratio of 7:3 (Biggerstaff 1969).

The incidence of CL±P displays considerable variation among racial groups. Such differences remain even where populations of different races are sympatric (Morton et al. 1967). Highest rates are reported for Mongoloid population samples including American Indians. Negro populations have a low frequency while Caucasoids express rates in an intermediate range. The average occurrence in Caucasians is slightly higher than 1 per 1000 births (Azaz and Koyoumdjisky-Kaye 1967; Conway and Wagner 1966; Erickson 1976; Gilmore and Hofman 1966; Saxen and Lahti 1974; Stevenson et al. 1966). Neel (1958) reported an incidence of 2.13/1000 for Japanese births and Emanuel et al. (1972) calculated a similar rate of 1.92/1000 in an epidemiological survey of congenital malformations in hospitals of Taipei, Taiwan. Rates for North American Indian tribes have been observed as high as 1:415 births, though the overall incidence (1.7/1000) is intermediate between Japanese and Caucasians (Bardenouve 1969; Jaffe and De Blanc 1970; Niswander et al. 1975). For North American Negroes, the rate is close to 0.34-0.41 per 1000 (Altemus 1966; Altemus and Ferguson 1965; Gilmore and Hofman 1966; Millard and McNeill 1965).

Isolated cleft palate shows less racial variation than CL±P and has a larger degree of variation between populations of the same primary race (Fraser 1970; Leck 1972). The range of occurrence in various studies is approximately 0.3 to 0.7 per 1000 births (Altemus 1966; Ching and Chung 1974; Chung and Myrianthopoulos 1968; Conway and Wagner 1966; Erickson 1976; Jaffe and De Blanc 1970; Morton et al. 1967; Neel 1958; Niswander et al. 1975; Saxen and Lahti 1974).

The incidence of facial clefts is unequal in males and females. In Caucasian samples, the ratio for CL+P suggests male predominance with ratios varying between 1.9/1 to 2.3/1. The difference is less for isolated cleft lip (Bardanouve 1969; Gilmore and Hofman 1966; Leck 1972; Ross and Johnston 1972; Saxen and Lahti 1974; Schilli et al. 1969). Such differences may prove race specific since epidemiological surveys reveal contrasting sex ratios for Mongoloid and Negro samples (Emanuel et al. 1972; Greene et al. 1965). In one Chinese series, Emanuel et al. (1972) found a slight excess of females with CL±P. Isolated cleft palate occurs more frequently in females irrespective of race (Bardanouve 1969; Conway and Wagner 1966; Greene et al. 1965; Leck 1972; Saxen and Lahti 1974).

Sex differences have been noted in the severity or completeness of facial clefts (Mazaheri 1958). Using pooled data from Fogh-Andersen (1943), Knox and Braitwaite (1963) and Mazaheri (1958), Meskin et al. (1968) demonstrated a higher proportion of severe clefts involving the hard and soft palate in females. In another comparison, Meskin et al. (1968) found 32 percent of Caucasian proband females as having complete clefts of the lip whereas only 9 percent of Caucasian proband

females as having complete clefts of the lip whereas only 9 percent of males were complete. Sixty-eight percent of females and 58 percent of males had complete clefts of the lip and palate.

Individuals with clefts have higher than normal incidences of additional congenital malformations besides those considered an integral part of the clefting process. This frequency difference decreases with increasing age level of the clinical samples examined, being highest in fetal abortuses (CL±P 11-20 percent; CP 13-23 percent) and lowest in clinical treatment samples (CL±P 3-7 percent; CP 4-24 percent)(Bear 1973; Drillien et al. 1966; Greene et al. 1965; Meskin and Pruzansky 1969; Ross and Johnston 1972; Saxen and Lahti 1974; Tolarova´ 1970).

Gross anatomical studies of cleft human abortuses have revealed high frequencies of external malformations as club hands or feet, brachydactyly, syndactyly, polydactyly, malformations of the ears or eyes, agenitalia, anal atresia and skeletal dysplasia (Kraus et al. 1963). Visceral abnormalities are more severe and frequent in fetuses with oral clefts than noncleft abortuses (Kitamura and Kraus 1964). The frequency or types of visceral malformations were not found to be associated with the type of cleft. However, a difference in frequency was noted for external malformations with 77 percent of CL±P and 50 percent of CP having additional anomalies (Kitamura and Kraus 1964; Kraus et al. 1963).

Most studies report higher incidences of additional anomalies with CP than with CL±P and a greater association of defects with CL±P than CL in newborn babies (Bardanouve 1969; Bear 1973; Conway and Wagner

1966; Gilmore and Hofman 1966; Green et al. 1965; Meskin and Pruzansky 1969; Pannbacker 1968; Spriestersback et al. 1962). Siblings of CP propositi also have higher malformation rates than siblings of CL or CL±P probands (Meskin and Pruzansky 1969). Probands with isolated CP have higher infant mortality rates than those with CL±P (Bardanouve 1969; Mackeprang and Hay 1972).

Reports of sex differences in incidence of accompanying malformations are inconsistent. There tends to be an excess of male propositi with additional defects (Bear 1973: Pannbacker 1968) although the reverse was found in one study (Meskin and Pruzansky 1969).

Especially high numbers of defects found in fetal and newborn studies are at least partially because fetuses with tirsomies or other chromosomal aberrations are included in samples (Ross and Johnston 1972). The effect of selection through spontaneous abortion, stillborn and neonatal death rapidly reduces the number of individuals with multiple or severe defects (Kitamura and Kraus 1964; Mackeprang and Hay 1962; Ross and Johnston 1972). Thus in clinical samples, patients with severe multiple defects caused by chromosomal aberration or other factors are generally missing. The incidence of CL±P patients with anomalies involving the hands still occurs with approximately five times the frequency of the general population and those involving the heart and feet are double the expected incidence (Ross and Johnston 1972).

Abnormalities of the deciduous and permanent dentitions such as supernumerary teeth, agenesis, malposition and anomalous crown morphology are frequently associated with cleft lip and/or palate (Ehmann et al. 1976; Zilberman 1973). Jordan et al. (1966) found 54.3 percent of a postnatal cleft sample to have dental abnormalities; 81 percent of these showed multiple incidence. These developmental irregularities were not considered entirely attributable to direct effects of the clefting process since even teeth far removed from the cleft were anomalous.

II. EMBRYOLOGICAL DEVELOPMENT OF THE PRIMARY AND SECONDARY PALATE

The midface and palate are formed through differentiation and interaction of the frontonasal and maxillary processes. Early in the fifth week, mesoderm covering the forebrain thickens to form the frontonasal process. On each side of this structure two depressions, the nasal placodes, begin receding into the underlying mesoderm forming the primitive nasal cavity and ultimately contacting the buccal cavity (Fitzgerald 1978). On the sides of the nasal placode, median and lateral nasal processes develop by proliferation of frontonasal mesoderm. Downward growth of the frontonasal process results in formation of the bridge of the nose and dorsal growth of the lower end of the frontonasal process forms the primary palate, the precursor of the premaxilla and its alveolus. The median nasal processes form the lower part of the nasal septum, the lateral nasal processes form the alae of the nose (Fitzgerald 1978).

The upper lip forms from the frontonasal and maxillary processes. The maxillary processes are derivatives of the first branchial arches. They join the median nasal processes below the nostril and the lateral nasal processes along lines of contact extending from the eyes to the nostrils. The appearance of the labiogingival sulcus during the eighth week separates the lip from the premaxilla and maxilla (Longacre 1970).

Development of the primary palate precedes that of the secondary palate. The hard and soft palate posterior to the incisive canal develop from the maxillary processes. Primordial palatal shelves first appear as swellings and then ridges on the surfaces of the maxillary processes during the sixth week (Fitzgerald 1978; Kitamura 1966). With enlargement, the shelves are deflected downward in a vertical orientation by a highly placed tongue in contact with the nasal septum. The shelves remain adjacent to the tongue until the eighth week when they assume a horizontal position in order to close. Transposition requires displacement of the intervening tongue and involves coordinated interaction of movements and growth of the palatal shelves, tongue, head and mandible (Fraser 1968). The dynamics of shelf elevation remain unclear although several mechanisms have been suggested. Those intrinsic to the shelves concern cellular proliferation, differential growth, vascular development and the synthesis of acid mucopolysaccharide (Fraser 1968; Gregg and Avery 1971; Nanda and Romeo 1975). Mechanical forces affecting tongue displacement through mandibular growth, muscular activity, or reduction in cervical flexure may also prove important in human palatal

morphogenesis by permitting or assisting shelf elevation (Burdi and Silvey 1969b; Fraser 1968; Ross and Lindsay 1965).

The epithelial surfaces of the midanterior component of the shelves first meet in the midline. After approximation, the epithelial seam between the processes breaks down and there is eventual consolidation by mesenchymal penetration (Kitamura 1966; Ross and Johnston 1972). Shelf fusion is complete by the tenth week (43.0 mm embryo) (Longacre 1970). The palatal shelves also fuse with the nasal septum.

Histological examination of human embryos during the stages from initial formation of the palatal shelves to completion of the secondary palate (7 through 10 weeks) reveal timing differences between males and females (Burdi and Silvey 1969a). Male palatal shelves attain horizontal positions at an earlier developmental stage and are more advanced in closure than females of the same age. "Trends indicate that the critical period of palatal closure for the male is the seventh week (25 mm crown-rump length) as compared to the mid-eighth week for the female embryo" (Burdi and Silvey 1969a:6).

Clefts of the primary palate are caused by interruption or lack of fusion of the median nasal, lateral nasal and maxillary processes (Fraser 1968). Ross and Johnston (1972) consider the lack of closure as primarily resulting from mesenchymal insufficiency caused by factors affecting the source, mitotic activity or distribution of facial mesenchyme.

Clefts of the secondary palate reflect a lack of fusion or post-fusion rupture of the palatal shelves. Possible causes include:

(1) delayed shelf movement or lack of elevation (Fraser 1968; Vij and Kanagasuntheram 1971); (2) shelf abnormality (Fraser 1968); (3) abnormal or excessive head or maxillary arch width (Smiley et al. 1971); (4) failure of shelf fusion (Smiley 1972); and (5) tissue deterioration following palate fusion with eventual rupture (Kitamura 1966; Kraus 1970; Lejour 1970). Embryological studies of rodents indicate that cleft palate in association with cleft lip may be a secondary consequence of the primary palates defect, not an intrinsic defect of the secondary palate (Trasler and Fraser 1963). Growth distortions associated with clefts of the primary palate decrease the likelihood of closure of the secondary palate. The severity or completeness of a cleft relates to the morphological point at which development was disturbed (Ross and Johnston 1972).

III. ETIOLOGY OF CLEFT LIP AND CLEFT PALATE

The etiologies of cleft lip with or without cleft palate and isolated cleft palate are heterogeneous. Both genetic and embryological data indicate CL±P and CP to be genetically and developmentally distinct (Curtis et al. 1961; Fraser 1970; Trasler and Fraser 1963; Woolf et al. 1963; Woolf 1971). In both types, a small percentage of cases are attributed to the effects of major mutant genes, chromosomal aberration or specific environmental agents. In this regard, more than 100 syndromes are recognized as including oral clefts as an associated abnormality (Gorlin et al. 1971). Less than 3 percent of CL±P are believed syndromic in origin although the incidence may be somewhat higher for CP (Fraser 1970). The majority of cases belong to a

multifactorial group encompassing the interaction of a large number of genetic and environmental factors.

A small number of CL±P cases occurs in syndromes caused by major mutant genes as the lower lip fistula, the oral-facial-digital and popliteal web syndromes (Smith 1970). Clefts of the lip and/or palate were noted in approximately 56 percent of patients expressing dominantly inherited lip-pits (Van der Wonde 1954). Isolated cleft palate frequently occurs in oral-facial-digital syndrome (autosomal dominant inheritance) and oto-palato-digital syndrome (probable autosomal recessive). CP appears in approximately 10 percent of patients with Cornelia De Lange syndrome (etiology unknown), 40 percent of those with Smith-Lemli-Opitz syndrome (probable autosomal recessive) and 25 percent of patients with distrophic nanism (autosomal recessive) (Fraser 1970; Smith 1970).

Complete bilateral cleft lip and palate is a nearly constant association with full D_1 trisomy (Subrt et al. 1966). Cytogenetic studies have also shown clefts in translocations resulting in D trisomy, mosaic D trisomy and partial D trisomy (Loevy et al. 1975). Cleft lip with or without cleft palate frequently accompanies the No. 4 short arm deletion syndrome and has occasional occurrence in the chat syndrome (Fraser 1970; Smith 1970). Isolated cleft palate is occasionally associated with trisomy 13, trisomy 18, No. 4 short arm deletion syndrome, No. 18 long arm deletion syndrome and the XXXXY syndrome (Smith 1970).

Exogenous factors are involved in the etiology of oral clefts although their exact nature is undefined and may differ by cleft type.

Many terotagenic agents are known to cause clefts in rodents including riboflavin deficiency, folic acid deficiency, vitamin B-12 deficiency, deprivation of water, deficiency of other nutrients, hypervitaminosis A, trypan blue, ionizing radiation, hormones, amniocentesis, salicylates, barbituates and tranquilizers, antibiotics, chlorcyclizine, 6-amino-nicotinamide, pteroylglutamic acid, and transportation during gestation (Brown, Johnston and Murphy 1974; Brown, Johnston and Niswander 1972; Kraus 1970; Lejour 1970; Nanda 1974; Walker and Patterson 1974).

In humans, specific environmental teratogens are implicated as causing clefts in syndromes induced by rubella, thalidomide and possibly aminopterin (Fraser 1970; Smith 1970). Affected children have been reported for women given high doses of cortisone early in pregnancy (Harris and Ross 1956). Other investigators suggested an association between clefts and: (1) miscellaneous drugs including antiemetics, anticonvulsants and antinuerotic drugs, (2) nausea and vomiting in pregnancy, (3) maternal bleeding, (4) toxemia, (5) increased maternal antagonism to insulin and (6) toxoplasmosis antibodies (Niswander and Wertelecki 1973; Pashayan et al. 1971; Richards 1969; Safra and Oakley 1976; Spriestersbach et al. 1973; Wilson 1977b). In general, however, specific drugs or environmental chemicals cannot be identified in the majority of cases (Kraus 1970). Teratologic agents used to produce clefts in rodents are not considered of major importance to humans because of their limited distribution and low dosages currently in circulation.

The majority of clefts are attributed to multifactorial (polygenic) inheritance with a threshold effect (Carter 1977; Fraser

1970). The variables responsible for oral-facial development follow a continuous distribution with abnormal expression resulting when individuals surpass a certain developmental threshold. The trait presents a discontinuous distribution being either present or absent. Factors influencing cleft formation are multiple and individually indistinguishable involving the interaction of both the genotype, as genetic predisposition, and the environment.

The multifactorial model has certain expectations concerning trait frequencies in the population and near relatives of propositi compatible with CL±P data. These include (1) a frequency in first degree relatives of probands equaling the square root of the population incidence, (2) rapid predictable decreases in incidence as the degree of relationship to the proband decreases, (3) increased risks for siblings when more than one are affected, and (4) increased risks of recurrence with increased severity of the defect (Fraser 1970; Tanaka et al. 1969; Woolf 1971).

The familial pattern shows recurrence risks of approximately 4.0 percent (CL±P) and 3.0 percent (CP) for siblings of index patients (Curtis et al. 1961; Fraser 1970; Fuhrman and Vogel 1969; Woolf 1971). If two siblings are affected the risk for subsequent siblings increases to about 9 percent (CL±P) and if one parent and one child are affected the risk for additional siblings is 15 percent (CL±P and CP). Reoccurrence rates vary with sex of the proband being higher for female CL±P probands and higher for male CP probands (Fraser 1970; Tanaka et al. 1967; Woolf 1971; Woolf et al. 1964). Concordance rates for monozygotic twins are about 38 percent in CL±P and 24 percent in

CP (Douglas 1958; Fraser 1970; Metrakos et al. 1958; Pruzansky et al. 1970).

Multifactorial determination is considered best established for CL±P. It probably also applies to isolated cleft palate, though the proportion of multifactorial cases to other causes may be smaller (Burdi et al. 1972). Alternative modes of inheritance are not entirely excluded although monogenic systems involving dominant or recessive inheritance are less likely. Nevertheless, there are difficulties in discriminating between genetic models involving single loci with multiple parameters, as degree of dominance and variable penetrance, and multifactorial inheritance (Chung et al. 1974). Melnick et al. (1977) reported genealogical data for CL±P and CP not conforming to predictions of a multifactorially determined trait. These researchers consider allelic restriction as an alternative genetic explanation.

A few studies report increasing parental age as having a positive affect on the incidence of CL±P (Fraser and Calnan 1961; Green et al. 1965; Hay 1967; Woolf et al. 1963) and CP (Bardanouve 1969; Greene et al. 1965; Hay 1967; Leck 1972). However, others have found no association between clefts and parental age (Ching and Chung 1974; Perry and Fraser 1962; Spriestersbach et al. (1962). Donahue (1965) found a slightly increased incidence of clefts associated with pleural births than single births.

The presence of maternal uterine factors affecting the frequency of clefts has been demonstrated in mice (Davidson et al. 1969). No evidence of this has been found in humans (Bingle and Niswander 1977; Ching and Chung 1974).

IV. EMBRYOLOGICAL DEVELOPMENT OF DERMAL RIDGES

Limb plates representing initial development of the hands are present by the fifth gestational week with five mesenchymal proliferations in each, the digital rays (Fitzgerald 1978). The rays undergo chondrification and ossification forming metacarpals and phalanges. The mesenchymal webb joining the rays soon degenerates resulting in separate digits.

Development of the dermal ridges begins in the sixth to seventh week and is essentially complete by the nineteenth week. Ridges are not discernible histologically until the fetus reaches a crown-rump length of at least 70 mm (Miller 1968). The chronology and embryological sequences presented in the following discussion derive from Babler (1978b) and other sources (Hale 1949, 1952; Holt 1970; Mulvihill and Smith 1969; Okajima 1975).

Elevations of mesenchymal tissue (fetal pads) appear during the sixth week first on the palms and then on the fingertips as sites of initial epidermal ridge formation. The pads enlarge in size becoming prominent through mitotic proliferation until the tenth week. In relation to overall fetal development, pad formation corresponds to the period or organogenesis. The volar pads then normally regress in relative size during the tenth to twelfth weeks while ridge formation begins. Primary dermal ridges develop at the dermal-epidermal juncture by projection of the epidermal stratum basalis into the dermis. The epidermal surface remains smooth. Ridge formation occurs first on the digits and later on the palms. After the ridges have covered the

volar surfaces and the general pattern configuration determined, the ridges undergo multiplication and increases in breadth in keeping up with surface growth. Ridge multiplication ceases in the 15-17 weeks. Contours of the primary ridges within the epidermal-dermal junction become reflected on the epidermal surface through deposition of keratin. Further differentiation in the fifth and sixth months produces secondary dermal ridges and dermal papillae.

The orientation of the ridges, and the patterns formed by them, has been related to the size and symmetry of the fetal pads (Mulvihill and Smith 1969).

> The final pattern may be interpreted as the consequence of the height and contour of the pad at the critical time when ridges are developing, a low pad with little disruption of the parallel lines resulting in an arch, a high pad giving a whorl, and a pad of intermediate height producing a loop. Furthermore, a pad with a steeper side on the radial aspect of the fingertip would lead to an ulnar loop (Mulvihill and Smith 1969:584).

Penrose (1969b) has suggested that the ridges follow the greatest positive curvature of the fetal pad's surface.

The levels of mitotic activity involved in fetal pad formation, the rate and timing of pad regression, and the rate and timing of primary ridge formation are important variables involved in pattern formation. An embryological study by Babler (1978a) illustrates the effects of early ridge differentiation as resulting in an increased frequency of whorls. Late ridge differentiation leads to an exceedingly high frequency of arches.

V. QUANTITATIVE GENETICS OF DERMAL RIDGES

The importance of heredity in the formation of dermal ridges was recognized in some very early studies (Bonnevie 1924; Galton 1892). Since then, considerable attention has been given to the genetics of quantitative ridge-counts and particularly total finger ridge-count (TRC). Beginning in 1952, Holt (1952, 1956, 1957a, 1957b) initiated a series of articles suggesting polygenic additive inheritance for total ridge-count. Evidence for multifactorial causation is based on a continuous frequency distribution and the agreement between observed familial correlations and theoretical expectations. Under assumptions of polygenic additivity and random mating, trait correspondence between relatives is determined by the proportion of genes shared in common (Fisher, 1918; Penrose 1949). Parent-child and sib-sib correlations have expected values of 0.5. Observed values in Holt's (1956, 1957a) English sample were 0.48 and 0.50. Predicted correlations of monozygotic and dizygotic twins are 1.0 and 0.5 respectively and again, sample values of 0.95 and 0.49 closely agree (Holt 1957b). Familial correlations for separate digital counts are generally lower than for TRC (Holt 1968a). However, the heritability of an overall "size" factor, which derives from the ten counts analyzed within a multivariate context is extremely high (0.97) (Rostron 1977). Estimates of genetic variance obtained from twin sets showed significant genetic components for all digital patterns and ridge counts except those of the thumb (Reed et al. 1975). Holt (1968a) concluded that only 5 percent of the variation in TRC derives from environmental effects (from the

twin-twin correlation). Other studies, however, have reported lower correlations than obtained by Holt (Froehlich 1976; Loesch 1971; Matsuda 1973). Loesch (1971) found sib-sib and parent-child correlations centering around 0.4. The differences are attributed to greater heterogeneity of Holt's family data which includes primarily English, but also Welsch and Jewish families. Loesch (1971), for example, was able to show significantly smaller variances for her Polish sample. Froehlich (1976) estimates the heritability of ridge-counts to be in the range of 60 percent to 80 percent indicating a far greater environmental component than initially considered. Froehlich believes a large part of the non-heritable component of dermatoglyphics represents accidents or chance events during development. Variation in the heritabilities of different traits reflects relative degrees of canalization.

The number of genes involved in determining TRC is generally considered to be few because of negative skewness in its frequency distribution (Holt 1955, 1968a). Matsunaga (1972) has estimated the minimum number of loci involved as about six. Familial correlations and tests of regression linearity of child or midparental values have revealed no signs of dominance, sex linkage, or maternal effect for TRC (Holt 1968a; Matsuda 1973; Penrose 1967).

Segregation of a major gene may be involved in the determination of TRC as certain evidence supports this possibility. Holt's (1958) examination of the regression of S^2, a measure of interfinger diversity in ridge-counts, on TRC showed nonlinearity and suggestions of trimodality. Holt concluded, however, that if the distribution was resolvable

into three phenotypes representing genotypic variation, environmental influences and modifier genes obscured their presence. The shape of the regression was instead interpreted in terms of overdominance relating to effects of gene heterozygosity and homozygosity. The distribution of scores in Knussman's (1969) "radiomedial" factor similarly indicated trimodality. Unfortunately, attempts at finding clear patterns of segregation within families proved inconclusive. The presence of a major gene is more clearly revealed in Jantz's (1977a) examination of the relationship between mean total ridge-count and its variance. When a large part of the variation of a quantitative trait is determined by alleles segregating at a single locus, the traits's variance presents a parabolic relationship to the mean. Using a quadratic regression formula, Jantz was able to demonstrate this type of curvilinear dependency for mean total ridge-count and its standard deviation. Spence et al. (1973) found it possible to account for as much as 60 percent of the variation in absolute ridge-count (a variable highly correlated with TRC) assuming a major gene with two segregating alleles.

The method of familial correlations has been applied to other quantitative traits as the finger diversity index (S/ 10) (Holt 1960), pattern intensity (Mukherjee 1966), atd angle (Penrose 1954), and interdigital ridge-counts (Pateria 1974; Pons 1964). All indicate high heritability attributable to additive genes although sib-pair and parent-child correlations are generally lower than 0.5, implying the presence of prenatal environmental effects. The heritability of

interdigital patterns has been investigated using the Penrose-Loesch topological classification system (Loesch 1971). Loops in the second interdigital area show high heritability. Loesch considers autosomal recessiveness a possible mode of inheritance for these patterns. Third and fourth interdigital areas have lesser amounts of genetic influence (Froehlich 1976; Loesch 1971; Reed et al. 1975). The position and size of interdigital patterns appears more susceptible to nongenetic modification than finger ridge-counts (Glanville 1965). Overall, environmental factors appear to affect palmar configurations to a greater extent than those on fingers. This may reflect the fact that ridges on palms develop later (Okajima 1975).

The number of sex chromosomes, both in normal and abnormal states, systematically affects the development of dermal ridges. For total ridge-count, this relationship has been described using the regression formula TRC = 187 - 30X - 12Y (Penrose 1967). With increased numbers of X or Y chromosomes, total ridge-count becomes smaller. Moreover, an extra X has a more pronounced effect (reduction by 30 ridges) than an additional Y (minus 12 ridges). Ridge breadth and the number of palmar patterns also decrease with additional X or Y chromosomes (Barlow 1973; Penrose and Loesch 1967; Saldaña-Garcia 1975). Reductions in ridge breath are probably somewhat responsible for the decrease in finger ridge-counts.

In terms of finger pattern types, the influence of the sex chromosomes are partly exemplified in increased numbers of large whorls and a shortage of arches and radial loops in Turner (XO) females (Holt and Lindsten 1964). Patterns are smaller in triple X females and

radial loops and arches are more common than normal (Saldaña-Garcia 1075). Even normal sex differences, as characterized by increased frequencies of whorls in males and arches in females, fall within the predicted framework.

This reduction in the number of ridges has been attributed to reductions in growth rate caused by heterochromatic sex chromosomes (Barlow 1973). Studies of cell kinetics show the presence of additional sex chromosomes retards rates of mitotic division. Barlow (1973) postulates increased mitotic rates of Turner females results in larger or higher volar pads and subsequently increased ridge counts. Slowed mitotic rates in multiple X anomalies produces lower fetal pads and smaller patterns.

Mittwoch (1969) believes the the Y chromosome regulates developmental rates and may increase the number of mitoses per given unit of time. Using embryological data from rat studies, Mittwoch et al. (1969) found evidence for Y chromosomal determination of sex by accelerating male gonadal development. The volume of rat gonads is larger in male embryos than in females. If the Y chromosome similarly affects the size of other tissues including the fetal pads, it could help explain determatoglyphic differences between males and females. Present evidence suggests the sex chromosomes have a significant role in dermal ridge development. Interpopulation differences and normal sex differences may be explainable with reference to the X and Y chromosomes (Jantz 1977b).

VI. INTERRELATIONSHIPS BETWEEN FACIAL CLEFTS AND DERMATOGLYPHICS

The following discussion explores possible interrelationships between facial clefts and dermatoglyphics. The objectives are to describe how dermatoglyphic data may provide information concerning developmental mechanisms involved in the formation of oral clefts, or why dermatoglyphic patterns of patients could differ from normal. The underlying theme for the discussion rests on the assertion that dermatoglyphics are not isolated phenomena. They are influenced by rates of growth and environmental factors affecting overall fetal development. Some of the same growth factors regulating dental and skeletal development appear to be important in dermal ridge formation including those affecting patterns of intraindividual variation and sexual dimorphism (Jantz 1977c). Webb's (1977) analysis, finding significant interrelationships between dermal ridges and size of the permanent dentition (even though secondary dental development is essentially postnatal), leads to similar conclusions.

Babler's (1978a, 1978b) comparisons of pattern frequencies in spontaneous abortions lacking overt clinical abnormality and elective abortuses indicate dermatoglyphics are interrelated with overall fetal development. Spontaneous abortuses have high frequency of arches and shallower primary ridges indicating less penetration into the dermis. Elective abortuses are developmentally more advanced in ridge maturation than spontaneous ones of comparable crown-rump length. The differences are thought to reflect the affects of stabilizing selection on factors

associated with ridge differentiation and particularly those involving developmental timing. Pattern formation involves interrelationships between growth of the volar pads, subsequent regression and ridge differentiation.

> Selection in this case, then, appears to involve deviations, including timing of ridge differentiation, from a common developmental pathway. Whether the high frequency of arches reflects early pad regression or late ridge differentiation can not yet be determined. An alternative explanation may be that pad height is reduced in fetuses that spontaneously abort. In either case, selection during the fetal period appears to involve deviations, including developmental timing and/or pad height, from a common developmental pathway (Babler 1978a:26).

The developmental imbalance is probably not specific to the formation of dermal ridges. In some way, it interrelates with the coordination or timing of other developmental processes or growth throughout the fetus since spontaneous abortion occurred. Dermatoglyphics, as products of developmental processes, seem potentially useful for investigating birth defects involving deviations from normal developmental sequences. In relation to facial clefts, development of the face and closure of the palate requires the coordinated interaction of several embryological structures. Cleft lip and cleft palate may represent poor synchronization in the growth of those structures. Such an imbalance (poor synchronization), if present throughout the fetus, may be reflected in other structures as dermal ridges. The presence of additional congenital malformations in CL±P and CP seems suggestive of a more generalized imbalance.

The association of additional malformations involving the hands, as polydactyly, syndactyly and brachydactyly, suggests another reason

why dermatoglyphic differences may occur. Abnormal ridge arrangements are associated with all malformations of the hands (Holt 1968a).

Reduced birth weight tends to accompany facial clefts. Green et al. (1964) reported lower birth weights for cleft children with 14 percent of the sample less than 2,500 grams as compared with 6 percent for controls. When births below 2,500 grams were tabulated according to cleft type, 8 percent were associated with isolated CL, CL±P, 15 percent and CP, 18 percent. Fraser and Calnan (1961) and Lutz and Moor (1955) reported lower birth weights for isolated cleft palate proband than those with cleft lip or cleft lip and palate. Meskin (1966) noted lower birth weights for cleft patients with patient-control differences significant for CP and CL±P but not cleft lip alone. Females, but not males, indicated possible weight heterogeneity for different types and degrees of clefting.

Lower average birth weights possibly signify slower rates of fetal growth. If the effect is general, such that even sizes of the volar pads are affected, dermatoglyphic differences could arise. Present evidence suggests a direct relationship between size of the volar pads and the patterns formed. Smaller pads (the predicted result of slow intrauterine growth) produces smaller patterns with lower ridge counts. If environmental teratogens are responsible for some cleft lip and/or cleft palate cases, the effects of the teratogen could simultaneously alter dermal ridge development. Wilson (1977a) summarizes the initial types of changes induced in developing cells or tissues by teratogenic insult in the following categories: (1) mutation,

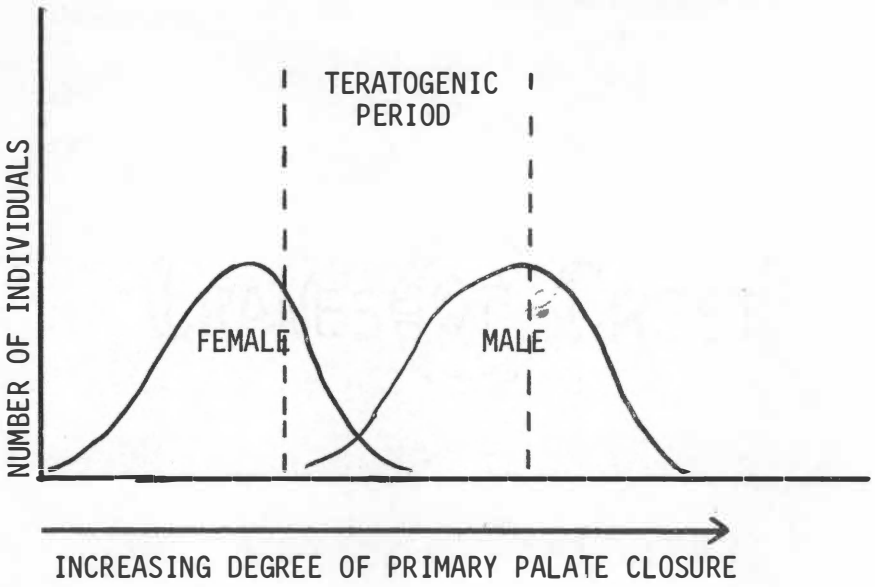
(2) chromosomal damage, (3) mitotic interference, (4) altered nucleic acid integrity or function, (5) lack of normal precursors or substrates, (6) altered energy sources, (7) changed membrane characteristics, (8) osmolar imbalance, and (9) enzyme inhibition. Although many of these "mechanisms" operate at the molecular or subcellular level, their ultimate effect generally results in fewer than the required number of cells or cell products to accomplish normal morphogenesis or functional maturation. The majority of teratologic agents (as radiation, mitotic inhibitors, and anticancer drugs) effective in inducing clefts of the primary palate in rodents are detrimental to rapidly dividing cell populations (Burdi et al. 1972). If cleft producing teratogenic agents affect histogenesis or organogenesis of the limb or other tissues, additional abnormalities or a general growth retardation might occur. For example, injection of 6-azauridine into pregnant mice retards overall fetal development (indicated by lower mean fetal weight), as well as delaying the process of palatal shelf horizontalization (Dostal and Jelinik 1974). Many teratogenic agents used to induce oral clefts in rodents affect development of the limb skeleton (Merker 1977). Thus, in humans, abnormal dermatoglyphics could result if cell numbers within the fetal pads are altered by an environmental teratogen.

Dermatoglyphic data may provide a means of determining whether specific growth rates are associated with cleft formation. Meskin et al. (1968) suggested a hypothetical model for palatal closure to explain Caucasian sex differences in incidence and severity of oral clefts.

Their assumptions are (1) the presence of a fixed gestational period when factors causing clefts are operative, and (2) males are more advanced in palatomorphogenesis than females. The model and its theoretical expectations are illustrated in Figure 1. The ordinates represent successive stages of increasing degrees of primary or secondary palate closure, the abscissa is the number of individuals at any given stage. A theoretically fixed teratogenic period is superimposed upon this framework.

The developmental density distributions of male and female embryos are different. Males have a higher incidence of CL±P because of a greater area under the male curve in the teratogenic period (Figure 1). At the same time, females are entering the teratogenic period and have fewer individuals within the "susceptible" zone. However, those females who are affected are in a relatively early stage of development, the consequence being a higher frequency of complete clefts. A similar rational is suggested for clefts of the secondary palate although now females have the largest number of individuals within the critical period (Figure 1). As a result, more females are affected by isolated cleft palate.

The relative lag in female palatal shelf movement and closure corresponds with this model (Burdi and Silvey 1969a). In fact there is considerable evidence for other sex-associated differences in maturational rates or timing of prenatal events. Before the vertical palatal shelves become horizontal, the upper face is prognathic over the mandible. In apparent synchrony with changes in shelf orientation, the maxillomandibular profile reverses with the lower jaw becoming



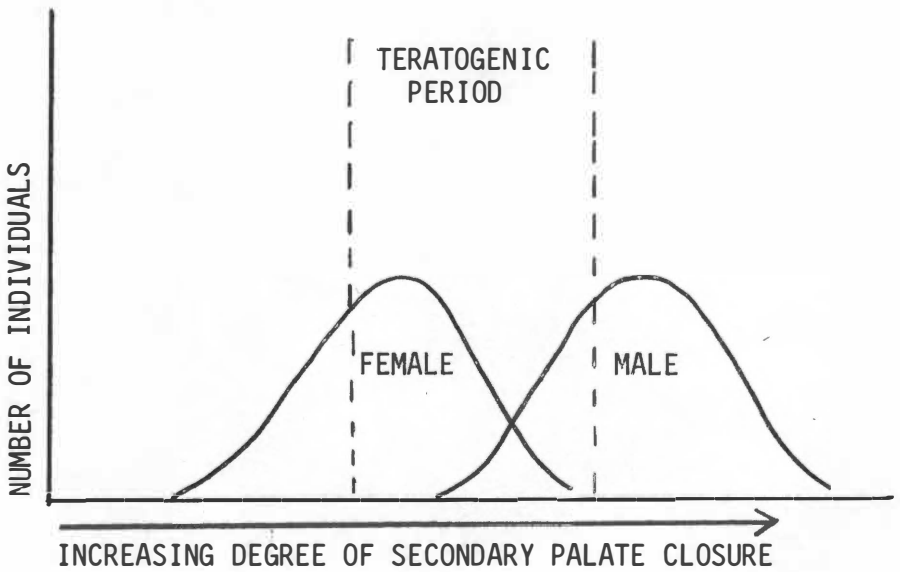


Figure 1. The Meskin et al. (1968) theoretical model for explaining Caucasian sex differences in incidence and severity of CL±P and CP. The ordinates represent successive stages of development, the abcissa is the number of individuals at each stage.

prognathic (Burdi and Silvey 1969b). Females complete the transition during the eighth week (29 mm crown-rump length). Males pass the stage with a crown-rump length of 25 mm. Males are advanced by as much as 10 percent in crown-rump length, development of the deciduous dentition and development of the hand skeleton (Garn et al. 1974; Garn and Burdi 1971). Male advancement in proximal (carpal) and distal (metacarpal-phalangeal) regions of the hand is greatest in younger embryos (crown-rump length of 15-30 mm) although sex differences are present until at least 75 mm (Garn et al. 1974).

Sex differences in rates of palatal closure are probably interrelated with overall differences in prenatal growth rates and maturation. If so, an additional expectation seems apparent in the Meskin et al. (1968) model. The model provides a relative scale reflecting an individual's status in palatal and general development. Individuals falling within the teratogenic period have achieved a predictable stage of development. Males in the zone can be considered somewhat less advanced relative to the male mean. Analagous females are advanced relative to their mean.

For morphological features where final expression depends on the stage of development at a specific prenatal time, one would expect reduced sexual dimorphism (relative to the total population) if "less advanced" males and "advanced" females were compared. It is, of course, impossible to identify all individuals belonging to a specific stage of development. The model does, however, allow the status of cleft lip and/or palate males and females to be predicted. Their amount of

sexual dimorphism can be compared with that of controls. This contrast, is depicted in Figure 2. The developmental density distribution of male and female embryos who develop facial clefts are shown as subsamples of the total population. If the samples differ according to predictions, the difference in sexual dimorphism B will be less than A. B is the distance between means of cleft males and females. A is the distance between all Caucasian males and females. The model applies only to morphological characteristics in which final form depends on the stage of development at a specific prenatal time.

In this regard, evidence indicating reduced or possibly reversed sexual dimorphism in size of the permanent dentition in unilateral cleft lip and palate patients is perhaps an expression of this phenomena. Unlike the control series, Foster and Lavelle (1971) found cleft females as having larger measurements than corresponding males.

> The upper incisors, canines and first premolars were larger in both dimensions in the females than in the males. In addition the lower incisors, canines, second premolars and second molars were larger in either the mesidistal or the buccolingual dimension in the females (Foster and Lavelle 1971:181-182).

To a certain degree, the data also support predictions that cleft males are developmentally less advanced and cleft females advanced in relation to normal controls:

> . . . essentially tooth dimensions for the cleft palate subjects were smaller than those for the normal subjects for most of the permanent teeth, but the female cleft subjects exhibited more tooth dimensions greater than those of the normal controls than did the male cleft subjects (Foster and Lavelle 1971:182).

The authors point out that the repair operation for patients could have affected dental size but not the pattern of sexual dimorphism.

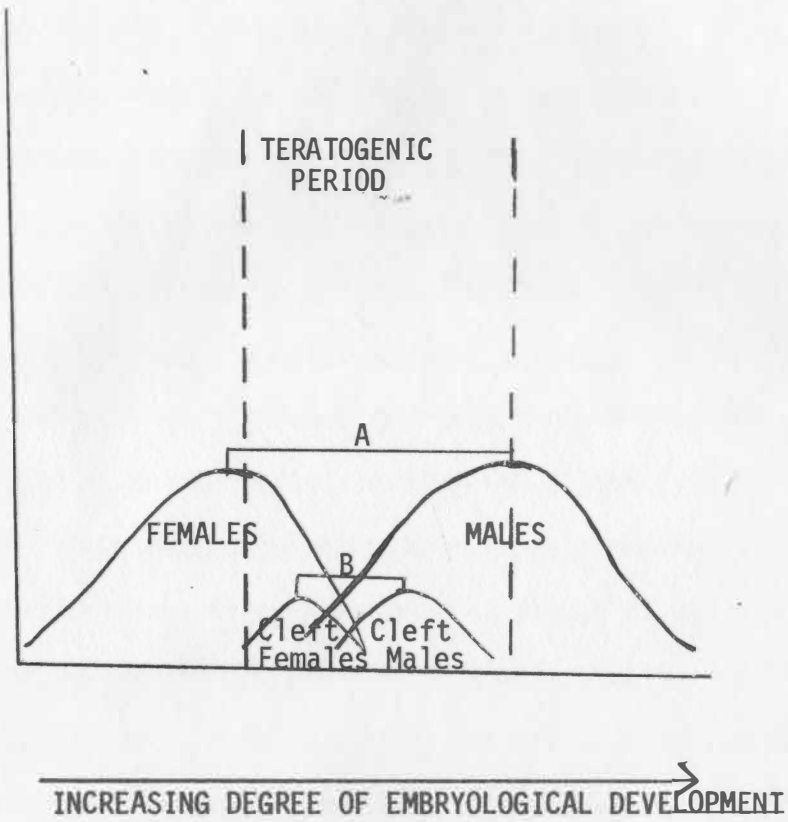


Figure 2. A hypothetical model concerning relative rates of embryological development in normal and cleft males and females. Embryological development of males with oral clefts is shown as less advanced relative to the male mean, cleft females are advanced relative to their mean. The distance "A" (the amount of sexual dimorphism in the total population) is greater than "B" (the amount in cleft males and females) for morphological traits in which final expression depends on the stage of development at a specific prenatal time.

Rates of development are important in tooth size. Mitotic activity in initial stages of tooth formation is responsible for size increase of the developing crown (Kraus and Jordan 1965). Later increase results from the activity of ameloblasts in enamel deposition and not from cellular proliferation. Nevertheless, factors affecting rates of initial mitotic activity or the number of possible cell divisions prior to the time of calcification could have an important effect on size of the tooth crown (Alvesalo et al. 1975).

Dermatoglyphics seem appropriate as test variables for the hypothesis of reduced sexual dimorphism. Pattern size and type depends on the hand's embryological development at the time primary ridges appear. Since embryological systems are interrelated, the presence of common factors influencing general growth of the dentition, skeleton and palatomorphogenesis are probably also important in the formation of dermal ridges. Reduced dermatoglyphic sexual dimorphism in cleft lip and/or palate patients would provide positive indirect evidence for overall validity of the Meskin et al. (1968) hypothetical model.

CHAPTER III

DEVELOPMENTAL STABILITY OF CLEFT LIP AND CLEFT PALATE PATIENTS

Fraser (1970) has suggested that cleft lip and cleft palate could result from generalized developmental instability. That is, something inherent in an individual's overall biological constitution determines whether development proceeds normally or abnormally in the presence of disturbing factors. This statement is illustrated by studies subjecting treatment populations of developing individuals to harmful substances. The effects are lethal to some, while others are seemingly impervious to any toxicity and develop normally (Waddington 1957). In relation to facial clefts, the presence of a malformation may signify reduced resistance to disturbing factors or reduced ability to recover once impairment occurs. Berndorfer (1970) expresses a similar perspective when he attributes proband differences in the severity of clefts to variation in individual "disposition" in biological adjustment and regenerative ability.

Developmental instability could explain increased frequencies of associated birth defects in CL±P and CP probands and relatives (Drillien et al. 1966; Meskin and Pruzansky 1969; Niswander and Adams 1968). Bear (1973) recognizes this association for CL±P while questioning whether CL and CL±P represent different levels of stability:

> If the inability of genetic control to stabilize development played a part in the occurrence of a facial cleft, its occurrence in a lesser degree could account for CL, while its occurrence in a greater degree could account not only for CL±P, but also for the greater association of noncleft defects with CL±P (Bear 1973:354-355).

Possibly the significantly higher break frequency (2.3 percent of chromosomes in leukocyte micro-cultures for first degree relatives of CL±P patients also reflects decreased stability. The incidence (1.2 percent) for families whose affected members had CP only was not statistically different from controls (0.6 percent) (Chang et al. 1970).

Highest rates of additional malformations are reported for isolated cleft palate. Meskin and Pruzansky (1969:312-313) think the differences in percent malformed are large enough to suggest that

> . . . the constitution of the patients with isolated cleft palate and their siblings may be more easily influenced to produce malformations than is the constitution of the other facial cleft patients and their siblings.

A measure which has been used to assess levels of developmental stability is differences between right and left sides of the body. Components of intraindividual variation in bilaterally symmetrical organisms include directional and fluctuating asymmetry. Directional asymmetry involves a tendency for greater development on one side of the sagittal plane relative to the other (Van Valen 1962; Soulé 1967). Fluctuating asymmetry is random minor deviation from perfect symmetry resulting from accidents or upsets in development. It reflects the inability of bilateral organisms to develop in precisely defined pathways since the genotype is usually identical for paired structures (Soulé 1967). Thoday (1958) considers the level of asymmetry a measure of the effectiveness of developmental control systems in buffering against accidents in development. In this sense, asymmetry measures developmental canalization, to use Waddington's (1957) terms, or the ability of the organism to control and regulate its development.

In the presence of disruptive factors, or if development is disturbed, canalized or buffered systems resist or display regulative behavior to repair themselves and return to normal. Poor canalization presumably results in increased asymmetry.

Certain evidence suggests fluctuating asymmetry is genotypically controlled. Selection and inbreeding experiments indicate the presence of a heritable component in the asymmetry of Drosophila chaeta (Mather 1953). Mather (1953) argues that the level of asymmetry depends on genic balance and is less a result of environmental forces. Under similar environmental conditions, population differences in fluctuating asymmetry reflect varied genotypic abilities in coping with, or resisting, disturbances in development. Thoday (1958) has shown that directional selection increases bilateral asymmetry of sternopleural chaeta-number. This deterioration was attributed to losses of balanced gene-complexes which are linked to genes for the character under selection.

Jantz (1978) has reported significant race differences in fluctuating asymmetry of the a-b interdigital count. African or African derived populations have lower asymmetry than Caucasian samples and American Indian populations are highest. Interpopulation variation within racial groups is minor as even populations of differing socioeconomic level are relatively similar. The overall pattern of heterogeneity within and between races provides little indication of an important environmental component in dermatoglyphic asymmetry. Singh (1970) found a genetic component for the asymmetry measure A_2 (which incorporates both fluctuating and directional asymmetry) of finger

ridge-counts using parent-child correlations. However, heritability values of 35 percent for males and 23 percent for females are low suggesting environmental and chance effects.

Males are consistently more asymmetrical than females in dermatoglyphics and dental size. This tendency suggests the sex chromosomes, and especially the X, provide a regulatory function affecting levels of asymmetry (Garn et al. 1966, 1967; Jantz 1978; Webb 1977). This position is strengthened since Turner (XO) patients show markedly increased asymmetry while Klinefelters (XYY) deviate only slightly from normal (Jantz 1978). Jantz (1978) questions whether this regulatory role lies within the heterochromatic nature of the inactive chromosome and its affect on mitotic activity. "The inactive X chromosome in females may be responsible for their slightly slower rate of intrauterine growth, and this in turn may partially explain the higher female correlations" (decreased asymmetry) (Jantz 1978:5).

Suarez (1974) attributes high magnitudes of fluctuating dental asymmetry in Neandertals to decreased heterozygousity caused by inbreeding. However, little evidence suggests a strong association between inbreeding and asymmetry. Experimental studies have shown increased dental asymmetry in isogenic inbred lines of mice as compared with isogenic hybrid populations (Bader 1965). Even so, differences between inbred and heterozygous wild type/random bred populations were slight and nonsignificant.

Bailit et al. (1970) tested the relationship between asymmetry and individual levels of inbreeding (as measured by the coefficient of

consanguinity) in the highly inbred Tristan da Cunha population. In both male and females, variation in the degree of inbreeding was not related to variation in the degree of dental asymmetry. Niswander and Chung (1965) found greater central incisor asymmetry in Japanese children of 1 1/2 cousins than those of unrelated parents. Since other inbred marriage classes (including first cousins) showed no significant effect, the results lacked substantial evidence for an overall inbreeding effect.

Experimental studies on Drosophila have shown that major changes in the external environment can affect sternopleural asymmetry (Beardmore 1960; Thoday 1956). An unfavorable environment may result in increased asymmetry. Various types of stress (i.e. audiogenic, temperature and behavioral modification) have caused increased fluctuating asymmetry of specific long bones and the dentition in rodents (Siegel and Smookler, 1973; Siegel and Doyle 1975a, 1975b; Siegel et al. 1977). However, DiBennardo and Bailit (1978) failed to find any relationship between magnitudes of fluctuating dental asymmetry and measures of prenatal stress, as parental age and socioeconomic status, in Japanese children. Parsons (1973) found possible evidence for a maternal age effect in ridge-count asymmetry with values tending to be highest in older mothers. Oliveira (1978) noted a similar relationship for paternal age and dermatoglyphic asymmetry, with the effect being most pronounced in male offspring.

A general trend in studies of human dental asymmetry has been to attribute population variation to environmental differences (Bailit

et al. 1970; Doyle and Johnston 1977; Perzigian 1977). Possible genotypic differences in buffering capacity have been given less consideration. Undoubtedly neither system is mutually exclusive. A "good genotype" serves to buffer the individual against developmental accidents in unfavorable environments (Thoday 1958).

There are certain attributes of dermatoglyphics for fluctuating asymmetry research which should be mentioned. These attributes include prenatal formation, ease in quantification and lack of selective regulation (Jantz 1978). The levels of stability achieved by different characters are probably conditioned by the relative disadvantage incurred with increased asymmetry (Mather 1953). Unlike many bilateral structures, the effect of selection in reducing dermatoglyphic asymmetry is probably limited.

The study of dermatoglyphic asymmetry is relevant to the formation of oral clefts if it provides information concerning overall developmental stability. Since morphogenetic systems are interrelated, it could be anticipated that the level of assymetry in one structure correlates with asymmetry of other characters within the individual. Webb (1977) has found several significant correlations between asymmetry values of the permanent dentition and digital and palmar ridge-counts. In other studies, asymmetry correlations of various structures, even related ones, are low suggesting little agreement in overall tendencies (Garn et al. 1966, 1967; Mason et al. 1967; Soulé and Baker 1968; Van Valen 1962). Some interrelationship is suggested in third molar agenesis which is accompanied by increased asymmetry of the other teeth (Garn et al. 1966).

Soulé and Baker (1968) disregard the importance of individual concordance suggesting the true concordance in asymmetry is a population phenomenon, termed the population asymmetry parameter. While individuals may display differences in buffering ability of specific characters, the levels of asymmetries in populations correspond. A population highly asymmetrical for one characteristic is likely to be so for other features (Soulé 1967). Soulé and Baker (1968) consider the regulatory variable affecting the overall level of asymmetry or buffering ability a reflection of the compatibility or coadaptation of genetic elements in each population.

Under predictions of the population asymmetry parameter, a population displaying a lack of buffering in facial development (the clinic sample) should show increased asymmetry in other characteristics as dermatoglyphics. Fraser (1970) seems to suggest that the sample which should show greatest assymetry is patients without a family history of oral clefts (FH-):

> Theoretically, cleft lip or cleft palate could occur as the result of a generalized developmental instability rather than a developmental deviation restricted to the face. This would account for the increase in other major malformations noted in children with CL(P) [CL±P] and CP.
>
> . . . . . .
>
> Drillien et al. (1966) found that the increase [in associated malformations] occurred mainly in families where the family history was negative for clefts of lip and palate, which one might expect if a proportion of cases resulted from generalized developmental instability (Fraser 1970:348).

From a teratogenic basis, individuals carrying mutant genes which destabilize developmental pathways are more easily diverted from normal development by environmental influences (Wilson 1977a). Thus, one might

expect decreased developmental stability in FH+ families. Previous studies have reported increased dermatoglyphic asymmetry for FH+ CL±P patients and first degree relatives but not for patients lacking this history (Woolf and Gianas 1976, 1977).

CHAPTER IV

DERMATOGLYPHIC DATA AND STATISTICAL METHODOLOGY

I. CLINICAL AND CONTROL SAMPLES

The clinic population is composed of cleft lip and/or cleft palate patients and normal first degree relatives attending the East Tennessee Crippled Children's Service Clinic, Ms. Norma Osborne, Director, or private patients of Drs. E.B. Andrews, J.B. Cox, K.A. Harper, R.E. Knowling, W.J. Schneider and J.W. Taylor, Knoxville, Tennessee. Participation of the families was voluntary with the prints being collected at the clinic or in the family homes. Whenever possible, data for the proband's parents and other siblings were collected.

Parents were interviewed regarding family histories of oral clefts and other biographical information. The patients are from Knox and surrounding counties in East Tennessee; all individuals were born within the continental United States and are Caucasian. The age span represented by patients and their siblings extends from approximately six months to sixty years.

Specific diagnoses as to cleft type and associated malformations were determined by consulting clinic and surgical records. Two diagnostic classifications were employed separating patients with cleft lip with or without cleft palate (CL$\pm$P) and patients with cleft palate only (CP). Patients with numerous developmental defects or Pierre Robin's syndrome were not included in the analysis.

CL±P patients and family members and CP patients and family members are analyzed separately. To fulfill statistical requirements of independent sampling, only one affected proband and one sibling were used to represent each family. If more than one proband or normal sibling were available, the representatives were randomly chosen. Both parents were included whenever possible. There is a possibility of inbreeding between parents which could affect sampling independence, however, any possible correlation is probably small. Table 1 provides the number of patients and first degree relatives by sex for each diagnostic category.

The CL±P sample was subdivided into (1) families having one or more known relatives with any type of cleft in addition to the proband (family history positive, FH+) and (2) families having no other relatives with any type of cleft (family history negative, FH-). In order to be classified as FH+, the coefficient of relationship (r) of the affected relative and proband was limited to at least r = 1/32. The coefficient of relationship represents the proportion of genes a pair of individuals have inherited from common ancestors (Fuhrmann and Vogel 1969; Thompson and Thompson 1965). A value of $r \geq 1/32$ with the proband includes monoyzgotic twins, dizygotic twins, parents, siblings, half siblings, grandparents, uncles, aunts, first cousins and second cousins. Table 2 gives the number of CL±P patients, siblings, and parents by family history.

The control series consists of 92 male and 95 female students attending introductory physical anthropology classes at the University

Table 1. Number of Individuals by Sex in the CL±P and CP Clinic Samples. Individuals Included in the Parents and Sibling Samples Do Not Have CL±P or CP

Cleft Type	Propositi			Siblings			Parents		
	Males	Females	Total	Males	Females	Total	Males	Females	Total
CL±P	48	40	88	21	22	43	45	74	119
CP	15	14	29	10	5	15	18	18	36

Table 2. Number of CL±P Probands, Siblings and Parents Having Positive (FH+) or Negative (FH-) Family Histories of Oral Clefts

Family History	Propositi[1]			Siblings			Parents		
	Males	Females	Total	Males	Females	Total	Males	Females	Total
FH+	15	13	28	8	8	16	5	15	20
FH-	39	20	59	13	14	27	40	59	89

[1]Because of adoption, family history is unknown for one proband.

of Tennessee. In addition, ten male and seven female children from the Knoxville area were included to provide subadult controls. The University of Tennessee student sample is a fraction of the series reported by Oliveira (1978). All individuals were born within the continental United States and are of the Caucasoid race.

Black printer's ink and inkless materials were used to print the fingers and palms following traditional procedures (Schaumann and Alter 1976). Finger prints of small children were frequently obtained by coating the finger surfaces with charcoal and applying adhesive tape.

II. DERMATOGLYPHIC VARIABLES

The majority of variables are measurements of finger and palmar patterns or dermatoglyphic areas, quantified by counting the number of ridges between two specific points. The counts were made using a binocular microscope with 10.5 to 45 power magnifications. Each digit has a radial and ulnar count giving a total of 20 variables as ridge-counts of finger patterns. These counts are defined as the number of primary ridges crossing or touching a straight line connecting the core of the pattern to the triradial point (Holt 1968a). The triradius, secondary ridges and the ridge forming the pattern center are excluded from the count. Whorls have radial and ulnar counts greater than zero; arches, which lack triradii, and tented arches have scores of zero on each count. Loops have one count greater than zero and one count equal to zero. Thus, ulnar loops have positive radial counts and

zero ulnar counts while the reverse is true for radial loops. This method of counting ridges is illustrated in Figure 3.

The size and presence of patterns in the second, third and fourth interdigital areas are quantified in a manner somewhat analagous to finger patterns, a count of the number of ridges between the pattern center and the triradius (Jantz 1977c). Five variables on each hand are recognized and define the size of the pattern, its location and the triradius or main line essential to its formation. Ridge-counts for second interdigital patterns are counted from the associated triradius to the pattern core. Third interdigital patterns are formed by radial curvature of main line C and are counted from the c triradius to the center of the pattern (Figure 4). Fourth interdigital patterns are formed by either ulnar curvature of main line C, radial curvature of main line D, or occasionally, by the presence of an accessory d triradius. The count is made from the accessory or specific interdigital triradii, c or d, associated with a particular pattern (Figure 4). Zero scores are given to variables lacking a ridge-count. As in the case of finger counts, the triradius and the ridge forming the core of the pattern are excluded from the count.

Ridge-counts for each interdigital area are obtained by counting along straight lines connecting pairs of triradii (a-b, b-c, c-d), but excluding the points (Figure 4) (Holt 1968b). Counts on each hand were analyzed separately rather than summed as is commonly done.

Four triradii are present at the base of the fingers except occasionally when the c triradius is missing. This problem is easily

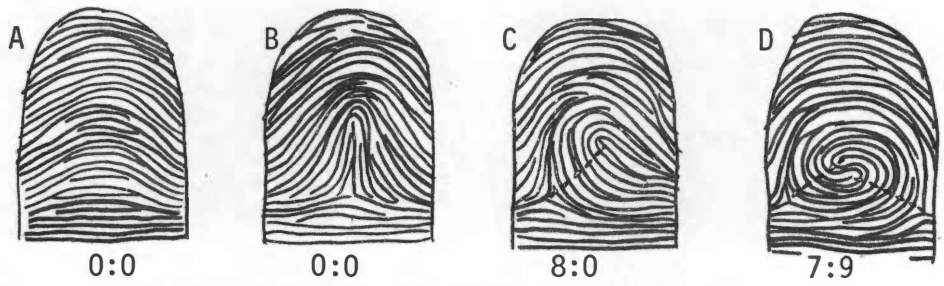


Figure 3. Finger tip patterns showing radial and ulnar ridge-counts for arch (A), tented arch (B), loop (C) and whorl (D).

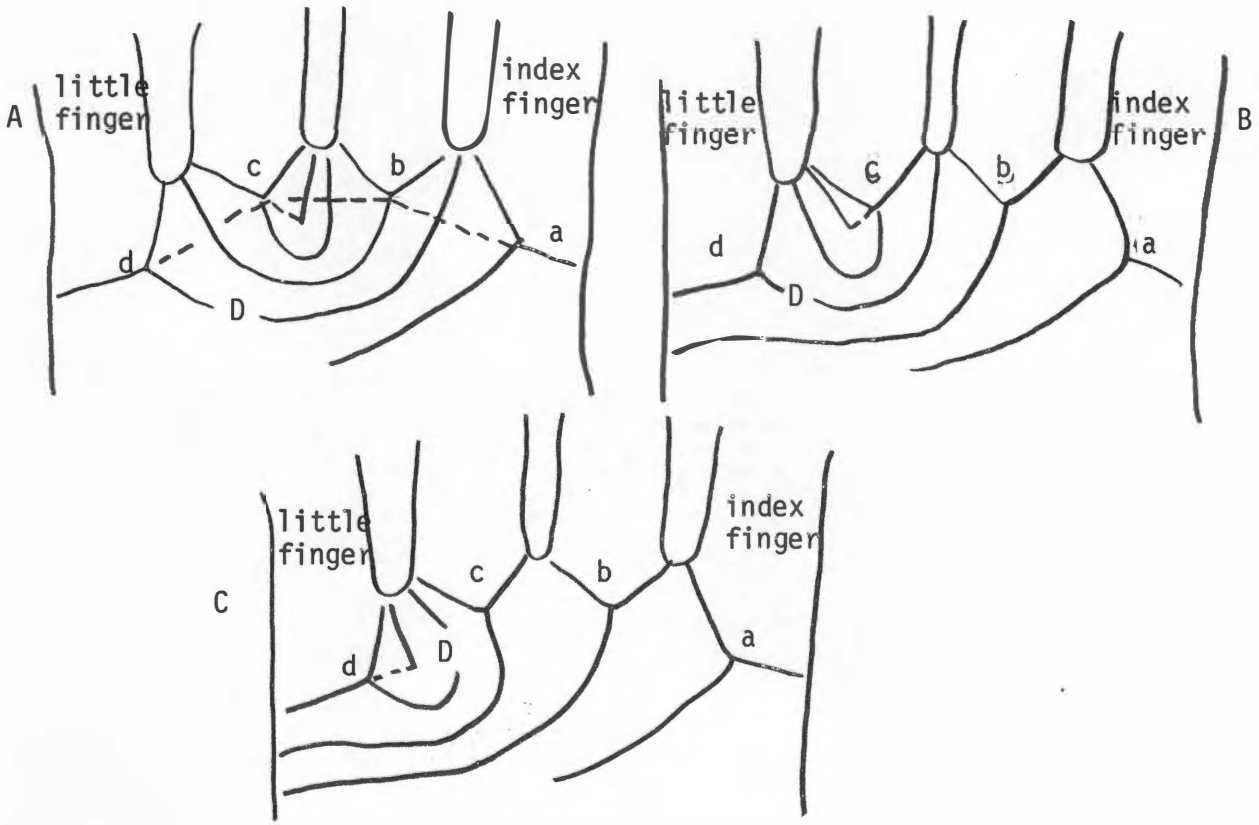


Figure 4. Common patterns in the third and fourth interdigital area of the palm. Figure 3A shows a loop formed by radial recurvature of the C-line, 3B by ulnar recurvature of the C-line and 3C by radial recurvature of the D-line. Counts are made from the appropriate triradius to the core of the pattern as shown by the dotted lines. Counts may also be made between the a, b, and c triradii themselves.

solved if a rudimentary triradius or field discontinuity can be distinguished and designated the point for counting. If not, the approximate location of c was determined by counting 11 ridges below the midline of the finger flexion crease (Baitsch and Schwarzfischer 1959). This procedure allows complete data sets to be obtained. The list of finger and palmar ridge-counts is given in Table 3.

A measure of ridge breadth (W) or ridge density was obtained for ridges in the second interdigital a-b interval. Ridge breadth refers to the distance in micrometers (μm) from the center of one furrow to the center of the next along a line perpendicular to the ridges (Jantz and Parham 1978). The concept possibly incorporates two distinct components, ridge breadth and furrow breadth. Two quantities are combined to calculate W, the sum of the left (D_L) and right (D_R) a-b distances measured in millimeters and the sum of the left (C_L) and right (C_R) a-b counts plus a factor of 2 as compensation for not having counted either triradius (Penrose and Loesch 1967). The following formula was used:

$$W = \frac{(D_L + D_R)}{(C_L + C_R + 2)}$$

If triradius a was duplicated, the more radial triradius was accepted as the terminal point (Jantz and Parham 1978).

Variation in ridge breadth is related to sex with males having wider ridges (Jantz and Parham 1978, Penrose and Loesch 1967), race (Jantz and Parham 1978) and hand or body size (Cummins et al. 1941;

Table 3. Name Abbreviations of Finger and Palmar Ridge Counts

Finger Ridge Count	Abbreviation	Palmar Ridge Count	Abbreviation
Left 5 Radial	L5R	Left c-d	LCD
Left 5 Ulnar	L5U	Left b-c	LBC
Left 4 Radial	L4R	Left a-b	LAB
Left 4 Ulnar	L4U	Right c-d	RCD
Left 3 Radial	L3R	Right b-c	RBC
Left 3 Ulnar	L3U	Right a-b	RAB
Left 2 Radial	L2R	Left d-radial	LDRad
Left 2 Ulnar	L2U	Left c-ulnar	LCUln
Left 1 Radial	L1R	Left c-radial	LCRad
Left 1 Ulnar	L1U	Left d-accessory	LDAccess
Right 5 Radial	R5R	Left a-accessory	LAAccess
Right 5 Ulnar	R5U	Right d-radial	RDRad
Right 4 Radial	R4R	Right c-ulnar	RCUln
Right 4 Ulnar	R4U	Right c-radial	RCRad
Right 3 Radial	R3R	Right d-accessory	RDAccess
Right 3 Ulnar	R3U	Right a-accessory	RAAccess
Right 2 Radial	R2R		
Right 2 Ulnar	R2U		
Right 1 Radial	R1R		
Right 1 Ulnar	R1U		

Ohler and Cummins 1942). Although the actual number of ridges is age independent, ridge width increases with age due to increasing size. Sample comparisons must be limited to subjects of comparable age or corrected accordingly (Penrose and Loesch 1967).

Maximal atd angle is defined by the most distal axial triradius, the most lateral a triradius and the most medial d triradius on each palm (Holt 1968). It is a means of quantifying the position of the axial triradius, the higher the position of t, the greater the angle (Holt 1968a). A highly placed triradius is often related to the presence of a hypothenar pattern. Females tend to have larger atd angles and somewhat greater variances than males (Penrose 1954). The angle is largest in children. With increasing age, the palms become longer and narrower and the angle gradually decreases. Since the variable is dependent on age and sex, these factors must be controlled when comparing samples.

III. STATISTICAL METHODOLOGY

Following collection of the data, the values were transferred to standard punch cards for computer processing. Three intermediate steps involving the replacement of missing values and data transformations were completed before testing the samples for differences.

Replacement of Missing Values

Multivariate analysis requires complete data sets for each individual (Howells 1973). Occasionally, due to missing digits or poor print quality, finger or palmar ridge-counts could not be obtained.

Cases with missing values were excluded unless missing information for a single digit and/or single palmar variable. To allow maximum sample sizes, missing observations were replaced by probable estimates using a regression method (Holt 1968a). The missing value was estimated using one of three formulae derived from the inverse variance-covariance matrix of either (1) radial finger ridge-counts, (2) ulnar finger ridge-counts, or (3) palmar pattern and interdigital ridge-counts. The rationale allowing development of separate formulae derives from factor analytical solutions which indicate internal consistency and integrity of finger versus palmar and radial versus ulnar finger ridge-counts (Jantz and Owsley 1977; Knussman 1967, 1969; Nance et al. 1974). The fortran program used for matrix inversion is given in Davies (1971). Thus, an individual missing a maximum of three variables (i.e. a finger radial count, ulnar count, or palmar count) could still be included in the analysis.

This procedure for replacing missing values avoids problems introduced by the alternative solution of substituting means for missing counts. The latter reduces the variance-covariance matrix and is more likely to insert a disproportionate value relative to other dimensions (Howells 1973). Mean substitution for missing values of large or small patterns is inappropriate since true values may differ considerably. A total of eight finger ridge-counts and seven palmar ridge-counts were estimated using this procedure.

Measurement Standardization

For certain tests, a combination of male and female subjects increase analytical efficiency by allowing larger samples. Since males and

females differ in ridge-counts and pattern frequencies (Holt 1968a), a Z score transformation was necessary. Standardization removes sex differences by equalizing males and females to the same mean while still preserving individual variation (McHenry and Giles 1971). The procedure is possible since males and females are fairly comparable in variable standard deviations.

The mean and standard deviation of each variable were used to transform raw scores into standardized scores using the formula below (Sokal and Rohlf 1969):

$$Z_{iv} = \frac{(X_{iv} - \overline{X}_v}{SD_v}$$

where X_{iv} is the original score for individual i on variable v, $\overline{X}_v$ is the male or female sample mean and SD_v is the male or female sample standard deviation. The new distribution has a mean of zero and a standard deviation of one. The transformation does not alter the mathematical form of the original distribution. "The frequency of any given Z score is exactly that of the X score corresponding to it in the distribution (Hays 1973:252). The raw-output-data file containing Z-scores was generated by the SPSS condescriptive procedure (Nie et al. 1974). Unstandardized variable means and standard deviations for the clinical and control groups are given in the Appendix.

Data Reduction Through Factor Analysis

A truncated components factor analysis was used to reduce the original set of variables into a smaller set of composite measures

which accounts for most of the reliable variance in the data. This variable synthesis results in very little loss of information since all major features are described. The general principle involves combining correlated measures which share overlapping variance (Gorsuch 1974). For each factor, the scores for an individual can be calculated. Factor scores can be used as criterion variables in the same way as any other measurement. A high factor score means that a given person has a high value on the specific characteristic measured by the factor. It is common practice to use factor scores as operational representatives in subsequent statistical tests to produce more powerful or more interpretable analyses (Gorsuch 1974). For one thing, the use of factor scores improves analytical efficiency by increasing the number of degrees of freedom. The approach is also a means of avoiding any degeneracy present in a multivariate distribution (Tatsuoka 1971).

In matrix algebra, the truncated components model defines the original data matrix as a function of the factor score and factor pattern matrices:

$$Z_{nv} = F_{nf} P'_{fv}$$

where Z_{nv} is the standard score data matrix for n individuals and v variables, F is the matrix of factor scores for n individuals and f factors and P' is the transposed f by v factor pattern matrix where f is less than v (Gorsuch 1974). The equation allows derivation of all relationships required to factor analyze a fixed product moment correlation matrix (R_{vv}) into f defined uncorrelated factors. Since

the main diagonal of the correlation matrix is unaltered, the procedure attempts to account for the entire variance of each variable (Gorsuch 1974). The factors are extracted from R_{vv} by a characteristic roots and vectors analysis.

The initial factor axes were rotated using a quartimin oblique rotation (Delta = 0.0) in order to clarify the patterning of the variables. Geometrically, this criterion permits close relationships between the factor axes and variable clusters by allowing a fairly correlated solution (Kim 1975). Oblique factor scores represent clusters more accurately than possible with orthogonal factor scores. The rotated factor pattern matrix (P_{vf}) and its transpose $(P_{vf})'$ are used to obtain the factor score coefficient matrix (W_{vf}) (Gorsuch 1974):

$$W_{vf} = P_{vf} (P'_{fv} P_{vf})^{-1}$$

The matrix of factor scores (F_{nf}) for all individuals was calculated from the matrix W_{vf} and the matrix of standardized scores Z_{nv}:

$$F_{nf} = Z_{nv} W_{vf}.$$

Methods considered in order to limit the number of factors include (1) the scree test (Cattell and Jaspers 1967), (2) the eigenvalue greater than or equal to 1 criterion (Guttman 1954), (3) Bartlett's test of the significance of principal components, and (4) Harris' (1967) strategy. Bartlett's (1950) significance test determines whether the residual matrix contains significant variance remaining after the extraction of a specific number of

factors. The test provides an upper bound for the number of potentially useful factors. The chi-square test statistic is calculated from the eigenvalues by the equation

$$\chi^2 = -(N-1- \frac{2v + 5}{6} - \frac{2f}{3}) \text{ Log}_e r_f$$

where N represents the size of the sample, v the number of variables and f the number of factors extracted. The symbol r_f is defined as

$$r_f = |R_{vv}| / [\lambda_1 \cdot \lambda_2 \cdots \lambda_f (\frac{v-\lambda_1 -\lambda_2 \cdots -\lambda_f}{v-f})^{v-f}]$$

where $|R_{vv}|$ is the determinant of R_{vv} and λ_i represents the eigenvalues corresponding to each factor. The associated degrees of freedom are

$$df = \frac{(v-f-1)(v-f+2)}{2}$$

Harris (1967) suggests comparing solutions derived from alternative factoring algorithms and rotational procedures as a means of determining the number of factors and insuring factor robustness. Meaningful factors are those which are consistent across methods. Minimum residual and alpha varimax orthogonal and oblique factors (Delta = 0.0) are compared with the truncated components solutions (Kim 1975). Minimum residual (Minres) analysis is an iterative common factor approach. It attempts to reduce or eliminate error in the communalities (diagonal elements of the target matrix) so as to provide more accurate estimation of the off-diagonal correlations. Alpha analysis adjusts the off-diagonal elements through a recycling process in its attempt to improve factor reliability.

The patient and control series were combined for calculating factor scores. Throughout this process, the linear factor model assumes identical factor to variable relationships for all individuals (Gorsuch 1974). Verification of this assumption is necessary because correlation matrices of patients and controls may differ in strength or patterning of variable relationships. Validation is especially important for CL±P patients since increased within individual variation has been found (Adams and Niswander 1967, Woolf and Gianas 1976, 1977).

It is possible to detect patient-control differences by comparing factor structures for the two samples. Varimax-rotated solutions (Kim 1975), obtained by separately analyzing the control and CL±P intercorrelation matrices, were compared using a technique discussed in Veldman (1967). The program Relate measures the correspondence in factor structures of two orthogonal sets of factor axes. The factor structure for one sample (CL±P) was rotated within a common factor hyperspace to maximum contiguity with another factor structure (controls). If the rotation yields similar positions, the two sets of test vectors are essentially similar and thus highly correlated. Factor structure differences are reflected by the size of the angles, expressed in terms of cosines, separating comparable factors.

Multivariate Tests for Sample Differences

In all comparisons, three assumptions are implicit in the multivariate model: (1) the samples are random and independent;

(2) the variates follow a multivariate normal distribution; and (3) the samples share a common variance-covariance matrix (Kramer and Jensen 1969; Morrison 1976). In practice, however, most multivariate tests have proven fairly robust even when assumptions are violated (Kshirsagar 1972).

Randomness means that the observational units of a specific category were drawn independently of one another. Separate control and family class comparisons are necessary because dermal patterns have a significant genetic basis. Including several brothers and sisters of the same family could also make a sample unrepresentative (Preus and Fraser 1972).

A nongaussian distribution has been reported for certain dermatoglyphic variables (Holt 1955; Potrzebowski 1974; Weninger et al. 1976). Nevertheless, the assumption of multivariate normality does not appear to present a serious problem. The calculation of factor scores produces a set of linearly transformed variates. Linear combinations of variables tend to approximate a multivariate normal distribution irrespective of the distributions of the individual measurements (Philpot, personal communication).

The equality of the control and patient variance-covariance matrices was examined using the test criterion

$$\chi_t^2 = -2\left[1-\left(\sum_{i=1}^{K}\frac{1}{n_i-1}-\frac{1}{N-K}\right)\frac{2v^2+3v-1}{6(v+1)(K-1)}\right]\ln\left[\frac{N^{vn/2}L}{\Pi n_i^{\,vn_i/2}}\right]$$

where: K = number of groups

v = number of variables

N = total number of individuals

n_i = number of individuals in the i'th group

$$L = \frac{\Pi |\text{within sums of squares matrix (i)}|^{N(i)/2}}{|\text{pooled Sums of Squares Matrix}|^{N/2}}$$

which under the null hypothesis is distributed approximately as a chi-square with $(K-1)(v)(v+1)/2$ degrees of freedom (Kendall and Stuart 1966). This statistic tests for size or orientation differences in the concentric density ellipsoids of each sample (Seal 1964). The test is available as part of the SAS 76 procedure Discrim (discriminant analysis) (Barr et al. 1976).

The primary objective is to determine whether the dermatoglyphics of patient and control samples differ significantly. In separate tests, the control series was compared with CL±P patients, siblings and parents. The comparisons were repeated for CP patients and their family members.

The significance of a difference between two centroids can be determined within a multivariate context using Hotellings T^2 for two independent samples (Tatsuoka 1971). The statistic tests the null hypothesis

$$H_0: \underline{\mu}_1 = \underline{\mu}_2$$

of identical mean population vectors ($\underline{\mu}_i$) as opposed to the alternative hypothesis

$$H_1: \underline{\mu}_1 \neq \underline{\mu}_2$$

of different means (Morrison 1976). The test statistic is

$$T_t^2 = \frac{n_1 n_2}{n_1+n_2} (\underline{\bar{X}}_1 - \underline{\bar{X}}_2)' \hat{\sum}_{\omega}^{-1} (\underline{\bar{X}}_1 - \underline{\bar{X}}_2)$$

where: $\underline{\bar{X}}_i$ = the vector of sample means of v variates

$\hat{\sum}^{-1}$ = the inverse of the pooled within groups variance-covariance matrix.

The transformation $\frac{N - v - 1}{(N-2)v} T_t^2 = F_t$

allows conversion of the T_t^2 value to an F_t which under the null hypothesis follows an F distribution with (v, N-v-1) degrees of freedom. The null hypothesis is accepted if $F_t \leq F_{\alpha}$ (v, N-v-1) or rejected and the alternative hypothesis taken as true if F_t is larger. The test statistic calculated from the data is designated by the subscript t, the subscript α indicates the significance level of the critical value taken from the tables.

If the overall test is significant, the significance of individual variates can be tested with simultaneous confidence for all such tests (Morrison 1976). For the vector $\underline{a}' [a_1, a_2, \ldots a_v]$

$$T_t^2 (\underline{a}) = \left[\frac{\underline{a}' (\underline{\bar{X}}_1 - \underline{\bar{X}}_2)}{\sqrt{\frac{N}{n_1 n_2} \underline{a}' \hat{\sum}_{\omega} \underline{a}}} \right]^2$$

$$F_t (\underline{a}) = \frac{N-v-1}{(N-2)v} T_t^2 (\underline{a})$$

has the probability statement

$$P\left[\text{all } F_t\ (\underline{a}) \leq F\alpha_{(v,\ N-v-1)}\right] = 1-\alpha.$$

The discriminant function program BND07M (Dixon 1976) provided the necessary information for completing these calculations.

The test for differences between affected individuals and their normal siblings represents a matched samples problem with paired members. Hotellings T^2 for paired groups tests the hypothesis that the population centroid of the difference scores ($\underline{\mu}_d$) equals

$$\underline{\mu}_d = \underline{\mu}_1 - \underline{\mu}_2$$

the null vector [$\underline{0}$] (Morrison 1976, Tatsuoka 1971). The statistic is calculated from the mean vector of difference ($\underline{\bar{d}}$) of paired observations

$$\underline{\bar{d}} = \underline{\bar{x}}_1 - \underline{\bar{x}}_2$$

and the inverse variance-covariance matrix of difference scores ($\hat{\sum}^{-1}$).

$$T_t^2 = N\underline{\bar{d}}'\hat{\sum}_d^{-1}\underline{\bar{d}}$$

where N equals the number of matched pairs. The expression

$$F_t = \frac{(N-v)}{(N-1)v}\ T_t^2$$

is used to convert T_t^2 to an F_t. The null hypothesis is accepted if $F_t \leq F\alpha_{(v,n-v)}$ or rejected if $F_t > F\alpha_{(v,n-v)}$.

$\underline{\bar{d}}$, $\hat{\sum}_d$ and $\hat{\sum}_d^{-1}$

were obtained with SPSS computer statements, the Pearson Corr program (Nie et al. 1975) and the matrix inversion program given in Davies (1971).

Conservative multiple comparison univariate tests

$$T_t^2(\underline{a}) = \left(\frac{\underline{a}' \, \underline{\bar{d}}}{\sqrt{\frac{1}{N} \underline{a}' \, \hat{\Sigma}_d \, \underline{a}}} \right)^2$$

$$F_t(\underline{a}) = \frac{(N-v)}{(N-1)v} F_t^2(\underline{a})$$

have the probability statement

$$P \left[\text{All } F_t(\underline{a}) \leq F\alpha_{(v,\, N-v)} \right] = 1 - \alpha$$

The effect on dermatoglyphics of a positive family history of oral clefts was examined in a multiple comparison test for three samples. Factor scores of controls, CL±P, FH+ and CL±P, FH- patients were compared to test the null hypothesis of equal population centroids

$$H_0: \ \underline{\mu}_1 = \underline{\mu}_2 = \underline{\mu}_3$$

as opposed to the alternative hypothesis of different variable means

$$H_1: \ \text{some } \underline{\mu}_i \neq \underline{\mu}_j; \ \text{where } i, j = 1, 2, 3 \text{ and } i \neq j$$

The test statistic (Wilks' likelihood-ratio criterion) is defined as

$$\Lambda = \frac{|W|}{|T|}$$

where $|W|$ is the determinant of the within-groups sums of squares and cross products (SSCP) matrix and $|T|$ is the determinant of the total SSCP matrix (Tatsuoka 1971).

In addition to the overall test for sample differences, multiple comparison subtests of each variable by itself and pairwise comparisons of group means are possible. Two steps are required to convert

individual analysis of variance tests statistics (F_{tI}) into multiple comparison (F_{tS}) statistics with simultaneous confidence for all tests. For each subtest, the F-ratio (F_{tI}) for the simple anova is converted to Λ using the anova's degrees of freedom (ν_{1I}, ν_{2I}):

$$\Lambda = \frac{\nu_{2I}}{(\nu_{2I} + \nu_{1I})\, F_{tI}}$$

A function of Λ

$$F_{tS} = \frac{1 - \Lambda^{1/s}}{\Lambda^{1/s}} \quad \frac{ms - v\,(K-1)/2 + 1}{v\,(K-1)}$$

where

$$m = N-1-(v + K)/2 \text{ and } s = \sqrt{\frac{v^2(K-1)^2 - 4}{v^2 + (K-1)^2 - 5}}$$

then allows an approximate F_{tS} variate with $\nu_1 = v(K-1)$ and $\nu_2 = ms - v(K-1)/2 + 1$ degrees of freedom to be estimated (Tatsuoka 1971).

A two factor multivariate analysis of variance was completed in order to determine whether the patient series show reduced sexual dimorphism in dermal ridge counts. The classification variables are sex (male-female) and sample (CL±P or CP- Control). Factor scores were obtained by factor analyzing the unstandardized patient and control data matrix, the objective being to retain differences according to sex. The manova model allows multiple variate testing for possible effects due to sex, sample or interaction between sex and sample (Morrison 1976; Tatsuoka 1971). The second test is comparable to the one completed using Hotellings T^2 for independent samples. The test for treatment interaction allows measuring sample differences in the degree

of sexual dimorphism. If sex differences are reduced in the patient series, a significant treatment interaction corresponding to the predicted directions would be found. The test was completed using the SAS Manova General Linear Model procedure which provides for unbalanced designs (Barr et al. 1976).

Univariate Tests for Sample Differences

Two variables, ridge breadth and maximum atd angle, require separate treatment because of age dependence. The limited size of the samples makes it impractical to follow the usual procedure of comparing only subjects of similar age. Instead, an analysis of covariance technique was used to adjust sample means for the effects of age. The significance of differences in adjusted values can then be tested.

Age-related developmental components affecting these variables may differ between males and females. For this reason, raw data were used and both sexes were considered separately. Controls, CL±P patients and CP patients were compared in the same analysis. The SAS general linear model and regression procedures (Barr et al. 1976) provided the statistics required for testing the three basic null hypotheses of an analysis of covariance comparison: (1) the hypothesis of equal population regression slopes, (2) the hypothesis of no linear regression in the population, and (3) the equality of population means after adjustment for the covariate (Morrison 1976, Tatsuoka 1971).

Measures of Fluctuating Asymmetry

Asymmetry can be measured in a variety of ways (Holt 1954; Parsons 1964; Singh 1968). One way is to calculate the difference

(d_k) in raw scores for pairs (k) of homologous variables:

$$d_k = left_k - right_k$$

Positive and negative signs are retained and indicate the direction of asymmetry. The variance of d_k (V_{dk}) represents the degree of fluctuating asymmetry (Jantz 1977c). The variances are calculated as follows:

$$V_{dk} = \frac{\Sigma\, d_k^2 - (\Sigma\, d_k)^2/n_i}{n_i - 1}$$

The standard procedure for making samples comparisons involves univariate testing for variance homogeneity. A multivariate test for equality of variance-covariance matrices is applicable and presents an efficient approach to sample comparisons. These tests are possible if covariances ($C_{d_j d_k}$) of the d_k measures are obtained:

$$C_{d_j d_k} = \frac{\Sigma\, d_j d_k - (\Sigma d_j)\,(\Sigma d_k)/n_i}{n_i - 1}$$

An overall test for variance-covariance homogeneity would measure sample differences in the patterning of relationships among asymmetry measures as well as differences in the variances (Seal 1964). If differences are found, subtest comparisons of the individual variances help clarify whether the heterogenity is in the level of asymmetry or its patterning.

Matrices for the following groups were separately compared with the control sample: (1) CL±P probands, (2) CL±P FH+ probands, (3) CL±P FH- probands, (4) CL±P normal siblings, (5) CL±P normal parents, (6) CP probands, (7) CP normal siblings, and (8) CP normal

parents. The test for matrix equality is avaliable as part of the SAS discriminant function program (Barr et al. 1976). Individual variances were compared using a standard analysis of variance for variance homogeneity (Hays 1973; Sokal and Rohlf 1969).

CHAPTER V

RESULTS

I. FACTORS DERIVED FROM QUANTITATIVE DERMATOGLYPHIC VARIABLES

Several methods were considered in order to determine the appropriate number of factors used in comparing the clinic and control samples. There are essentially two steps included in this process. The first step involves determining the number of factors which adequately define the control data. The objective is to account for much of the variance in a limited number of factors. The control sample was examined using the following procedures to limit the number of factors: (1) Bartlett's test of the significance of principal components; (2) the scree test; (3) the eigenvalue greater than or equal to 1 criterion; and (4) Harris' strategy. The second step is to determine whether the factor structures of the clinic samples are comparable to that of controls. The linear factor model assumes identical factor to variable relationships for all individuals. This assumption requires justification since correlation matrices of patients and controls may differ. If any of the six clinic samples (CL±P or CP probands, CL±P or CP noncleft siblings, CL±P or CP normal parents) have different factor structures, it would probably be the CL±P or CP proband series. The correlation matrix for the larger sample (CL±P probands) was factor analyzed extracting the number of factors determined for controls. The factor structures of controls and CL±P probands were then compared by visual inspection and by use of the program Relate. This process helps assess the comparability

of the individual factors across samples and provides a guideline for determining whether the factors legitimately can be used in control-clinic sample comparisons. Oblique factor scores for the entire sample (controls plus all clinic samples) were calculated based on the number of factors determined for controls. Comparisons between samples were then accomplished using factors concordant in CL+P probands and controls. CL+P and CP probands (and CL+P and CP first degree relatives) were analyzed separately.

Bartlett's (1950) significance test of principal components determines whether the residual matrix retains significant variance after a given number of factors are removed. This test is calculated from the eigenvalues of the control correlation matrix (Table 4). As many as 25 factors can be extracted before the test statistic (chi square = 83.506 with 65 degrees of freedom) assumes a nonsignificant value ($\alpha = 0.05$).

The scree test is graphically displayed in Figure 5. A scree diagram is formed by plotting the value of the characteristic roots along the ordinate and the roots' factor number as the abscissa (Gorsuch 1974). An estimate of the appropriate number of factors can be obtained by searching the diagram for breaks signifying a marked reduction in the information contained in subsequent factors. In this case, however, the diagram is not very helpful. The first two eigenvalues are considerably larger than subsequent values and substantial breaks occur between the first and second and second and third factors. More than two factors are required since these account for only 32.2 percent of the total variance. The remaining

Table 4. Eigenvalues and Percent of Variance Accounted for by Truncated Component Factors of Controls, CL±P Probands and the Total Sample

	Controls		CL±P Probands		Total Sample	
Factor	Eigenvalues	Cumulative Percent of Variance	Eigenvalues	Cumulative Percent of Variance	Eigenvalues	Cumulative Percent of Variance
1	7.62	21.2	9.04	25.1	8.41	23.4
2	3.97	32.2	3.61	35.1	3.75	33.8
3	2.23	38.4	2.45	41.9	2.07	39.6
4	1.98	43.9	2.05	47.6	1.81	44.6
5	1.84	49.0	1.96	53.1	1.61	49.1
6	1.57	53.3	1.64	57.6	1.53	53.3
7	1.54	57.6	1.52	61.8	1.32	57.0
8	1.27	61.1	1.39	65.7	1.21	60.3
9	1.19	64.4	1.28	69.3	1.14	63.5
10	1.13	67.6	1.16	72.5	1.07	66.5
11	1.09	70.6	1.06	75.4	1.02	69.3
12	1.00	73.4	0.98	78.1	0.94	71.9
13	0.95	76.0	0.89	80.6	0.90	74.4
14	0.86	78.4	0.78	82.8	0.85	76.8
15	0.73	80.4	0.74	84.8	0.83	79.1
16	0.64	82.2	0.65	86.6	0.79	81.3
17	0.63	84.0	0.58	88.2	0.64	83.1
18	0.61	85.7	0.49	89.6	0.61	84.8
19	0.52	87.1	0.44	90.8	0.57	86.4
20	0.50	88.5	0.43	92.0	0.55	87.9

Table 4 (Continued)

Factor	Controls		CL±P Probands		Total Sample	
	Eigenvalues	Cumulative Percent of Variance	Eigenvalues	Cumulative Percent of Variance	Eigenvalues	Cumulative Percent of Variance
21	0.43	89.7	0.37	93.0	0.43	89.1
22	0.42	90.8	0.31	93.9	0.42	90.2
23	0.37	91.8	0.29	94.7	0.36	91.2
24	0.34	92.8	0.26	95.4	0.35	92.2
25	0.32	93.7	0.25	96.1	0.32	93.1
26	0.29	94.5	0.23	96.7	0.31	93.9
27	0.28	95.3	0.20	97.3	0.29	94.8
28	0.26	96.0	0.18	97.8	0.27	95.5
29	0.23	96.6	0.15	98.2	0.26	96.2
30	0.22	97.3	0.14	98.6	0.24	96.9
31	0.22	97.9	0.12	98.9	0.22	97.5
32	0.20	98.4	0.10	99.2	0.21	98.1
33	0.16	98.9	0.09	99.5	0.20	98.6
34	0.16	99.3	0.08	99.7	0.19	99.1
35	0.13	99.7	0.06	99.9	0.17	99.6
36	0.11	100.0	0.04	100.0	0.14	100.0

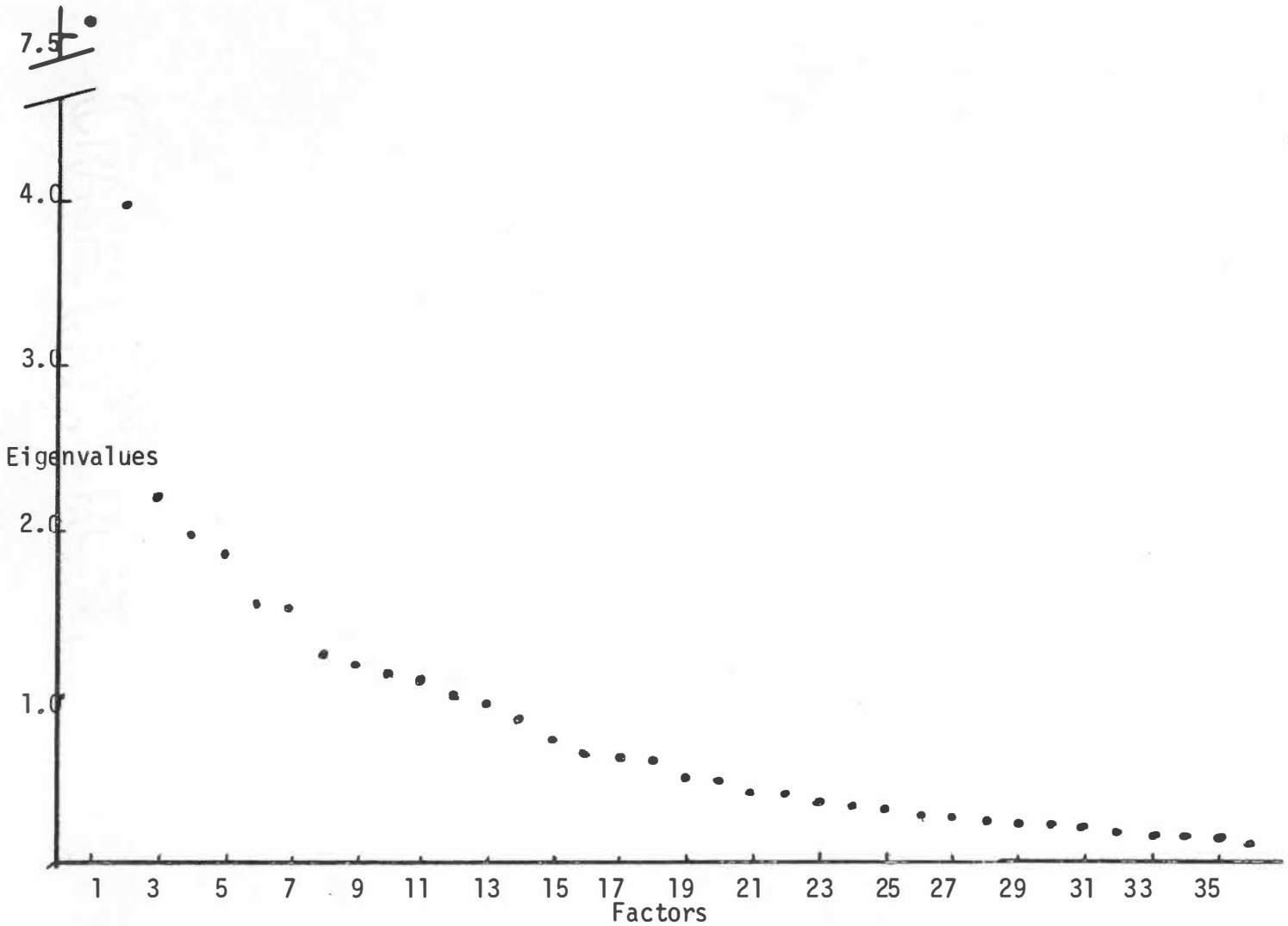


Figure 5. Scree test for eigenvalues of the control sample's correlation matrix.

points form a fairly smooth curve although there are minor breaks between factors 5 and 6 and factors 7 and 8. Six factors are required to account for more than 50 percent of the total variance while 10 factors account for 67.6 percent. The correlation matrix for the control sample has 12 factors with eigenvalues greater than or equal to one.

Solutions obtained using different procedures were compared to identify factors consistent across various methods (Harris 1967). Table 5 lists the solutions compared in determining the number of robust factors. The factor loadings of truncated components, minres, and alpha orthogonal and oblique solutions revealed 10 relatively stable factors having at least two variables with high loadings and a few variables with moderate loadings. Extracting larger numbers of factors produced factors with only two variables loading on them, suggesting larger numbers are unnecessary. Fewer numbers of factors result in lower final communalities (the proportion of each variable's variance accounted for by the factors) than is normally desirable. Ten factors were retained for further analysis. Ten is close to the number indicated using Guttman's root ≥ 1 criterion but is considerably lower than that indicated by Bartlett's test. The latter result is not unexpected since Bartlett's test provides an upper bound for the number of factors (Gorsuch 1974).

Once the number of factors was determined, the objective was to interpret the solutions and to assess the comparability of control and CL±P proband factors. The eigenvalues extracted from CL±P

Table 5. Factor Solutions Compared in Determining the Number of Robust Factors

Factor Solution	Number of Factors Extracted	Type of Rotation Varimax	Oblique
Truncated Components	8	X	
	9	X	X
	10	X	X
	11	X	X
	12	X	X
	15	X	
	20	X	
Minres	9	X	X
	10	X	X
	11	X	X
	12	X	X
	15	X	
	20	X	
Alpha	10	X	X

proband and total sample 36X36 correlation matrices (and the cumulative percentage of variance accounted for) are presented in Table 4. The first eigenvalue is larger in CL±P probands than is the case in controls and accounts for 25 percent of the total variance. The initial eigenvalue for controls accounts for 21 percent while the eigenvalue for the total series falls within the range prescribed by the previous samples. The difference in the cumulative percentages of variance remain throughout the solutions. Thus, ten factors account for 67.6 and 72.5 percent of the variances in controls and CL±P probands respectively.

Tables 6 through 10 present loadings from the factor structure matrices of controls, (varimax rotation) CL±P probands (varimax rotation) and the total sample (oblique rotation). Five general types of factors are represented in each group: (1) finger radial count factors (Table 6); (2) finger ulnar count factors (Table 7); (3) thumb factors (Table 8); (4) palmar interdigital factors (Table 9); and (5) palmar pattern factors (Table 10). Factors of each category are grouped together for clarity and ease in making sample comparisons. Salient loadings were used to cluster the factors. Also, significant loadings are underlined to help illustrate the meaning of each factor. Factor loadings were underlined if having a value greater than 0.41 (controls), 0.37 (CL±P probands) and 0.49 (total sample). These values are specific to each solution and were determined by marking the highest loading in each row of the factor structure matrix and then selecting the lowest of these high values. All loadings higher than this "minimum" value were then underlined. The procedure

Table 6. Finger Radial Count Factors for Controls, CL±P Probands and the Total Sample

Variables	Controls VIII (11.47%)	Controls II (8.24%)	CL±P Probands I (18.32%)	Total Sample I
L5R	0.76	0.13	0.79	0.82
L5U	0.08	0.09	0.03	0.12
L4R	0.76	0.28	0.83	0.86
L4U	0.37	0.08	0.38	0.40
L3R	0.58	0.58	0.66	0.73
L3U	0.16	0.00	0.22	0.26
L2R	0.29	0.57	0.63	0.49
L2U	0.09	0.16	0.38	0.41
L1R	0.16	0.77	0.65	0.51
L1U	-0.01	0.18	0.35	0.19
R5R	0.76	0.21	0.83	0.82
R5U	0.19	0.03	0.36	0.24
R4R	0.77	0.34	0.83	0.86
R4U	0.51	0.10	0.55	0.56
R3R	0.41	0.60	0.69	0.68
R3U	0.31	0.10	0.21	0.32
R2R	0.16	0.63	0.58	0.44
R2U	0.43	0.07	0.48	0.52
R1R	0.20	0.71	0.66	0.51
R1U	0.33	0.10	0.18	0.28

Note: The percentages of variance explained by the orthogonal factors are given in parentheses. Salient loadings are underlined.

Table 7. Finger Ulnar Count Factors for Controls, CL±P Probands and the Total Sample

Variable	Controls		CL±P Probands		Total Sample	
	V (7.22%)	III (7.43%)	VIII[1] (4.99%)	IV[2] (9.81%)	IV	VI
L5R	0.22	0.16	0.02	0.15	0.31	0.43
L5U	0.83	0.04	0.80	0.04	0.81	0.08
L4R	0.12	0.22	0.06	0.21	0.31	0.43
L4U	0.64	0.27	0.44	0.45	0.69	0.52
L3R	0.07	0.21	-0.02	0.40	0.34	0.46
L3U	0.22	0.67	0.15	0.76	0.32	0.75
L2R	0.34	0.07	0.04	0.32	0.49	0.34
L2U	0.06	0.80	0.00	0.69	0.11	0.73
L1R	-0.11	0.11	-0.07	-0.03	0.19	0.22
L1U	0.03	0.21	-0.02	0.34	0.15	0.53
R5R	0.19	0.18	-0.15	0.13	0.29	0.37
R5U	0.79	0.06	0.45	0.09	0.83	0.22
R4R	0.15	0.20	0.12	0.26	0.31	0.43
R4U	0.42	0.34	0.36	0.42	0.58	0.58
R3R	0.23	0.25	0.25	0.17	0.39	0.37
R3U	0.09	0.65	-0.06	0.81	0.25	0.76
R2R	0.42	-0.01	0.18	0.09	0.55	0.20
R2U	-0.06	0.69	-0.10	0.63	0.13	0.73
R1R	-0.06	0.01	-0.03	-0.13	0.16	0.12
R1U	0.00	0.22	0.02	0.17	0.29	0.56

[1]Moderate loading on the palmar count (RAAccess = 0.54).

[2]Low loading on a palmar count (RDAccess = 0.37).

Note: The percentages of variance explained by the orthogonal factors are given in parentheses. Salient loadings are underlined.

Table 8. Thumb Factors for Controls, CL±P Probands and the Total Sample

Variables	Controls II (8.24%)	Controls VI (4.89%)	CL±P Probands V (6.14%)	Total Sample VII
L5R	0.13	0.12	0.00	0.32
L5U	0.09	0.01	-0.08	0.08
L4R	0.28	0.08	-0.09	0.32
L4U	0.08	0.01	0.20	0.12
L3R	0.58	-0.08	0.07	0.39
L3U	0.00	0.03	0.28	0.12
L2R	0.57	0.09	0.11	0.43
L2U	0.16	0.17	0.05	0.36
L1R	0.77	0.16	0.52	0.80
L1U	0.18	0.79	0.68	0.66
R5R	0.21	0.09	0.02	0.35
R5U	0.03	0.02	0.11	0.12
R4R	0.34	0.06	0.05	0.36
R4U	0.10	-0.05	0.15	0.14
R3R	0.60	-0.08	0.24	0.48
R3U	0.10	0.10	0.16	0.16
R2R	0.63	0.00	0.31	0.44
R2U	0.07	0.13	0.06	0.34
R1R	0.71	0.15	0.47	0.73
R1U	0.10	0.69	0.74	0.65

Note: The percentages of variance explained by the orthogonal factors are given in parentheses. Salient loadings are underlined.

Table 9. Interdigital Count Factors for Controls, CL±P Probands and the Total Sample

Variables	Controls I (9.12%)	Controls X (4.36%)	CL±P Probands II (8.53%)	CL±P Probands VII (5.42%)	Total Sample II	Total Sample IX
LCD	0.56	0.57	0.76	0.36	0.64	0.59
LBC	0.70	0.00	0.59	0.14	0.83	0.33
LAB	0.79	0.06	0.20	0.81	0.26	0.88
RCD	0.52	0.44	0.73	0.31	0.53	0.61
RBC	0.71	-0.01	0.72	0.04	0.85	0.32
RAB	0.81	0.00	0.23	0.89	0.29	0.90
LDRad	0.14	-0.19	0.09	-0.09	0.24	-0.03
LCUln	0.17	0.12	0.20	-0.04	0.19	0.18
LCRad	0.25	0.17	0.48	-0.05	0.29	0.09
LDAccess	0.04	0.16	0.07	-0.07	0.09	0.05
LAAccess	0.04	-0.12	-0.22	0.05	-0.10	-0.07
RDRad	0.03	0.16	0.14	-0.02	0.05	0.09
RCUln	0.29	0.14	0.18	0.04	0.46	0.09
RCRad	-0.02	-0.02	0.29	-0.03	0.12	0.03
RDAccess	-0.01	-0.83	0.26	-0.11	-0.03	-0.05
RAAccess	-0.17	-0.26	-0.04	-0.08	-0.11	-0.19

Note: The percentages of variance explained by the orthogonal factors are given in parentheses. Salient loadings are underlined.

Table 10. Palmar Pattern Factors for Controls, CL+P Probands and the Total Sample

Palmar Pattern Factors	Controls IX (4.41%)	Controls X (4.36%)	CL+P Probands IX (4.48%)	CL+P Probands X (4.11%)	Total Sample VIII	Total Sample X	Controls IV (5.73%)	CL+P Probands III (5.45%)	Total Sample III	Controls VII (4.74%)	CL+P Probands VI (5.28%)	Total Sample V
LCD	-0.14	0.57	-0.03	0.11	0.20	-0.32	0.09	0.14	0.08	-0.03	-0.01	-0.16
LBC	0.13	0.00	-0.29	-0.10	-0.08	0.01	-0.15	-0.10	0.04	0.17	0.36	0.15
LAB	-0.07	0.06	-0.03	0.02	-0.03	-0.05	0.03	-0.01	0.01	-0.05	-0.04	-0.02
RDC	-0.18	0.44	0.04	0.23	0.19	-0.25	0.17	0.20	0.10	-0.18	-0.05	-0.22
RBC	0.21	-0.01	-0.25	-0.08	-0.05	-0.02	-0.19	-0.04	0.02	0.13	0.27	0.11
RAB	-0.14	0.00	0.03	-0.11	-0.10	-0.09	0.01	0.02	-0.05	0.01	-0.04	0.02
LDRad	-0.17	-0.19	-0.03	0.03	-0.08	0.22	-0.04	-0.05	-0.08	0.69	0.82	0.74
LDUln	0.17	0.12	-0.05	0.00	-0.12	-0.27	-0.52	0.80	-0.59	-0.46	-0.23	-0.42
LCRad	0.26	0.17	0.16	-0.22	0.19	-0.00	0.64	-0.58	0.69	0.00	-0.17	-0.10
LDAccess	0.81	0.16	0.08	0.77	0.55	0.15	0.05	-0.09	0.03	0.07	0.07	-0.05
LAAccess	0.51	-0.12	0.78	-0.13	0.63	-0.02	0.16	-0.09	0.08	-0.08	-0.03	-0.02
RDRad	0.02	0.16	-0.01	0.08	-0.11	-0.15	-0.02	-0.03	-0.05	0.79	0.73	0.75
RDUln	-0.02	0.14	0.00	-0.19	0.00	-0.05	-0.68	0.70	-0.66	-0.15	0.05	-0.18
RCRad	0.04	-0.02	0.06	0.38	0.06	0.03	0.82	-0.53	0.82	-0.24	-0.51	-0.29
RDAccess	0.01	-0.83	0.09	-0.07	0.24	0.71	0.11	0.08	0.09	-0.03	-0.09	0.05
RAAccess	0.52	-0.26	0.62	0.11	0.56	0.16	0.07	-0.05	0.12	-0.05	-0.07	-0.05

Note: The percentages of variance explained by the orthogonal factors are given in parentheses. Salient loadings are underlined.

provides a general cutoff point for determining the relative importance of a given variable to different factors of a particular solution. The tables present the loadings essential for interpreting the factors. Because finger and palmar variables contribute significantly to different sets of factors, the tables include only the variables of either type which are relevant. The factor loadings have a possible range of -1.0 to 1.0 and represent the correlation of each variable with a given factor.

The control sample (C.) has two radial count factors (C. II, VIII), one expressing radial counts on digits 1, 2 and 3 and the other loading digits 3, 4 and 5 (Table 6). Ulnar counts on the second and fourth digits load moderately on factor VIII, the 4-5 radial count factor. CL±P probands and the total sample (T.S.) differ from controls in having single radial count factors (CL±P I, T.S. I) which weigh all radial counts. Ulnar counts on the second and fourth fingers resemble controls (C. VIII) in having moderate loadings. Radial counts on the fourth and fifth digits have the highest loadings suggesting these factors most closely allign with C.VIII.

Each sample has two ulnar count factors, one emphasizing digits 2 and 3 and the other, digits 4 and 5. Factors correlated with digits 2 and 3 display some inconsistency in variables having moderate loadings. The proband factor (CL±P IV) has significant loadings on ulnar counts of the fourth digits and radial counts of the left third digit which are not important to the control factor. The total sample's factor (T.S. VI) is correlated with two variables (ulnar

counts on the thumbs) not identified in comparable factors of controls or CL±P probands. There is some intersample consistency in factors concerning ulnar counts on the little fingers. Control (C.V.) and total sample (T.S.IV) factors match in giving high loadings to the fifth digits and moderate loadings to ulnar counts of the fourth and radial counts of the second digits. The proband factor (CL±P VIII) places lower loadings on the left hand than the right and is unique in having a moderate loading on a plamar count.

The control sample has separate factors (C. II, C. VI) for radial and ulnar counts on both thumbs (Table 8). The radial count factor is correlated with radial counts on the second and third digits. CL±P probands and the total sample identify single thumb factors with high radial and ulnar loadings suggesting less independence of the two variables.

Each sample defines two interdigital count factors (Table 9), although there is variability in the specific expression of the individual factors. Factor I of controls has high loadings on all six counts and especially the a-b counts. The loadings then decrease in a gradient across the palms with the c-d counts having moderate loadings on this factor and also correlating with a second factor (C.X.). Factors concerning a-b counts in CL±P probands (CL±P VII) and the total sample (T.S. IX) have low loadings for fourth interdigital counts, yet exclude the b-c counts. The b-c counts relate to other factors (CL±P II, T.S. II) which include moderate loadings for the c-d counts.

In each sample, four factors are correlated with ridge-counts of palmar patterns (Table 10). a accessory counts define a second interdigital pattern size factor. In controls (C. IX) and the total sample (T.S. VIII) three factors also load the left d-accessory count. The right d-accessory loads significantly on other factors (C.X: T.S. X). In probands (CL±P IX, X), homologous variables of the accessory counts (LAAccess, RAAccess; LDAccess, RDAccess) pair and the pairs are correlated with separate factors. This pattern intuitively seems more reasonable than that for controls where LDAccess and RDAccess load on separate factors. Ridge-counts of third and fourth interdigital patterns formed by main line C define similar factors (C. IV, CL±P III, T.S. III) in all samples. In controls and the total sample, ridge-counts of patterns formed by radial curvature of (LCRad and RCRad) have positive loadings while counts for fourth interdigital patterns formed by ulnar curvature of C (LCUln, RCUln) have negative loadings. The same relationship is evident in SL±P probands except the signs are reversed. Positive-negative contrasts seem justifiable since formation of either a third or fourth interdigital pattern by C is a mutually exclusive event. Ridge-counts of patterns formed by D main line (LDRad, RDRad) load on a fourth interdigital pattern size factor (C. VII, CL±P VI, T.S. V). The ridge-count LCUln has a negative loading on this factor although in probands and the total sample the correlation is low. A negative relationship is expected since fourth interdigital patterns are generally formed by either C or D main lines. It is noteworthy that RCUln of the right hand shows practically no correlation. This difference may reflect bimanual

differences in developmental components affecting interdigital pattern formation. Bilateral differences have been noted in the frequencies of ulnar and radial C line terminations (Plato 1970). Patterns formed by the radial type are more frequent in the right hand whereas the ulnar type is more frequent in the left. The proband factor shows a higher negative correlation with right third interdigital patterns than the other solutions.

The matrix of cosines required to rotate the factor structure of CL±P probands to maximum congruence with that of controls is given in Table 11. These cosines may be interpreted as correlations (r) between the two sets of factors (Veldman 1967). The two CL±P factors most similar to specific control factors are: (1) the 5-4 ulnar count factor (C.V and CL±P VIII, r=0.94), and (2) the fourth interdigital pattern factor which loads LDRad and RDRad (C. VII and CL±P VI, r=0.92). The other correlations are lower indicating differences in loading patterns. For example, the 1,2,3 radial count factors of controls (C.II) is similar to two CL±P factors (CL±P I and V). The highest correlation is only 0.61. The thumbs' ulnar count factor has its highest correlation (r=0.70) with the CL±P thumb factor (C. V).

Analytical rotation of the two sets of factors to maximum allignment answers a second question besides measuring general factor similarity. Table 12 presents cosines of the angles between corresponding pairs of variable vectors. Low correlations signify differences in the content of the factors derived in the two analyses (Veldman 1967). Two palmar ridge-counts (LDAccess and RDAccess) have low

Table 11. Cosines Among CL+P Proband and Control Factor Axes

Control Factors	CL+P Factors									
	I	II	III	IV	V	VI	VII	VIII	IX	X
I	0.07	0.64	0.02	-0.02	-0.03	0.14	0.74	-0.01	0.00	-0.16
II	0.61	-0.30	-0.24	-0.27	0.45	-0.11	0.18	-0.19	-0.21	-0.28
III	0.06	-0.36	0.00	0.81	0.17	0.06	0.36	-0.07	0.13	0.16
IV	-0.04	0.27	-0.86	0.20	-0.05	-0.33	-0.10	0.09	-0.06	0.14
V	0.11	-0.04	0.04	0.10	0.11	0.09	-0.01	0.94	-0.19	-0.19
VI	-0.10	0.42	0.15	0.22	0.70	-0.06	-0.37	-0.08	0.24	-0.22
VII	-0.05	0.00	-0.33	-0.03	0.12	0.92	-0.14	-0.09	-0.08	0.10
VIII	0.75	0.31	0.18	0.24	-0.30	0.08	-0.32	-0.03	0.02	0.22
IX	0.16	-0.10	-0.14	-0.29	0.08	0.03	0.12	0.23	0.85	0.25
X	-0.03	0.10	0.15	-0.18	0.39	-0.09	0.12	0.08	-0.33	0.81

Table 12. Cosines of the Angles Between Corresponding Variable Vectors of CL±P Probands and Controls After Rotation of the Two Factor Structures to Maximum Alignment

Variable	Cosine	Variable	Cosine
L5R	0.86	R1R	0.82
L5U	0.80	R1U	0.77
L4R	0.92	LCD	0.89
L4U	0.92	LBC	0.75
L3R	0.79	LAB	0.91
L3U	0.76	RCD	0.89
L2R	0.84	RBC	0.66
L2U	0.83	RAB	0.89
L1R	0.89	LDRad	0.91
L1U	0.87	LCUln	0.98
R5R	0.90	LCRad	0.84
R5U	0.70	LDAccess	0.37
R4R	0.99	LAAccess	0.74
R4U	0.91	RDRad	0.92
R3R	0.89	RCUln	0.93
R3U	0.89	RCRad	0.90
R2R	0.90	RDAccess	0.24
R2U	0.93	RAAccess	0.73

correlations being less than 0.4. Correlations less than 0.8 are indicated for L3U, R5U, R1U, LBC, RBC, LAAccess and RAAccess. Factors for the two data sets differ primarily in the contributions of these variables. The remaining correlations are higher being 0.8 or above.

Scores for comparing patients and controls derive from factor analysis of the total sample. To insure that this solution accurately represents the factor structure of all samples, CL±P and control factors were checked for equivalency. Some differences are indicated suggesting certain factors are inappropriate for more extensive analysis. Those factors were eliminated following subjective and analytical evaluation of two criteria: (1) the final communalities of each variable; and (2) consistency in patterns of loadings. Factors emphasizing variables with particularly low communalities were excluded. Variables with low communalities are poorly represented by the factors. Final communalities for the truncated component solutions of controls, CL±P probands and the total sample are given in Table 13. The table reveals low communalities for ridge-counts from accessory a (LAAccess, RAAccess) and accessory d (LDAccess, RDAccess) triradii. These observations question the validity of two factors (T.S. VIII and X) requiring their elimination. In case of T.S. X, the loss is minor. The factor is poorly defined lacking several significant loadings by variables loading specifically on T.S. X. The only loading of consequence is for RDAccess.

T.S. factors were retained for analysis if emphasizing the same variables as found in parallel factors of CL±P probands and controls (consistency in loading patterns). Factors were excluded if

Table 13. Truncated Component Final Communalities for Controls, CL±P Probands and the Total Sample

Variable	Control Communality	CL±P Communality	Total Communality
L5R	0.72	0.77	0.75
L5U	0.72	0.71	0.70
L4R	0.74	0.80	0.78
L4U	0.65	0.68	0.64
L3R	0.78	0.75	0.72
L3U	0.56	0.78	0.60
L2R	0.61	0.68	0.61
L2U	0.73	0.72	0.62
L1R	0.73	0.78	0.75
L1U	0.71	0.75	0.65
R5R	0.76	0.80	0.75
R5U	0.67	0.56	0.71
R4R	0.79	0.80	0.77
R4U	0.63	0.69	0.67
R3R	0.68	0.72	0.70
R3U	0.57	0.79	0.62
R2R	0.65	0.63	0.59
R2U	0.70	0.77	0.66
R1R	0.62	0.74	0.69
R1U	0.71	0.70	0.64
LCD	0.75	0.76	0.70
LBC	0.67	0.66	0.75
LAB	0.70	0.75	0.80
RCD	0.68	0.74	0.62
RBC	0.68	0.75	0.74
RAB	0.72	0.88	0.83
LDRad	0.63	0.79	0.68

Table 13 (Continued)

Variable	Control Communality	CL+P Communality	Total Communality
LCUln	0.62	0.78	0.64
LCRad	0.69	0.77	0.62
LDAccess	0.71	0.66	0.39
LAAccess	0.40	0.70	0.42
RDRad	0.67	0.61	0.64
RCUln	0.69	0.73	0.72
RCRad	0.79	0.82	0.78
RDAccess	0.75	0.37	0.60
RAAccess	0.42	0.71	0.37

combining variables not held in common. T.S. VI (a 2-3 ulnar count factor) was excluded for loading significantly ulnar counts of digits 1 and 4, variables not emphasized in related factors C. III and CL+P IV. The thumb factor (T.S. VII) was eliminated for combining radial and ulnar ridge-counts. These variables load on separate control factors (C. II, VI). Thumb factors of CL+P probands and controls show lower correlations than other factor pairs (Table 11). Finally, T.S. II, a b-c count factor, was discarded because of a low inter-sample correlation (Table 12). Factor loading differences between CL+P probands and controls could reflect random variation. The sample size for probands is smaller than generally desired for factor analysis. Nevertheless, the aforementioned conservative approach was followed.

Five factors were retained for further comparisons: a radial count factor (T.S. I); a 5-4 ulnar count factor (T.S. IV); an a-b interdigital count factor (T.S. IX); and two palmar pattern factors (T.S. III, V). Technically T.S. I belongs in the rejection category because of moderate loadings for radial counts on the thumbs. These counts are not emphasized in the corresponding control factor (C. VIII). The factor was retained being the only one summarizing radial counts for digits 2-5. Together these five factor variables represent much of the information contained in left and right ridge-counts of digits 2-5, a-b and c-d interdigital counts and counts of third and fourth interdigital patterns formed by C or D main lines. Oblique rotation was used so the factors are slightly correlated (Table 14). The highest correlation between any of the pairs is

Table 14. Correlations Among Obliquely Rotated Factors from the Total Sample Solution

Factor	I	II	III	IV	V	VI	VII	VIII	IX	X
I	1.00									
II	0.09	1.00								
III	0.02	0.00	1.00							
IV	0.28	0.00	0.29	1.00						
V	-0.02	-0.02	-0.01	0.01	1.00					
VI	0.35	0.04	0.01	0.24	-0.01	1.00				
VII	0.30	0.11	0.01	0.17	-0.01	0.25	1.00			
VIII	-0.07	-0.04	0.09	-0.05	-0.01	0.04	-0.02	1.00		
IX	0.09	0.30	0.01	-0.15	-0.04	-0.08	0.01	-0.10	1.00	
X	0.12	-0.06	0.07	0.11	0.03	0.11	0.08	0.05	-0.14	1.00

r=0.34 for T.S. I and VI. T.S. IV and III have the highest correlation among the five factors kept for additional comparisons. For most of the factors, the correlations are low suggesting considerable independence.

II. MULTIVARIATE TESTS FOR SAMPLE DIFFERENCES

Multivariate tests for mean differences rest on assumptions of multivariate normality and equal variance-covariance matrices. The null hypothesis that the clinic populations have the same dispersion matrix as controls for the five factor variables was tested. The clinic samples were individually compared with controls. The results are presented in Table 15. Chi square values for three clinic samples (CL±P probands, CL±P siblings and CL±P parents are significant. The test statistics present a gradient on the chi square variate continuum. CL±P probands have the highest chi square, their siblings are intermediate and their parents have the lowest chi square. These values are on the same scale and can be interpreted as meaning CL±P probands are most different from controls. CL±P parents also differ but not to the same degree. The results are inconsistent with assumptions of homogeneity as applied to CL±P samples. Observed values for CP probands and first degree relatives are not significant at a 5 percent level although the CL±P trend is apparent. According to Cooley and Lohnes (1971) many researchers ignore the issue of variance-covariance homogeneity relying on robustness of multivariate tests in testing for mean differences.

Table 15. Tests for Homogeneity of Control-Clinic Sample Variance Covariance Matrices

Comparison Groups	Sample Size	Chi Square	D.F.	Significance Level
(1) CL±P Probands (2) Controls	88 204	39.685	15	.0005 > p
(1) CL±P Sibs (2) Controls	43 204	34.687	15	.005 > p > .001
(1) CL±P Parents (2) Controls	119 204	27.372	15	.05 > p > .01
(1) CP Probands (2) Controls	29 204	17.661	15	.30 > p > .25
(1) CP Sibs (2) Controls	15 204	13.923	15	.55 > p > .50
(1) CP Parents (2) Controls	36 204	10.927	15	.80 > p > .75

Nevertheless, a finding of unequal dispersions has a great deal of significance in its own right.

Two components of variation contribute to the differences noted between controls and the three samples: (1) the variances representing the spread of the multivariate density distributions; and (2) the covariances or interrelationships among the measures. To a certain degree it is possible to estimate the contribution made in rejecting the null hypothesis by variance heterogeneity. The individual factor score variances were tested for homogeneity with controls using a two-tailed analysis of variance test. The F_{tI} values, calculated as the ratio of the greater variance over the lesser one (Sokal and Rohlf 1969), are presented in Table 16. The variances for the individual tests are given in standard deviation form in Table 17. Of 15 tests, only four achieve significance at the 5 percent level if the tests are treated independently. It seems noteworthy that in these cases controls have the larger variance. Variance heterogeneity is suggested, but not to an extreme degree. Variance differences are probably not the only reason for rejecting the null hypothesis of equal control-CL±P dispersion matrices. The covariances probably also differ.

The next hypothesis considered was whether the populations differ in means of the five factor variables. The results from comparing each clinic sample with controls is presented in Table 18. None of the samples differ from controls at the 5 percent level although CL±P probands and their parents almost significantly differ. Table 17 gives sample means and standard deviations for each of the

Table 16. Analysis of Variance Tests for Factor Score Variance Heterogeneity

	Samples Compared								
	(1) CL±P Probands (2) Controls			(1) CL±P Siblings (2) Controls			(1) CL±P Parents (2) Controls		
Factor	SLV	F_{tI}	D.F.	SLV	F_{tI}	D.F.	SLV	F_{tI}	D.F.
I	1	1.261	87,203	2	1.192	203,42	1	1.124	118,203
III	2	1.261	203,87	2	1.106	203,42	1	1.074	118,203
IV	2	2.433*	203,87	2	1.625	203,42	2	1.961*	203,118
V	2	1.356	203,87	2	3.529*	203,42	2	1.467*	203,118
X	2	1.248	203,87	2	1.253	203,42	2	1.322	203,118

Abbreviation: SLV--Sample with largest variance.

*$p < .05$.

Note: The F-ratios are two-tailed tests and do not have simultaneous confidence.

Table 17. Factor Score Means and Standard Deviations for Controls and Clinic Samples

Factor	Mean	S.D.	Mean	S.D.	F_{tI}	D.F.	F_{tS}	D.F.	Mean	S.D.	F_{tS}	D.F.	F_{tI}	D.F.	Mean	S.D.	F_{tI}	D.F.	F_{tS}	D.F.
	Controls (n=204)		CL+P Probands (n=88)						CL+P Siblings (n=43)						CL+P Parents (n=119)					
I	0.084	0.950	-0.200	1.067	5.089*	1,290	1.004	5,286	-0.127	0.870	1.803	1,245	--	5,241	0.067	1.007	0.022	1,321	--	5,317
III	-0.001	1.041	0.017	0.927	0.021	1,290	--	5,286	-0.160	0.990	0.841	1,245	--	5,241	0.067	1.079	0.317	1,321	--	5,317
IV	0.121	1.137	-0.166	0.729	4.760*	1,290	0.939	5,386	-0.146	0.892	2.085	1,245	--	5,241	-0.130	0.812	4.465*	1,321	0.882	5,317
V	-0.035	1.084	0.062	0.931	0.541	1,290	--	5,286	-0.177	0.577	0.691	1,245	--	5,241	-0.070	0.895	0.087	1,321	--	5,317
IX	0.105	1.068	0.006	0.956	0.571	1,290	--	5,286	-0.150	0.954	2.097	1,245	--	5,241	-0.077	0.929	2.400	1,321	--	5,317
			CP Probands (n=29)						CP Siblings (n=15)						CP Parents (n=36)					
I			-0.025	1.262	0.309	1,231	--	5,227	-0.023	0.720	0.183	1,217	--	5,213	0.028	1.081	0.101	1,238	--	5,234
III			-0.186	0.712	0.853	1,231	--	5,227	0.167	0.704	0.380	1,217	--	5,213	0.056	1.008	0.094	1,238	--	5,234
IV			0.194	1.187	0.103	1,231	--	5,227	0.197	1.113	0.062	1,217	--	5,213	0.113	1.164	0.001	1,238	--	5,234
V			0.348	1.188	3.092	1,231	--	5,227	0.548	1.369	3.896*	1,217	0.765	5,213	-0.103	0.883	0.125	1,238	--	5,234
IX			-0.139	0.956	1.365	1,231	--	5,227	-0.272	1.001	1.763	1,217	--	5,213	0.034	1.052	0.138	1,238	--	5,234

*$p < .05$.

Note: The table also provides independent (F_{tI}) and simultaneous (F_{tS}) F-ratios and degrees of freedom from control-clinic sample comparisons.

Table 18. Test Statistics from Multivariate Comparisons of Control and Clinic Sample Factor Score Means

Comparison Groups	Sample Size	Test of Significance[1]	D.F.
(1) CL±P Probands (2) Controls	88 204	$F_t = 1.793$	5,286
(1) CL±P Siblings (2) Controls	43 204	$F_t = 1.403$	5,241
(1) CL±P Parents (2) Controls	119 204	$F_t = 1.759$	5,317
(1) CP Probands (2) Controls	29 204	$F_t = 1.101$	5,227
(1) CP Siblings (2) Controls	15 204	$F_t = 1.200$	5,213
(1) CP Parents (2) Controls	36 204	$F_t = 0.087$	5,234
(1) CL±P (FH-) Probands (2) CL±P (FH+) Probands (3) Controls	59 28 204	$F_t = 0.851$	10,568

[1]None of the F-ratios are significant at $\alpha = .05$.

factors along with analysis of variance comparisons. The univariate F-ratios (F_{tI}) achieve significance in a few instances although when transformed (since these are not independent tests) to a simultaneous confidence level (F_{tS}), their significance disappears.

Technically univariate F-ratios should be interpreted only if the null hypothesis is initially rejected by the overall test. In the case of CL±P probands, however, further analysis seems necessary. This sample has two factors (I and IV) with significant univariate F-ratios. Do these test statistics reflect real differences simply lost in the presence of other variables without intersample variation? The factors involved are finger radial and ulnar count factors. In both cases, proband means are lower than those of controls. Lower average factor scores imply smaller ridge counts on the original variables.

Three validation procedures were applied to clarify the issue. A split sample analysis was first. Both samples were randomly separated into equal halves (CL±P A, CL±P B; C.A, C.B). CL±P A and C.A were then tested for differences and so were the B samples. Results from the overall tests are presented in Table 19 and these are not significant. These results, however, are not the focus of the test. What is important is whether there is consistency in conclusions derived from univariate F-ratio tests of factors I and IV. Constancy in the results of the split sample comparisons lends support to the hypothesis that the differences are real. Means and standard deviations for each split sample and analysis of variance tests are shown in Table 20.

Table 19. Multivariate Test Results from Split Sample, Separate Sex and Raw Data Comparisons of CL±P Probands and Controls

Comparison Groups	Sample Size	Test of Significance[1]	D.F.
(1) CL±P Probands (Sample A) (2) Controls (Sample A)	44 102	$F_t = 1.404$	5,104
(1) CL±P Probands (Sample B) (2) Controls (Sample B)	44 102	$F_t = 1.017$	5,140
(1) CL±P Males (2) Control Males	48 102	$F_t = 1.186$	5,144
(1) CL±P Females (2) Control Females	40 102	$F_t = 1.814$	5,136
(1) CL±P Probands (Summed radial counts) (2) Controls (Summed radial counts)	88 204	$F_t = 1.122$	5,286
(1) CL±P Probands (Summed ulnar counts) (2) Controls (Summed ulnar counts)	88 204	$F_t = 1.200$	5,286

[1]None of the F-ratios are significant at $\alpha = .05$.

Table 20. Factor Score Means, Standard Deviations and Anova Tests for CL±P Proband and Control Split Samples

Factor	Mean	S.D.	Mean	S.D.	F_{tI}	D.F.	F_{tS}	D.F.
	Split Sample A Control (n=102)		Split Sample A CL+P (n=44)					
I	0.212	0.966	-0.175	1.034	4.744*	1,144	0.922	5,140
III	0.022	1.039	-0.049	0.852	0.160	1,144	--	5,140
IV	0.122	1.121	-0.048	0.702	0.866	1,144	--	5,140
V	-0.055	1.090	0.087	0.946	0.566	1,144	--	5,140
IX	0.143	0.982	-0.080	0.960	1.604	1,144	--	5,140
	Split Sample B Control (n=102)		Split Sample B CL+P (n=44)					
I	-0.044	0.919	-0.224	1.111	1.034	1,144	--	5,140
III	-0.025	1.047	0.084	1.001	0.342	1,144	--	5,140
IV	0.119	1.159	-0.285	0.744	4.533*	1,144	0.881	5,140
V	-0.15	1.082	0.038	0.926	0.080	1,144	--	5,140
IX	0.068	1.151	0.091	0.956	0.014	1,144	--	5,140

*$p < .05$.

Factor I significantly differs in the A samples, but not IV. The reverse is true for the B samples. The findings are inconsistent suggesting the effects are due to change.

A second analysis determined whether the F-ratios of factors I and IV reflect differences in only one sex. Possible sex specific differences were alluded to in an earlier theoretical consideration of the Meskin et al. (1968) model. It was hypothesized that CL±P males would have lower means and CL±P females higher means than corresponding controls. Factor scores were recalculated without standardizing the original variables. Probands and controls of each sex were compared separately. In both cases, the overall tests are not significant (Table 19). Inspection of the means and simple analysis of variance (anova) results (Table 21) also fails to reveal differences even for the two factor variables in question. There is no evidence for mean differences between male or female CL±P probands and their respective matched controls.

The final validation procedure was based on the original variables instead of factor scores. Factors I and IV are radial and ulnar ridge-count factors. Therefore, in separate analyses, radial and ulnar counts for digits 1-5 of CL±P probands were compared with controls. The variables were not standardized to remove sex differences. However, counts for homologous digits were summed (i.e. L5R + R5R) to avoid entering large numbers of highly correlated variables into the multivariate tests.

The overall F-ratios for the two tests are given in Table 19. The results are not significant. Means and standard deviations of

Table 21. Factor Score Means, Standard Deviations and Anova Tests for Male and Female CL+P Probands and Controls

Factor	Mean	S.D.	Mean	S.D.	F_{tI}	D.F.	F_{tS}	D.F.
	Control Males (n=102)		CL+P Males (n=48)					
I	0.259	0.964	-0.045	0.970	3.233	1,148	--	5,144
III	0.048	1.030	0.248	0.925	1.314	1,148	--	5,144
IV	0.085	1.156	-0.183	0.700	2.206	1,148	--	5,144
V	-0.046	1.069	-0.108	0.815	0.129	1,148	--	5,144
IX	0.030	1.103	-0.012	0.868	0.055	1,148	--	5,144
	Control Females (n=102)		CL+P Females (n=40)					
I	-0.081	0.931	-0.370	1.176	2.376	1,140	--	5,136
III	-0.054	1.049	-0.248	0.890	1.064	1,140	--	5,136
IV	0.160	1.127	-0.171	0.788	2.880	1,140	--	5,136
V	-0.025	1.097	0.268	1.046	2.108	1,140	--	5,136
IX	0.176	1.037	0.029	1.062	0.569	1,140	--	5,136

*$p < .05$.

the 5 radial and 5 ulnar sums are presented in Table 22. Although nearly all CL±P means are lower than those of controls, only two variables (L3R + R3R; L5U + R5U) achieve univariate significance (until simultaneous confidence bounds are applied). The results are evidence that the factor scores accurately represent the information of the raw data. The three validation procedures have found no differences between means of CL±P probands and controls.

CL±P probands were subdivided according to family history of additional clefts. The two samples (FH+ and FH-) were simultaneously compared with each other and controls to determine whether a positive or negative history produces a systematic effect evident in the means. Table 18 (page 107) gives overall test results and Table 23 the means, standard deviations and analysis of variance tests for the three samples. The proband samples do not differ from controls or one another.

Hotelling's paired T^2 was used to test differences in the means of CL±P or CP probands and their noncleft siblings. Table 24 presents the means and standard deviations for CL±P and CP pairs and means and standard deviations of the difference scores. The test results (Table 25) are not significant for either cleft type.

Factor scores derived from unstandardized data allowed testing for effects due to sex, cleft type (CL±P or CP, control) and interaction between sex and cleft type. Multivariate analysis of variance (manova) tests for cleft effects are mathematically comparable to Hotellings T^2 and provide no additional information. The

Table 22. Means and Standard Deviations of Radial and Ulnar Ridge-Count Sums for Homologous Digits and Anova Tests for CL±P Probands and Controls

Variable	Mean	S.D.	Mean	S.D.	F_{tI}	D.F.	F_{tS}	D.F.
	Control (n=204)		CL+P (n=88)					
L5R+R5R	26.799	10.286	24.568	11.041	2.766	1,290	--	5,286
L4R+R4R	32.083	12.528	28.955	14.117	3.548	1,290	--	5,286
L3R+R3R	22.966	11.243	19.830	12.439	4.483*	1,290	0.884	5,286
L2R+R2R	15.392	12.380	12.886	12.648	2.486	1,290	--	5,286
L1R+R1R	35.706	10.512	32.875	13.395	3.756	1,290	--	5,286
L5U+R5U	3.412	7.594	1.443	4.040	5.263*	1,290	1,038	5,286
L4U+R4U	10.897	13,157	8.909	12.605	1.439	1,290	--	5,286
L3U+R3U	5.049	10.364	5.023	11.097	0.001	1,290	--	5,286
L2U+R2U	13.265	14.796	12.057	14.715	0.411	1,290	--	5,286
L1U+R1U	9.750	12.553	10.261	14.103	0.095	1,290	--	5,286

*p < .05.

Table 23. Factor Score Means, Standard Deviations and Anova Tests for CL±P (FH-) Probands, CL±P (FH+) Probands and Controls

	Controls (n=204)		CL±P (FH-) Probands (n=59)		CL±P (FH+) Probands (n=28)		Test of Significance	
Factor	Mean	S.D.	Mean	S.D.	Mean	S.D.	F_{tI}	D.F.
I	0.084	0.950	-0.173	1.038	-0.196	1.117	2.200	(2,288)
III	-0.001	1.041	0.055	1.026	-0.031	0.686	0.094	(2,288)
IV	0.121	1.137	-0.178	0.743	-0.126	0.719	2.288	(2,288)
V	-0.035	1.084	0.078	0.970	0.037	0.876	0.293	(2,288)
IX	0.105	1.068	0.013	0.895	-0.016	1.106	0.297	(2,288)

*p < .05.

Table 24. Factor Score Means and Standard Deviations for Paired Samples of CL±P Probands and Siblings and CP Probands and Siblings

Factor	Mean	S.D.	Mean	S.D.	Mean	S.D.
	CL±P Proband (n=41)		CL±P Sib (n=41)		Difference	
I	-0.424	1.109	-0.106	0.871	-0.318	1.127
III	-0.160	0.940	-0.188	0.984	0.028	1.149
IV	-0.130	0.605	-0.125	0.908	-0.005	0.932
V	0.110	1.067	-0.174	0.566	0.284	0.955
IX	0.092	0.955	-0.149	0.954	0.241	1.336
	CP Proband (n=15)		CP Sib (n=15)		Difference	
I	-0.219	1.100	-0.023	0.720	-0.196	1.049
III	-0.187	0.848	0.167	0.704	-0.354	1.014
IV	-0.020	0.963	0.197	1.113	-0.217	0.793
V	0.365	1.300	0.548	1.369	-0.183	1.376
IX	-0.161	1.181	-0.272	1.001	0.111	0.870

Note: Means and Standard deviations of the difference scores are also shown.

Table 25. Test Statistics from Multivariate Paired Comparisons, of CL±P or CP Probands and Noncleft Siblings

Comparison Groups	Sample Size	Test of Significance[1]	D.F.
(1) CL±P Probands (2) CL±P Sibs	41 41	$F_t = 0.383$	(5,36)
(1) CP Probands (2) CP Sibs	15 15	$F_t = 0.440$	(5,10)

[1]None of the F-ratios are significant at $\alpha = .05$.

unique contribution of multivariate analysis of variance lies in testing for sex and particularly sex x cleft interactions. Manova test criteria for the hypotheses of no overall sex effect, no overall cleft effect and no interaction effect are presented in Table 26. Factor score means for the appropriate samples are shown in Table 27 and two-way analysis of variance tests for sex, cleft and sex x cleft interaction effects are given in Table 28. The test discloses a significant sex effect when control plus CL±P males and females are examined. Rather surprisingly, the same is not true for smaller samples of control plus CP males and females. The univariate tests suggest rejection of the overall test for sex differences is due to differences in factor I. CP-control samples show this pattern, although not to the same degree. The difference is lost when a simultaneous test is applied. The male mean is higher on factor I than that of females. This factor has high loadings for radial counts on all digits, particularly 3, 4 and 5. Thus, males have larger radial ridge-counts than females. Ulnar counts, interdigital counts and palmar pattern counts do not present a pattern of sex differences. The overall test and univariate tests for interaction between clefts and sex are not significant.

III. UNIVARIATE TESTS FOR SAMPLE DIFFERENCES

Two variables, atd angle and ridge breadth were analyzed separately using analysis of covariance to make adjustments for uncontrolled effects of age. Analysis of covariance is attempted only when the domain of the covariates is roughly similar for all

Table 26. Manova Test Criteria for the Hypothesis of No Overall Sex Effect, No Overall Cleft Effect and No Overall Sex X Cleft Interaction Effect

Test Effect and Comparison Groups	Sample Size	Manova Test Criteria	D.F.	Significance Level
Sex				
(1) Control and CL±P Males	150			
(2) Control and CL±P Females	142	$F_t = 3.39$	5,284	.01 > p > .005
Cleft Type				
(1) Controls	204			
(2) CL±P Probands	88	$F_t = 1.92$	5,284	.10 > p > .05
Sex X Cleft Type		$F_t = 0.90$	5,284	.50 > p > .45
Sex				
(1) Control and CP Males	117			
(2) Control and CP Females	116	$F_t = 1.25$	5,225	.30 > p > .25
Cleft Type				
(1) Controls	204			
(2) CP Probands	29	$F_t = 1.05$	5,225	.40 > p > .35
Sex X Cleft Type		$F_t = 0.30$	5,225	.95 > p > .90

Table 27. Factor Score Means for Samples Compared by a Two Factor Multivariate Analysis of Variance

Sample	Sample Size	Factor Score Means				
		I	III	IV	V	IX
Controls	204	.089	-0.003	0.123	-0.035	0.103
CL+P Probands	88	-0.193	0.023	-0.177	0.063	0.007
Males	150	0.161	0.112	0.000	-0.066	0.017
Females	142	-0.163	-0.109	0.067	0.058	0.134
Controls	204	0.089	-0.003	0.123	-0.035	0.103
CP Probands	29	-0.021	-0.183	0.188	0.335	-0.142
Males	117	0.231	0.010	0.072	0.019	-0.008
Females	116	-0.082	-0.061	0.189	0.003	0.154

Note: Factor scores were derived from nonstandardized data.

Table 28. Two-way Anova Tests for Sex, Cleft and Sex X Cleft Interaction Effects for Male and Female Probands and Controls

Source	Dependent Variables (Factors) I F_{tI}	D.F.	III F_{tI}	D.F.	IV F_{tI}	D.F.	V F_{tI}	D.F.	IX F_{tI}	D.F.
Sex (Male-Female)	7.90*	1,288	3.53	1,288	0.31	1,288	1.03	0.31	0.94	1,288
Cleft (CL±P-Control)	5.58*	1,288	0.02	1,288	5.04*	1,288	0.62	0.43	0.47	1,288
Sex X Cleft	0.00	1,288	2.36	1,288	.05	1,288	1.79	0.18	0.16	1,288
Sex (Male-Female)	5.79*	1,229	0.29	1,229	0.61	1,229	0.01	1,229	1.35	1,229
Cleft (CP-Control)	0.34	1,229	0.82	1,229	0.09	1,229	2.91	1,229	1.32	1,229
Sex X Cleft	0.30	1,229	0.34	1,229	0.59	1,229	0.41	1,229	0.07	1,229

*$p < .05$.

groups. The clinic sample includes a large number of individuals less than 15 years of age. Therefore, the control series was designed so it contains several children allowing age overlap. Ages were designated in years according to the closest birthdate. Adults, age 17 and older, were assigned an age of 17. Changes in these variables do not occur after growth ceases. Males and females were examined separately. Controls and both proband samples are compared in the same analysis.

Analysis of covariance problems have three essential tests. The first checks for equality of the regression slopes of each sample. The null hypothesis of equal slopes must be accepted before additional tests are possible. Otherwise, further tests concerning average differences in the variate, as adjusted for the covariate age, are invalid. The second test determines whether a linear regression exists between the variate and covariate. The null hypothesis states the regression slope is zero. The final test determines whether the means of the three groups (adjusted for differences on the covariate) are equal. The three test results are shown for atd angle (measured as left atd angle plus right atd angle) and ridge breadth in Table 29. Test 1 for atd angle allows acceptance of the null hypothesis indicating similarity of the populations' regression slopes. Test 2 is significant meaning there is a linear regression between age and atd angle. As expected, the regression slope (not shown) is negative describing the known inverse relationship between these variables. The final F-ratio is nonsignificant in both males and females. Inspection of the adjusted sample means (Table 30) reveals only slight

Table 29. Analysis of Covariance Tests for atd angle and Ridge Breadth of CL+P Probands, CP Probands and Controls

Significance Tests	Males			Females		
	F Value	D.F.	Probability	F Value	D.F.	Probability
atd Angle						
Test 1	2.29	2,161	.15 > p > .10	0.760	2,140	.50 > p > .45
Test 2	13.214	1,163	.0005 > p > .0001	13.550	1,142	.0005 > p > .0001
Test 3	0.520	2,163	.65 > p > .60	0.370	2,142	.70 > p > .65
Ridge Breadth						
Test 1	0.080	2,159	.95 > p > .90	1.000	2,150	.40 > p > .35
Test 2	215.939	1,161	.0001 > p	101.173	1,152	.0001 > p
Test 3	0.454	2,161	.65 > p > .60	4.513	2,152	.05 > p > .01

Note: Test 1 tests the equality of the individual regression slopes. Test 2 determines whether there is a linear regression between the variates and the covariate age. Test 3 compares the adjusted means of the three groups.

Table 30. Adjusted Means for atd Angle and Ridge Breadth

Sample	atd Angle[1]				Ridge Breadth[2]			
	Males		Females		Males		Females	
	Sample Size[3]	Mean	Sample Size	Mean	Sample Size	Mean	Sample Size	Mean
CL+P Probands	50	88.320	39	90.767	48	557	40	552
CP Probands	15	87.760	12	87.061	15	550	14	501
Controls	102	85.692	95	88.583	102	549	102	496

[1]Left atd angle + Right atd angle.

[2]$(D_L + D_R) / (C_L + C_R + 2) \times 1000$.

[3]Note: Sample sizes for atd angle differ slightly from those reported in Table 1. Brother and sister probands were included since males and females were analyzed separately. Print quality occasionally prevented measuring atd angle on all prints.

differences between controls and either proband series. In general, males have smaller angles than females.

Ridge breadth has a very strong regression on age in both sexes (Table 29, Test 2). The slopes, which are similar between samples (Test 1) are positive signifying increased ridge breadth with advancing age. The final test (Test 3) of the adjusted means attains significance in the case of females, but not males. Females CL±P probands have greater ridge breadth than corresponding CP probands or controls (Table 30). The same trend occurs in males even though the differences are not as pronounced.

IV. FLUCTUATING ASYMMETRY VALUES FOR CONTROL AND CLINIC SAMPLES

Asymmetry variance-covariance matrices of eight clinic samples, including CL±P (FH+) and CL±P (FH-) probands, were individually compared with controls using a multivariate test for dispersion homogeneity (Kendall and Stuart 1966). The 18 asymmetry variables on which the matrices are based represent contrasts between left and right homologous variables (i.e. $d_k = left_k - right_k$). Table 31 presents the mean asymmetry values and variances for digital and palmar ridge-counts of all samples. The means measure directional asymmetry, the sign indicating the direction. Positive values denote larger average counts on the left hand. The variances represent fluctuating asymmetry. These values constitute the diagonal elements of the various dispersion matrices. Chi square statistics from the comparisons are shown in Table 32. CL±P probands (total

Table 31. Mean Asymmetry Values and Variances for Digital and Palmar Ridge-Counts of Controls and Clinic Samples

Asymmetry Variable	Mean	Variance	Mean	Variance	Mean	Variance	Mean	Variance
	Controls (n=204)		CL±P Probands (n=88)		CL±P (FH+) Probands (n=28)		CL± (FH-) Probands (n=59)	
L5R-R5R	0.064	12.001	0.182	17.415	-0.214	6.915	0.390	22.759
L5U-R5U	0.108	13.456	-0.807	9.675	-1.071	9.476	-0.695	10.043
L4R-R4R	0.152	19.647	-0.227	25.810	-0.393	17.507	-0.153	30.545
L4U-R4U	-1.436	44.740	-1.773	31.166	-2.571	35.661	-1.424	29.662
L3R-R3R	0.240	20.400	1.011	38.126	2.571	26.106	0.271	43.305
L3U-R3U	-0.186	37.305	0.273	15.810	0.286	15.101	0.271	16.684
L2R-R2R	-0.775	37.377	-0.159	35.354	-0.500	26.407	0.000	40.655
L2U-R2U	-1.255	47.068	-0.125	31.237	-1.250	29.750	0.407	32.108
L1R-R1R	-2.549	19.254	-2.330	18.384	-2.107	7.210	-2.458	24.149
L1U-R1U	-2.407	52.095	0.057	39.548	0.607	25.581	0.051	43.359
LCD-RCD	-1.279	34.409	-0.886	30.010	-1.429	32.476	-0.678	29.567
LBC-RBC	-0.549	14.929	-0.477	16.229	-0.107	14.840	-0.678	17.291
LAB-RAB	0.593	18.972	0.989	14.609	1.821	23,337	0.593	10.556
LDRad-RDRad	1.284	38.914	0.341	27.859	0.107	13.507	0.458	35.459
LCUln-RCUln	-0.922	47.344	-0.011	31.161	-0.286	32.286	0.136	31.636
LCRad-RCRad	-2.103	21.147	-2.568	20.524	-2.107	24.099	-2.831	19.281
LDAccess-RDAccess	0.000	6.177	-0.420	8.568	-0.036	3.665	-0.610	11.035
LAAccess-RAAccess	-0.152	1.056	-0.170	1.223	0.107	0.618	-0.305	1.492
atd angle	-0.102	28.704	0.375	75.961				

Table 31 (Continued)

Asymmetry Variable	Mean	Variance	Mean	Variance	Mean	Variance	Mean	Variance	Mean	Variance
	CL±P Sibs (n=43)		CL±P Parents (n=119)		CP Probands (n=29)		CP Sibs (n=15)		CP Parents (n=36)	
L5R-R5R	0.116	18.819	0.529	13.861	-1.862	3.552	-0.600	7.400	-0.500	14.257
L5U-R5U	-0.628	6.715	-0.588	9.261	-1.172	8.291	-1.467	18.552	-0.556	4.540
L4R-R4R	-0.465	11.207	-1.227	22.601	-1.034	8.892	-0.533	34.124	-0.389	15.044
LrU-R4U	-0.535	24.350	-2.664	41.039	-2.172	23.433	-2.333	32.095	-1.722	41.578
L3R-R3R	-0.395	15.530	-0.336	33.344	-0.138	15.195	-2.867	12.124	0.306	12.961
L3U-R3U	0.279	32.682	1.017	30.457	-0.690	34.793	-0.667	20.238	-0.417	18.079
L2R-R2R	0.860	37.171	-0.286	52.765	-0.172	28.576	-0.267	17.353	0.194	26.904
L2U-R2U	-1.791	37.360	-0.050	43.014	-2.724	67.778	0.867	48.838	0.417	46.650
L1R-R1R	-1.837	11.473	-3.361	18.978	-1.724	17.207	-2.200	5.743	-1.917	20.707
L1U-R1U	-1.233	33.802	-1.151	35.265	-2.241	35.975	-2.200	14.743	-1.917	47.564
LCD-RCD	-2.070	19.447	-1.958	28.464	-1.586	21.894	-3.133	14.124	-1.500	26.086
LBC-RBC	-0.791	13.836	-0.454	13.436	-1.069	16.567	0.200	16.029	-1.778	17.949
LAB-RAB	0.744	11.957	0.521	14.642	0.862	10.837	1.000	7.143	0.444	16.883
LDRad-RDRad	0.419	34.011	1.000	32.949	0.034	41.677	0.467	23.838	1.667	24.514
LCU1n-RCU1n	0.256	26.338	0.092	36.796	0.586	20.108	-0.400	8.257	-1.389	58.130
LCRad-RCRad	-1.535	11.207	-2.529	23.811	-2.862	15.337	-3.133	25.410	-2.611	29.273
LDAccess-RDAccess	-0.558	7.300	-0.244	4.101	0.207	5.741	0.000	0.000	-0.306	2.218
LAAccess-RAAccess	-0.186	0.774	-0.185	1.355	-0.138	0.552	-0.467	5.838	-0.222	1.949
atd angle					-0.929	83.476				

Table 32. Tests for Variance-Covariance Homogeneity of Asymmetry Matrices

Comparison Groups	Sample Size	Chi Square	D.F.	Significance Level
(1) CL±P Probands (2) Controls	88 204	220.259	171	.01 > p > .005
(1) CL±P (FH+) Probands (2) Controls	28 204	181.293	171	.30 > p > .25
(1) CL±P (FH-) Probands (2) Controls	59 204	226.548	171	.005 > p > .001
(1) CL±P Sibs (2) Controls	43 204	216.392	171	.05 > p > .01
(1) CL±P Parents (2) Controls	119 204	192.856	171	.15 > p > .10
(1) CP Probands (2) Controls	29 204	218.448	171	.01 > p > .005
(1) CP Sibs (2) Controls	15 204	241.442	171	.0005 > p > .0001
(1) CP Parents (2) Controls	36 204	192.754	171	.15 > p > .10

sample) CL±P noncleft siblings, CP probands and CP noncleft siblings differ from controls. Most differences are highly significant. The chi squares are on the same scale and if interpreted suggest CL±P probands differ more than their siblings. CP probands and their siblings show the reverse pattern. However, the number of CP siblings is quite small. When CL±P probands are subdivided into FH+ and FH- groups, only patients without a history of clefts (FH-) show a difference. Neither parent test reaches significance at a 5 percent level of confidence.

The individual variances for the unique samples were tested for homogeneity with controls using a two-tailed analysis of variance test (Table 33). The tests achieve significance for 5-7 variables in each series. However, the tests are not independent. If a simultaneous confidence level was applied, several ratios would lose significance. Nevertheless, the variances of the dispersion matrices show some heterogeneity. There is no consistent pattern across samples for the variables or types of variables showing asymmetry differences. All variable classes (as finger ridge-counts or interdigital counts) have significant ratios in some instances. The expected tendency for increased clinic sample asymmetry is missing. In some cases, the clinic variances are larger, but more often the reverse is true.

The asymmetry of atd angle was examined for controls, CL±P probands and CP probands; these means and variances are included in Table 31. The measure was calculated as left atd minus right atd angle. Palms were not excluded if having a large angle on one hand because of the unilateral presence of a hypothenal pattern. Thus

Table 33. Analysis of Variance Tests for Asymmetry Score Variance Heterogeneity

Asymmetry Variable	Samples Compared: (1) CL+P Probands (2) Controls			(1) CL+P(FH-)Probands (2) Controls			(1) CL+P Sibs (2) Controls			(1) CP Probands (2) Controls			(1) CP Sibs (2) Controls		
	SLV	F_{tI}	D.F.	SLV	F_{tI}	D.F.	SLV	F_{tI}	D.F.	SLV	F_{tI}	D.F.	SLV	F_{tI}	D.F.
L5R-R5R	1	1.451	87,203	1	1.896*	58,203	1	1.568	42,203	2	3.379*	203,28	2	1.622	203,14
L5U-R5U	2	1.391*	203,87	2	1.340	203,58	2	2.004*	203,42	2	1.623	203,28	1	1.379	14,203
L4R-R4R	1	1.314	87,203	1	1.555*	58,203	2	1.753*	203,42	2	2.210*	203,28	1	1.737	14,203
L4U-R4U	2	1.436*	203,87	2	1.508	203,58	2	1.837*	203,42	2	1.909*	203,28	2	1.394	203,14
L3R-R3R	1	1.869*	87,203	1	2.123*	58,203	2	1.314	203,42	2	1.343	203,28	2	1.683	203,14
L3U-R3U	2	2.360*	203,87	2	2.236*	203,58	2	1.141	203,42	2	1.072	203,28	2	1.843	203,14
L2R-R2R	2	1.057	203,87	1	1.088	58,203	2	1.006	203,42	2	1.308	203,28	2	2.154	203,14
L2U-R2U	2	1.507*	203,87	2	1.466	203.58	2	1.260	203,42	1	1.440	28,203	1	1.038	14,203
L1R-R1R	2	1.047	203,87	1	1.254	58,203	2	1.678	203,42	2	1.119	203,28	2	3.353*	203,14
L1U-R1U	2	1.317	203,87	2	1.201	203,58	2	1.541	203,42	2	1.448	203,28	2	2.682*	203,14
LCD-RCD	2	1.147	203,87	2	1.164	203,58	2	1.769*	203,42	2	1.572	203,28	2	2.436	203,14
LBC-RBC	1	1.087	87,203	1	1.158	58,203	2	1.079	203,42	1	1.110	28,203	1	1.074	14,203
LAB-RAB	2	1.299	203,87	2	1.797	203,58	2	1.587	203,42	2	1.751	203,28	2	2.656*	203,14
LDRad-RDRad	2	1.397*	203,87	2	1.097	203,58	2	1.144	203,42	1	1.071	28,203	2	1.632	203,14
LCUln-RCUln	2	1.519*	203,87	2	1.497	203,58	2	1.798*	203,42	2	2.354*	203,28	2	5.734*	203,14
LCRad-RCRad	2	1.030	203,87	2	1.097	203,58	2	1.887*	203,42	2	1.379	203,28	1	1.202	14,203
LDAccess-RDAccess	1	1.387	87,203	1	1.786*	58,203	1	1.182	42,203	2	1.076	203,28	2	-----	203,14
LAAccess-RAAccess	1	1.158	87,203	1	1.413	58.203	2	1.364	203,42	2	1.913*	203,28	1	5.528*	14,203
atd angle	1	2.646*	87,196							1	2.908*	27,196			

Abbreviation: SLV--Sample with largest variance.

*p < .05.

Note: The F-ratios are two-tailed tests and do not have simultaneous confidence.

the variance includes hypothenar pattern asymmetry as one component. Analysis of variance tests for proband-control similarity are shown in Table 33. Both CL+P and CP probands have significantly larger variances for atd angle than controls.

CHAPTER VI

DISCUSSION

Investigations of the interrelationships among quantitative dermatoglyphic variables have relied on factor analysis to simplify or clarify the patternings of variable intercorrelations. Beginning with Knussman's (1967) factor analyses of English (Holt 1951, 1959) and Parsis' (Mavalwala 1962) ten finger ridge-count (each finger represented by its largest count) correlation matrices, three finger factors were tentatively identified. One factor referred to ridge-counts on left and right thumbs, a second, the radiomedial factor, related to digits II and III, while a third was correlated with ridge-counts on the little fingers. Those factors remained even when additional variables such as finger pattern intensity, palmar main line terminations, atd angles, or ridge-counts on toes were included (Knussman 1967; Nance et al. 1974). Palmar variables as interdigital ridge-counts or atd angles were correlated with new separate factors. Knussman (1969) identified nine hypothetical factors following analysis of ten finger ridge-counts, pattern intensity for each finger and sixteen palmar variables including six interdigital ridge-counts (a-b, b-c and c-d). Besides three finger factors and three palmar main line factors in accordance with Knussman (1967), three new factors with symmetrical loadings for left and right hands were identified: an atd angle factor, a II-IV interdigital count factor, and a third interdigital count factor. Knussman (1967) had previously

defined three factors as relevant to left and right interdigital ridge counts using the correlation matrix provided by Baitsch and Schwarzfischer (1959). Salient loadings for second and fourth interdigital counts involved distinct factors.

Further understanding of the biological components affecting the development of dermal ridges on various digits seems possible when basing the analysis on twenty ridge-counts, each finger having a radial and ulnar count. Jantz and Owsley (1977) demonstrated relative independence of the radial and ulnar sides of the fingers. Only after this initial dichotomy did radial and ulnar counts generally result in two factors relating to counts on the median or ulnar digits. Factors reflecting the relative independence of the thumb were also indicated. Somewhat similar conclusions concerning the radial-ulnar contrast between finger sides or the difference between medial and lateral digits were also obtained by Roberts and Coope (1975) using a principal components solution without rotation. Those solutions possibly represented the most accurate delineation of the developmental components affecting finger dermatoglyphics. Yet, to a certain degree, the number of biologically meaningful finger and palmar factors remains somewhat ill-defined. Few studies have considered both radial and ulnar finger counts or simultaneously incorporated finger and palmar variables in one analysis.

A total of 36 finger and palmar ridge-counts were factor analyzed in the present study. The variables include a new series of quantitative measures of palmar interdigital pattern size. Interdigital patterns were counted using a system somewhat similar to that

of finger patterns (Jantz 1977b). The counts specify the directionality of the main lines forming the patterns. Such information is seemingly necessary since recent studies emphasize the importance of the C line to third and fourth interdigital pattern formation (Rife 1968a, 1968b) and the presence of considerable racial variation in C line terminations (Plato 1970). Preliminary tests of this new set of variables using data from India provide evidence indicating its usefulness in biological distance studies (Jantz and Chopra 1977). As quantitative variables, the statistical methods available for comparing populations are less restrictive than if the patterns were handled in a qualitative sense. Learning how the variables are reproduced by the factors may increase our understanding of the developmental forces affecting interdigital pattern formation.

Four of the 10 factors extracted for controls concern ridge-counts on digits 2-5 with medial and lateral digits contributing to separate factors. As noted previously (Jantz and Owsley 1977), the factors distinguish radial and ulnar counts. However, this separation is less complete than initially indicated. The radial and ulnar count factors in all samples often include significant loadings for counts on the opposing side. In factor analysis of the total sample, a positive interrelationship between radial and ulnar counts is indicated by correlations between their respective factors. The higher oblique factor correlations involve radial and ulnar count factors. There is a difference between CL+P probands and controls in the finger count factors. CL+P probands, and also the total sample, define single

radial count factors weighing digits 2 through 5. This pattern suggests higher intercorrelations among radial finger ridge-counts of CL±P probands. A higher average correlation would account for the larger first eigenvalue found for this sample.

Controls and CL±P probands also contrast in relative contributions of radial and ulnar thumb counts to specific factors. Two thumb factors, one emphasizing ulnar and the other radial counts (plus radial counts on 2 and 3), were identified in controls. CL±P probands and the total sample have single factors emphasizing both radial and ulnar components. Whether this difference is biologically meaningful is difficult to assess. Both patterns have been observed in factorial solutions of various populations (Jantz and Owsley 1977). There appears to be a general tendency for thumb counts to contribute to unique factors rather than loading highly with other digital counts. The thumb's relative independence probably reflects effects of structural and developmental uniqueness affecting dermal ridge formation. The thumb's growth orientation and timing for initial separation from the index finger does differ from other digits (Arey 1965).

Finger and palmar ridge-counts are not strongly related. With rare exceptions, factors which correlate with finger variables express no relationship with palmar counts. This finding corresponds with low cross correlations reported by Loesch (1971) for palmar and fingertip patterns. Palmar pattern counts express little correlation with interdigital count factors. The size or presence of interdigital patterns apparently responds to different influences than those determining size or width of the interdigital areas as measured in ridge-counts.

Each sample has two interdigital count factors, although there is variability in the specific loading expressions. Controls have a single well defined factor loading all six counts. The second control factor suggests some independence of fourth interdigital counts as it accounts for a small proportion of their variance. In contrast, CL+P probands combine third and fourth interdigital counts in a single factor while separating a-b counts. The total sample's pattern is more similar to probands in combining third and fourth interdigital counts in one factor while emphasizing second interdigital counts in another. However, the latter factor combines second and fourth interdigital counts by loading fourth interdigital counts to a greater degree than probands. A II, IV factor has been identified previously by Knussman (1969).

Two of the four palmar pattern factors are consistent across samples by sharing common emphases on fourth interdigital pattern counts formed by main line D or third and fourth interdigital patterns form by C. Those two factors are essentially independent revealing only slight evidence for identical developmental components affecting C and D directionality or size of the associated patterns. The factor with high loadings for LDRad and RDRad does have low negative loadings for LCUln suggesting some interaction between measures. However, the association has unilateral occurrence and lacks constancy in all samples. Fourth interdigital patterns associated with accessory d triradii express no relationship (not even a negative one) with either factor. A negative correlation would seem meaningful since ulnar

curvature of C, radial curvature of D or the presence of d accessory triradii represent common ways of forming simple patterns in area IV. When one option is followed, the chance for additional patterns formed by either of the other two lessons. Nevertheless, d accessory counts are largely independent of all variables and are not accurately reproduced by the factor pattern matrices. Second interdigital pattern counts also relate poorly with the other variables and the solutions have difficulty integrating LAAccess and RAAccess into factors.

Much of the analysis focused on comparing mean values of controls with various clinic samples. Several reasons have been suggested as developmental mechanisms possibly leading to dermatoglyphic abnormalities in probands. These factors include teratogenic interference affecting overall development, delayed or increased rates of intrauterine growth and the association of additional malformations, particularly defects of the hands. Few differences in the means are discernible between any of the clinic series and controls. A possible exception is a significantly higher tendency for increased ridge breadth in CL±P females. Only CL±P females significantly differ from controls in ridge breadth although the value for CL±P males is increased. According to the multifactorial concept for the etiology of CL±P, affected individuals lie beyond a certain developmental threshold. Fraser (1970) has pointed out that in sex related traits such as CL±P, it is assumed that the threshold is nearer the tail of the distribution in the sex which is less often affected. Since CL±P occurs more frequently in males it has been assumed that female probands have a larger number

of predisposing genes than corresponding males. Possibly the tendency for increased ridge breadth in CL±P females and to a lesser extent CL±P males is somehow related to the genetic background involved in cleft formation. Wider ridges may reflect reduced rates of intrauterine growth as indicated by an association with chromosomal compliments involving additional X or Y chromosomes. There are indications of slightly lower ridge-counts for probands but the differences are not significant. If growth rates are slower in children with oral clefts, the effects are not readily apparent in the dermatoglyphics. When male and female probands were examined separately, the means were similar to corresponding controls. Predictions based on a theoretical extension of the Meskin et al. (1968) model were not confirmed. Male proband means were not less than controls, female proband means were not greater than controls, and probands do not show decreased sexual dimorphism. These findings are not evidence against the model because the data are not intimately related to palatomorphogenesis. On the other hand, the tests are not supportive either. The data provide no evidence that a positive or negative family history of oral clefts has any effect on mean values. Noncleft and proband siblings are not different from one another in their means.

Dermatoglyphics may not seem useful in detecting differential growth rates in embryos developing oral clefts. However, it is possible that the actual differences from noncleft embryos are quite minor even when measured using other quantitative variables such as birth weight. The differences reported for birth weights of cleft patients are not very large (Green et al. 1964; Meskin 1966). Cleft lip and

cleft palate may not be associated with markedly different rates of embryological growth.

Previous studies have reported a few mean differences between probands and controls in variables such as total ridge-count, frequencies of interdigital patterns and atd angle (Achs et al. 1966; Dzuiba 1972; Piatkowska and Sokolowski 1972, 1973; Potrzebowski 1974; Usoev 1972; Wittwer 1967). These results are not confirmed in this analysis or in studies by Silver (1966) and De Bie et al. (1977). Many of the reports mentioning contrasts are based on small samples and univariate testing procedures. The differences noted may have resulted from examining large numbers of variables without simultaneously controlling confidence limits.

If dermatoglyphics are used as a guideline, clefts symbolize abnormal interrelationships among the developing structures. The clinic samples are unusual in the dispersion matrices for factor scores and asymmetry measures. CL±P probands, siblings and parents significantly depart from controls in variance-covariance homogeneity; probands being the extreme in this regard. When individual variances are examined only a few display heterogeneity suggesting the lack of a well defined effect involving only the variances. Interrelationships among the variables, or covariances, probably display different patternings than found in normal individuals. This tendency seems traceable to the original variables even though factor scores represent the units of observation tested. Factor structure differences between CL±P probands and controls, as in the thumb or radial count factors, show different factor to variable correlations which implies different

relationships among the variables themselves. When factor structures of CL±P probands and controls are compared, a few variables, notably L3U, R5U, RIU, RBC, RBC, LAAccess display lower correlations between factor solutions than other variables. These variables, when represented as vectors, apparently occupy differing aspects of multivariate space. Such variation becomes manifest in subtle factor structure contrasts between samples as well as meaning different correlations among the original variables as far as their interrelationship with those designated as being somewhat unique.

Asymmetry matrices for CL±P probands, their noncleft siblings, CP probands and their siblings differ from controls although not the parent samples. It is interesting the CP samples indicate high significance probabilities for asymmetry and yet reveal no differences when considering factor score dispersion matrices. The asymmetry between homologous variables and the interrelationships among these measures are components (measured as correlations between homologous and non-homologous variables) of the original 36 x 36 correlation matrix analyzed to obtain factor scores. CL±P parents differ from controls in factor score dispersion matrices but not in asymmetry. The contrast in factor score dispersions, however, is not as pronounced as for their cleft and noncleft offspring.

The suggestion of covariance differences between controls and certain clinic samples seems particularly relevant as far as cleft formation. The development of the face involves a highly coordinated or correlated interaction of several embryological structures. Deviation from normal developmental pathways, as involved in the

formation of cleft lip and cleft palate, would seem a likely possibility when normal relationships are not maintained among developing facial structures. Because the variables examined bear no direct relationship with the actual morphogenesis of the face, the types of covariance differences indicated in the dermatoglyphics are probably fairly generalized throughout the individual. The presence of additional malformations in CL±P and CP probands and also their siblings is certainly compatible with this concept. Since the variance-covariance differences are not restricted to probands, being present in lesser degrees in noncleft siblings and parents (in the case of CL±P), suggests a genetic basis as being responsible.

When the asymmetry dispersion matrices for CL±P probands were determined according to family history, only the FH- (not FH+) subsample significantly differed from controls. This result seems compatible with Fraser's (1970) comments implying that generalized developmental instability could be involved when CL±P occurs without a familial background. However, the results are in contrast with those of previous dermatoglyphic studies (Adams and Niswander 1967; Woolf and Gianas 1976, 1977). Additional research is necessary to clarify the issue.

When fluctuating asymmetry values (the intra-pair variances) are individually inspected and compared with controls, the results are somewhat inconsistent. The majority of asymmetry scores reveal no difference between controls and selected clinic samples (those differing in an overall test of variance-covariance homogeneity). A

number of variables do indicate increased asymmetry in one sample although a constant effect is lacking. The clinic samples are not always the ones with increased variance. In fact, more often controls are the ones possessing the larger variance. Any reverse in expected patterns of fluctuating asymmetry (i.e. where controls indicate greater asymmetry than affected individuals) does not support the theory that fluctuating asymmetry measures developmental stability. Perhaps it is relevant in this regard that Hook et al. (1971) found greater dermatoglyphic fluctuating asymmetry in controls than in infants with rubella embryopathy. Smaller variances could signify the presence of shared environmental or genetic factors related to cleft formation among individuals in the clinic samples. This type of commonality could result in increased homogeneity relative to a control sample where membership is less defined being determined only by the absence of a cleft.

Two of the variables, atd angle and a-b interdigital ridge-counts, are comparable to those examined in previous studies. Adams and Niswander (1967) and Woolf and Gianas (1976) have reported increased atd angle asymmetry for CL±P probands with a positive family history but no difference between controls and CP probands or CL±P FH- probands. Both CL±P and CP probands show greater variances than controls in this study, although family history was not taken into account. The measure used does vary from earlier studies by including hypothenar pattern asymmetry. The a-b interdigital counts reveal no tendency for greater asymmetry in present tests although Woolf and Guianas (1977) found increased asymmetry in CL±P FH+ probands and first degree relatives.

The approach followed in this study will hopefully prove useful to others investigating the dermatoglyphics of cleft lip and cleft palate or other medical disorders. Multivariate testing procedures are one technique for avoiding spurious results and yet identifying significant differences when examining large numbers of correlated measures. The emphasis placed on hypothesis testing based on a theoretical or developmental framework may also prove useful. Dermatoglyphic studies are often cast within a descriptive framework of searching for differences but not explaining them in developmental terms. While it is valid to search for differences because of diagnostic objectives such that the abnormalities characterizing a syndrome are to be used in clinical diagnosis or screening, this approach has not proved fruitful. Dermatoglyphic anomalies are useful in diagnosis of a few chromosomal defects as Down's syndrome or trisomy E. However, this usefulness is tempered by tne realization that more accurate and efficient means, as karyotyping, are available to assist diagnosis. Instead, dermatoglyphics may eventually have its greatest contribution to medicine in helping understand the causes of malformations or the growth patterns associated with particular clinical syndromes. Dermatoglyphic variables, as developmental phenomena, seem potentially useful as indicators of developmental control or as measures of growth rates. Increasing emphasis should be placed on dermal ridges as tools for this type of hypothesis testing to determine whether this approach is valid. Future studies should also focus on family members in addition to the propositi. When siblings and parents have been examined, as in Down's syndrome (Penrose 1954; Priest et al. 1973) and cleft lip

and cleft palate, differences have been reported. Much information concerning the etiology of cleft lip and cleft palate may be gained by additional dermatoglyphic study of the relatives of cleft probands separated according to family history of clefts.

BIBLIOGRAPHY

BIBLIOGRAPHY

Achs, R., R.G. Harper and N.J. Harrick
1966 Unusual dermatoglyphics associated with major congenital malformations. New England Journal of Medicine 275:1273-1278.

Adams, M.S. and J.D. Niswander
1967 Developmental "noise" and a congenital malformation. Genetic Research 10:313-317.

Altemus, L.A.
1966 The incidence of cleft lip and palate among North American Negroes. Cleft Palate Journal 3:357-361.

Altemus, L.A. and A.D. Ferguson
1965 Comparative incidence of birth defects in Negro and White children. Pediatrics 36:56-61.

Alter, M.
1965 Is hyperploidy of sex chromosomes associated with reduced total finger ridge count? American Journal of Human Genetics 17:473-475.

1966 Dermatoglyphic analysis as a diagnostic tool. Medicine 46:35-56.

Alter, M., R. Gorlin, J. Yunis, F. Peagler and H. Bruhl
1966 Dermatoglyphics in XXYY Klinefelter's syndrome. American Journal of Human Genetics 18:507-513.

Alvesalo, L., R.H. Osborne and M. Kari
1975 The 47, XYY male, Y chromosome, and tooth size. American Journal of Human Genetics 27:53-61.

Arey, L.B.
1965 Developmental Anatomy. Philadelphia: W.B. Saunders Company.

Azaz, B. and E. Koyoumdjisky-Kaye
1967 Incidence of clefts in Israel. Cleft Palate Journal 4:227-233.

Babler, W.J.
1978a Prenatal selection and dermatoglyphic patterns. American Journal of Physical Anthropology 48:21-27.

1978b Quantitative differences in morphogenesis of human epidermal ridges (in press).

Bader, R.S.
1965 Fluctuating asymmetry in the dentition of the house mouse. Growth 29:291-300.

Bailit, H.L., P.L. Workman, J.D. Niswander and C.J. MacLean
1970 Dental asymmetry as an indicator of genetic and environmental conditions in human populations. Human Biology 42: 626-638.

Baitsch, H. and F. Schwarzfischer
1959 Zur Biometrie und geographischen Verteilung der Leistenzahlen in den Interdigitalfeldern der Handflache. Homo 10:226-236.

Bardanouve, V.T.
1969 Cleft palate in Montana: A 10-year report. Cleft Palate Journal 6:213-220.

Barlow, P.
1973 The influence of inactive chromosomes on human development. Humangenetik 17:105-136.

Barr, A.J., J.H. Goodnight, J.P. Sall and J.T. Helwig
1976 A User's Guide to SAS 76. Raleigh: Sparks Press.

Bartlett, M.S.
1950 Tests of significance in factor analysis. British Journal of Psychology 3:77-85.

Bear, J.C.
1973 The association of fetal wastage with facial cleft conditions. Cleft Palate Journal 10:346-359.

Beardmore, J.A.
1960 Developmental stability in constant and fluctuating temperatures. Heredity 14:411-422.

Beckman, L., K.H. Gustavson and A. Norring
1962 Finger and palm dermal ridge patterns in normal and mongoloid individuals (the Down syndrome). Acta Genetica et Statistica Medica 12:20-27.

Beckman, L. and A. Norring
1963 Finger and palm prints in schizophrenia. Acta Genetica et Statistica Medica 13:170-177.

Berg, J.M.
1968 Observations on the thenar/first interdigital dermatoglyphic patterns in mongolism. Journal of Mental Deficiency Research 12:307-311.

Berg, J.M., G.F. Smith, M.A.C. Ridler, G. Dutton, E.A. Green and B.W. Richards
1966 On the association of broad thumbs and first toes with other physical pecularities and mental retardation. Journal of Mental Deficiency Research 10:204-220.

Berndorfer, A.
1970 The importance of the individual biological constitution in the development of the various forms of cheilo-gnatho-palatoschisis. Cleft Palate Journal 7:502-505.

Biggerstaff, R.H.
1969 Classification and frequency of cleft lip and/or palate. Cleft Palate Journal 6:40-45.

Bingle, G.J. and J.D. Niswander
1977 Maternal effects in human cleft lip and palate. American Journal Human Genetics 29:605-609.

Bishara, S.E. and W.W. Iversen
1974 Cephalometric comparisons on the cranial base and face in individuals with isolated clefts of the palate. Cleft Palate Journal 11:162-175.

Bolling, D.R., D.S. Borgaonker, H.M. Herr and M. Davis
1971 Evaluation of dermal patterns in Down's syndrome by predictive discrimination. II. Composits score based on the combination of left and right pattern areas. Clinical Genetics 2:163-169.

Bonnevie, K.
1924 Studies on papillary patterns of human fingers. Journal of Genetics 15:1-111.

Borgaonkar, D.S., D.R. Bolling and H.M. Herr
1973 Evaluation of dermal patterns in the diagnosis of the Down syndrome by predictive discrimination. III. Variations due to sex and ethnic background and its effect on the use of indices. Human Heredity 23:442-453.

Borgaonkar, D.S., M. Davis, D.R. Bolling and H.M. Herr
1971 Evaluation of dermal patterns in Down's syndrome by predictive discrimination. I. Preliminary analysis based on frequencies of patterns. John Hopkins Medical Journal 128: 141-152.

Breg, W.R., M.W. Steele, O.J. Miller, D. Warburton, A. de Capoa and P.W. Allderdice
1970 The Cri-du-Chat syndrome in adolescents and adults: Clinical findings in 13 patients with partial deletion of the short arm of chromsome No. 5 (5p-). Journal of Pediatrics 77:782-791.

Brehme, H.F., H.F. Härle
1971 Über die Hautleistenbefunde von 18 Patienten mit Robin-Syndrom Humangenetik 13:163-165.

Brown, K.S., M.C. Johnston and P.F. Murphy
1974 Isolated cleft palate in A/J mice after transitory exposure to drinking-water deprivation and low humidity in pregnancy. Teratology 9:151-158.

Brown, K.S., M.C. Johnston and J.D. Niswander
1972 Isolated cleft palate in mice after transportation during gestation. Teratology 5:119-124.

Bryant, J.I., I. Emanuel, S.-W. Huang, R. Kronmal and J. Lo
1970 Dermatoglyphics of Chinese children with Down's syndrome. Journal of Medical Genetics 7:338-344.

Burdi, A.R., M. Feingold, K.S. Larsson, I. Leck, E.F. Zimmerman and F.C. Fraser
1972 Etiology and pathogenesis of congenital cleft lip and cleft palate, an NIDR state of the art report. Teratology 6:255-270.

Burdi, A.R. and R.G. Silvey
1969a Sexual differences in closure of human palatal shelves. Cleft Palate Journal 6:1-7.

Burdi, A.R. and R.G. Silvey
1969b The relation of sex-associated facial profile reversal and stages of human palatal closure. Teratology 2:297-303.

Carter, C.O.
1977 Principles of polygenic inheritance. Birth Defects: Original Article Series 13 (3A):69-74.

Cattell, R.B. and J.A. Jaspers
1967 A general plasmode (No. 30-10-5-2) for factor analytic exercises and research. Multivariate Behavioral Research Monographs 67.

Chang, T., D.B. Coursin and M. Hauck
1970 Leukocyte chromosome study of 22 families with cleft lip and/or cleft palate. Cleft Palate Journal 7:402-410.

Ching, G.H.S. and C.S. Chung
1974 A genetic study of cleft lip and palate in Hawaii. I. Interracial crosses. American Journal of Human Genetics 26: 162-176.

Chung, C.S., G.H.S. Ching and N.E. Morton
1974 A genetic study of cleft lip and palate in Hawaii. II. Complex segregation analysis and genetic risks. American Journal of Human Genetics 26:177-188.

Chung, C.S. and N.C. Myrianthopoulos
1968 Racial and prenatal factors in major congenital malformations. American Journal of Human Genetics 20:44-60.

Coccaro, P.J. and S. Pruzansky
1965 Longitudinal study of skeletal and soft tissue profile in children with unilateral cleft lip and cleft palate. Cleft Palate Journal 2:1-12.

Conway, H. and K.J. Wagner
1966 Incidence of clefts in New York City. Cleft Palate Journal 3:284-290.

Cooley, W.W. and P.R. Lohnes
1971 Multivariate Data Analysis. New York: John Wiley and Sons Inc.

Cummins, H.
1939 Dermatoglyphic stigmata in mongoloid imbeciles. Anatomical Record 73:407-414.

Cummins, H. and R.V. Platou
1946 Mongolism: An objective early sign. Southern Medical Journal 39:925.

Cummins, H.W., J. Waits and J.T. McQuitty
1941 The breadths of epidermal ridge on the finger tips and palms: A study of variation. American Journal of Anatomy 68:127-151.

Curtis, E., F.C. Fraser and D. Warburton
1961 Congenital cleft lip and palate: risk figures for counseling. American Journal of Diseases of Children 102: 853-857.

Cushman, C.J. and H.C. Soltan
1969 Dermatoglyphics in Klinefelter's syndrome (47,XXY). Human Heredity 19:641-653.

Dallapiccola, B., B. Bagni and G. Pistocchi
1972 Dermatoglyphic and skeletal hand abnormalities in Turner's syndrome. A tentative scoring method. Acta Geneticae Medicae et Gemellogiae 21:69-78.

Dallapiccola, B. and N. Ricci
1967 Dermatoglyphics in typical and atypical Down's syndrome. Acta Geneticae Medicae et Gemellologiae 16:384-394.

Davidson, J.G., F.C. Fraser and G. Schlager
1969 A maternal effect on the frequency of spontaneous cleft lip in the A/J mouse. Teratology 2:371-376.

Davies, R.H.
1971 Computer Programming in Quantitative Biology. New York: Academic Press.

DeBie, S., M. Hayashi, M.TH. Matton, G. Matton, S. Vrijdagh, M. Lejour, R. Buts and A. Castermans
1977 Dermatoglyphic analysis of primary and secondary cleft palate patients. Cleft Palate Journal 14:222-225.

Deckers, J.F.M., A.M.A. Oorthuys and W.H. Doesburg
1973a Dermatoglyphics in Down's syndrome. I. Evaluation of discriminating ability of pattern areas. Clinical Genetics 4:311-317.

1973b Dermatoglyphics in Down's syndrome. II. Evaluation of scoring methods. Clinical Genetics 4:318-327.

1973c Dermatoglyphics in Down's syndrome. III. Proposal of a simplified scoring method. Clinical Genetics 4:381-387.

DiBennardo, R. and H.L. Bailit
1978 Stress and dental asymmetry in a population of Japanese children. American Journal of Physical Anthropology 48:89-94.

Dixon, W.J.
1976 Biomedical Computer Programs. Los Angeles: University of California Press.

Donahue, R.F.
1965 Birth variables and the incidence of cleft palate: Part 1. Cleft Palate Journal 2:282-290.

Dostal, M. and R. Jelinik
1974 Morphogenesis of cleft palate induced by exogenous factors VI. The question of delayed palatal-process horizontalization. Teratology 10:47-54.

Douglas, B.
1958 The role of environmental factors in the etiology of "so-called" congenital malformations. I. Deductions from the presence of cleft lip and palate in one of identical twins, from embryology and from animal experiments. Plastic and Reconstructive Surgery 22:94-108.

Doyle, W.J. and O. Johnston
1977 On the meaning of increased fluctuating asymmetry: a cross populational study. American Journal of Physical Anthropology 46:127-134.

Drillien, C.M., T.T.S. Ingram and E.M. Wilkinson
1966 The Causes and Natural History of Cleft Lip and Palate. Edinburgh: Livingstone.

Dziuba, P.
1972 Studies on palm and sole dermatoglyphics in patients with clefts of upper lip, alveolar process, and palate. Czas. Stomat. 25:1195-1200.

Ehmann, G.W., G. Pfeifer and K. Gundlach
1976 Morphological findings in unoperated cleft lips and palates. Cleft Palate Journal 13:262-272.

Emanuel, I., S. Huang, L.T. Gutman, F. Yu and C. Lin
1972 The incidence of congenital malformations in a Chinese population: The Taipei collaborative study. Teratology 5:159-169.

Erickson, J.D.
1976 Racial variations in the incidence of congenital malformations. Annals of Human Genetics 39:315-320.

Fang, T.C.
1950 The third interdigital patterns on the palms of the general British population, mongoloid and non-mongoloid mental defectives. Journal of Mental Science 96:780-787.

Fisher, R.A.
1918 The correlation between relatives on the supposition of Mendelian inheritance. Transactions of Royal Society of Edinburgh 52:399-433.

Fitzgerald, M.J.T.
1978 Human Embryology: A Regional Approach. Hagerstown, Maryland. Harper and Row.

Fogh-Andersen, P.
1943 Inheritance of Harelip and Cleft Palate. Kobenhavn: Ejnar Munksgaard.

Forbes, A.P.
1964 Fingerprints and palm prints (dermatoglyphics) and palmar-flexion creases in gonadal dysgenesis, pseudohypoparathyroidism and Klinefelter's Syndrome. New England Journal of Medicine 270:1268-1277.

Foster, T.D. and C.L.B. Lavelle
1971 The size of the dentition in complete cleft lip and palate. Cleft Palate Journal 8:177-184.

Fraser, F.C.
1968 Cleft lip and cleft palate. Cleft Palate Journal 5: 187-194.

1970 The genetics of cleft lip and cleft palate. American Journal of Human Genetics 22:336-352.

Fraser, G.R. and J.S. Calnan
1961 Cleft lip and palate: seasonal incidence, birth weight, birth rank, sex, size, associated malformations, and parental age: a statistical survey. Archives of Disease in Childhood 36:420-423.

Froehlich, J.W.
1976 The quantitative genetics of fingerprints. In: The Measures of Man. E. Giles and J.S. Friedlaender, eds. Cambridge: Peabody Museum Press.

Fuhrmann, W. and F. Vogel
1969 Genetic Counseling. New York: Springer-Verlag.

Fujita, H.
1969 A comparative study on finger patterns of patients with Down's syndrome in Japan. Japanese Journal of Human Genetics 14:198-209.

Gall, J.C., J.R. Hayward, S.M. Garn and M.L. Harper
1973 Studies of dysmorphogenesis in children with oral clefts: II. Variant palmar patterning, imperfect ridge formation and increased palmar creasing as indices of dysmorphogenesis. Cleft Palate Journal 10:203-209.

Gall, J.C. Jr., A.M. Stern, M.M. Cohen, M.S. Adams and R.T. Davidson
1966 Holt-Oram syndrome: clinical and genetic study of a large family. American Journal of Human Genetics 18:187-200.

Galton, F.
1892 Fingerprints. London: Macmillan.

Garn, S.M. and A.R. Burdi
1971 Prenatal ordering and postnatal sequence in dental development. Journal of Dental Research 50:1407-1414.

Garn, S.M., A.R. Burdi and W.J. Babler
1974 Male advancement in prenatal hand development. American Journal of Physical Anthropology 41:353-360.

Garn, S.M., A.B. Lewis and R.S. Kerewsky
1966 The meaning of bilateral asymmetry in the permanent dentition. Angle Orthodontist 36:55-62.

1967 Buccolingual size asymmetry and its developmental meaning. Angle Orthodontist 37:186-193.

Gilmore, S.I. and S.M. Hofman
1966 Clefts in Wisconsin: incidence and related factors. Cleft Palate Journal 3:186-199.

Giovannucci, M.L. and G. Bartolozzi
1968 The hands of subjects and Langdon Down syndrome. Study of 135 subjects hospitalized in the Clinica Pediatrica of Florence. Minerva Pediatrica 20:729-748.

Glanville, E.V.
1965 Heredity and dermal patterns in the interdigital areas of the palm. Acta Geneticae Medicae et Gemellologicae 14: 295-302.

Goor, D., Y. Rotem, A. Friedman and H.N. Newfeld
1965 Ellis-van Creveld syndrome in identical twins. British Heart Journal 27:797-804.

Gorlin, R.J., J. Cervenka and S. Pruzansky
1971 Facial clefting and its syndromes. Birth Defects Original Article Series 7:3-49.

Gorsuch, R.L.
1974 Factor Analysis. Philadelphia: W.B. Saunders Company.

Greene, J.C., J.R. Vermillion and S. Hay
1965 Utilization of birth certificates in epidemiologic studies of cleft lip and palate. Cleft Palate Journal 2:141-156.

Greene, J.C., J.R. Vermillion, S. Hay, S.F. Gibbens and S. Kerschbaum
1964 Epidemiologic study of cleft lip and cleft palate in four states. Journal of American Dental Association 68:387-404.

Gregg, J.M. and J.K. Avery
1971 Experimental studies of vascular development in normal and cleft palate mouse embryos. Cleft Palate Journal 8:101-117.

Guttman, L.
1954 Some necessary conditions for common factor analysis. Psychometrilca 19:149-161.

Hale, A.R.
1949 Breadth of epidermal ridges in the human fetus and its relation to growth of the hand and foot. Anatomical Record 105:763-776.

Hale, A.R.
1952 Morphogenesis of volar skin in the human fetus. American Journal of Anatomy 91:147-173.

Hale, A.R., J.H. Phillips and G.E. Burch
1961 Features of palmar dermatoglyphics in congenital heart disease. Journal of the American Medical Association 176:41.

Harkins, C.S., A. Berlin, R.L. Harding, J.J. Longacre and R. M. Snodgrasse
1962 A classification of cleft lip and cleft palate. Plastic and Reconstructive Surgery 29:31-39.

Harris, C.W.
1967 On factors and factor scores. Psychometrika 32:363-379.

Harris, J. and J. Ross
1956 Cortisone therapy in early pregnancy: relation to cleft palate. Lancet 1:1045-1047.

Hay, S.
1967 Incidence of clefts and parental age. Cleft Palate Journal 4:205-213.

Hays, W.L.
1973 Statistics for the Social Sciences. New York: Holt, Rinehart and Winston, Inc.

Hecht, F., J.S. Bryant, A.G. Motulsky and E.R. Giblett
1963 The No. 17-18 (E) trisomy syndrome. Studies on cytogenetics, dermatoglyphics, paternal age, and linkage. Journal of Pediatrics 63:605-621.

Hilman, B.C.
1953 Dermatoglyphics of anencephalic monsters. Anatomical Record 116:295-300.

Hirsch, W. and G. Geipel
1960 Das Papillarleistensystem der Hand und seine Beziehung zu cerebralen Storungen. Acta Genetica et Statistica Medica 10:103-182.

Holt, S.B.
1951 The correlation between ridge-counts. Annals of Eugenics 16:287-297.

1952 Genetics of dermal ridges: inheritance of total finger ridge-count Annals of Eugenics 17:140-161.

1954 Genetics of the dermal ridges-bilateral asymmetry in finger ridge-counts. Annals of Eugenics 18:211-231.

Holt, S.B.
1955 Genetics of dermal ridges: frequency distributions of total finger ridge-count. Annals of Human Genetics 20:159-170.

1956 Genetics of dermal ridges: parent-child correlations for finger ridge-count. Annals of Human Genetics 20:270-281.

1957a Genetics of dermal ridges: sib pair correlations for total finger ridge-count. Annals of Human Genetics 21:352-362.

1957b Quantitative genetics of dermal ridge-patterns on fingers. Acta Genetica et Statistica Medica 63:473-476.

1958 Genetics of dermal ridges: the relation between total ridge-count and the variability of counts from finger to finger. Annals of Human Genetics 22:323-339.

1959 The correlations between ridge-counts on different fingers estimated from a population sample. Annals of Human Genetics 23:459-460.

1960 Genetics of dermal ridges: familial correlations for $(S/\sqrt{10})$, a measurement of the diversity of ridge-counts from finger to finger. Annals of Human Genetics 24:253-269.

1964 Finger-print patterns in mongolism. Annals of Human Genetics 27:279-282.

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

1968b Palmar ridge-counts. The anthropologist (Special Volume): 117-120.

1969 Dermatoglyphics and sex chromosomes. In: Selected Topics on Genital Anomalies and Related Subjects. M.N. Rashad and W.R.M. Morton eds. Springfield, Ill: Charles C. Thomas pp. 375-395.

1970 The morphogenesis of volar skin. Developmental Medicine and Child Neurology 12:369-371.

1970 Dermatoglyphics in mongolism. Annals of the New York Academy of Sciences 171:602-616.

Holt, S.B. and J. Lindsten
1964 Dermatoglyphic anomalies in Turner's syndrome. Annals of Human Genetics 28:87-100.

Hook, E.B., R.S. Achs and R. Harper
1971 An investigation of dermatoglyphic asymmetry in rubella embryopathy. Teratology 4:405-408.

Howells, W.W.
1973 Cranial Variation in Man. Papers of the Peabody Museum 67.

Hubbell, H.R., D.S. Borgaonkar and D.R. Bolling
1973 Dermatoglyphics studies of the 47,XYY male. Clinical Genetics 4:145-157.

Hunter, H.
1968 Finger and palm prints in chromatin-positive males. Journal of Medical Genetics 5:112-117.

Jaffe, B.F. and G.B. DeBlanc
1970 Cleft palate, cleft lip and cleft uvula in Navajo Indians: incidence and otorhinolarygologic problems. Cleft Palate Journal 7:300-305.

Jantz, R.L.
1977a The genetic significance of the relationship between total finger ridge-count and its variability. Homo 28:1-11.

1977b Sex and race differences in finger ridge-count correlations. American Journal of Physical Anthropology 46:171-176.

1977c Dermatoglyphics and genetic control of canalization. Unpublished Manuscript on file Department of Anthropology, University of Tennessee, Knoxville.

1978 Asymmetry of dermal ridge-counts in relation to fetal growth. Paper given at 47th annual meeting of American Association of Physical Anthropologists. Toronto, Canada.

Jantz, R.L. and V.P. Chopra
1976 A comparison of dermatoglyphic methodologies in population studies. Paper presented at the Vth International Congress of Human Genetics, Mexico City, Mexico.

Jantz, R.L. and D.W. Owsley
1977 Factor analysis of finger ridge-counts in Blacks and Whites. Annals of Human Biology 4:357-366.

Jantz, R.L. and K.R. Parham
1978 Racial differences in dermal ridge breadth. Human Biology 50:33-40.

Jordan, R.E., B.S. Kraus and C.M. Naptune
1966 Dental abnormalities associated with cleft lip and/or palate. Cleft Palate Journal 3:22-25.

Kendall, M.G. and A. Stuart
1966 The Advanced Theory of Statistics. Vol. 3. New York: Hafner Publishing Company.

Kim, Jae-On
1975 Factor analysis. In: Statistical Package for the Social Sciences. N.H. Nie, C.H. Hull, J.G. Jenkins, K. Steinbrenner and D.H. Bent eds. New York: McGraw-Hill Book Company.

Kitamura, H.
1966 Epithelial remnants and pearls in the secondary palate in the human abortus: a contribution to the study of the mechanism of cleft palate formation. Cleft Palate Journal 3:240-257.

Kitamura, H. and B.S. Kraus
1964 Visceral variations and defects associated with cleft lip and palate in human fetuses: a macroscopic description. Cleft Palate Journal 1:99-115.

Knox, G. and F. Braithwaite
1963 Cleft lips and palates in Northumberland and Durham. Archives of Disease in Childhood 38:66-70.

Kramer, C.Y. and D.R. Jensen
1969 Fundamentals of multivariate analysis. Part I. Inference about means. Journal of Quality Technology 1:120-133.

Kraus, B.S.
1970 Basic research in cleft palate--an appraisal and some suggestions. Cleft Palate Journal 7:1-26.

Kraus, B.S. and R.E. Jordan
1965 The Human Dentition Before Birth. Philadelphia: Lea and Febiger.

Kraus, B.S., H. Kitamura and T. Oee
1963 Malformations associated with cleft lip and palate in human embryos and fetuses. American Journal of Obstetrics and Gynecology 86:321-326.

Knussman, R.
1967 Interkorrelationen im Hautleistensystem des Menschen und ihre faktorenanalytische Auswertung. Humangenetik 4:221-243.

1969 Biostatistische Familienuntersuchen zur Hautleistenvariabilität des Menschen. Humagenetik 8:208-216.

Krogman, W.M., M. Mazaheri, R.L. Harding, K. Ishigum, G. Bariana, J. Meier, H. Canter and P. Ross
1975 A longitudinal study of the craniofacial growth pattern in children with clefts as compared to normal, birth to six years. Cleft Palate Journal 12:59-84.

Kshirsagar, A.M.
1972 Multivariate Analysis. New York: Marcel Dekker, Inc.

Leck, I.
1972 The etiology of human malformations: insights from epidemiology. Teratology 5:303-314.

Lejour, M.
1970 Cleft lip induced in the rat. Cleft Palate Journal 7:169-186.

Lindsten, J., M. Fraccaro, D. Ikkos, K. Kaijser, H.P. Klinger and R. Luft
1963 Presumptive iso-chromosomes for the long arm of X in man. Analysis of five families. Annals of Human Genetics 26:383-394.

Loesch, D.
1971 Genetics of dermatoglyphic patterns on palms. Annals of Human Genetics 34:277-293.

Loevy, H.T., E. Krmpotic and I.M. Rosenthal
1975 Cleft lip and cleft palate in D trisomy. Cleft Palate Journal 12:33-43.

Longacre, J.J.
1970 Cleft Palate Deformation. Springfield: Charles C. Thomas.

Lutz, K.R. and F.B. Moor
1955 Study of factors in the occurrence of cleft palate. Journal of Speech and Hearing Disorders 20:271-276.

Mackeprang, M. and S. Hay
1972 Cleft lip and palate mortality study. Cleft Palate Journal 9:51-63.

Mason, L.G., P.R. Ehrlich and T.C. Emmel
1967 The population biology of the butterfly, Euphydryas Editha. V. Character clusters and asymmetry. Evolution 21:85-91.

Mastroiacovo, P., V. Curró, A. Calabro and B. Dallapiccola
1976 Hand dermatoglyphics in trisomy 4 p. Human Genetics 34: 271-276.

Mather, K.
1953 Genetical control of stability in development. Heredity 7:297-336.

Matsuda, Ei
1973 Genetic studies on total finger ridge-count among Japanese. Japanese Journal of Human Genetics 17:293-318.

Matsui, I., Y. Nakagome and M. Higurashi
1966 Dermatoglyphic study of Down's syndrome in Japan. Pediatrics 13:43-48.

Matsunaga, Ei
1972 An estimate of the effective number of loci for total finger ridge-count. Annual Report of National Institute of Genetics (Japan) 22:95-96.

Mavalwala, J.
1962 Correlations between ridge-counts on all digits of the Parsis of India. Annals of Human Genetics 26:137-138.

Mavalwala, J., M.G. Wilson and C.E. Parker
1970 The dermatoglyphics of the 18q-syndrome. American Journal of Physical Anthropology 32:443-449.

Mazaheri, M.
1958 Statistical analysis of patients with congenital lip and/or palate at the Lancaster Cleft Palate Clinic. Plastic and Reconstructive Surgery 21:193-203.

McHenry, H. and E. Giles
1971 Morphological variation and heritability in three Melanesian populations: a multivariate approach. American Journal of Physical Anthropology 35:241-253.

Melnick, M., E.D. Shields, D. Bixler and P. Michael Conneally
1977 Facial Clefting: An alternative biologic explanation for its complex etiology. Birth Defects: Original Article Series 13 (3A):93-112.

Merker, Hans-J.
1977 Considerations on the problem of critical period during the development of the limb skeleton. Birth Defects: Original Article Series 13(1):179-202.

Meskin, L.H.
1966 An Epidemiological Study of Factors Related to the Extent of Facial Clefts. Ph.D. Dissertation, University of Minnesota.

Meskin, L.H. and S. Pruzansky
1969 A malformation profile of facial cleft patients and their siblings. Cleft Palate Journal 6:309-315.

Meskin, L.H., S. Pruzansky and W.H. Gullen
1968 An epidemiologic investigation of factors related to the extent of facial clefts. 1. Sex of patient. Cleft Palate Journal 5:23-29.

Metrakos, J.D., K. Metrakos and H. Baxter
1958 Clefts of the lip and palate in twins. Plastic and Reconstructive Surgery 22:109-122.

Millard, D.R. and K.A. McNeill
1965 The incidence of cleft lip and palate in Jamaica. Cleft Palate Journal 2:384-388.

Miller, J.R.
1968 Dermal ridge patterns: technique for their study in human fetuses. Journal of Pediatrics 73:614-616.

Miller, O.J., W.R. Breg, D. Warburton, D.A. Miller, A. de Capoa, P. Allerdice, J. Davis, H.P. Klinger, E. McGilvray and F.H. Allen
1970 Partial deletion of the short arm of chromosome No. 4 (4 p-). Clinical studies in five unrelated patients. Journal of Pediatrics 77:792-801.

Mittwoch, U.
1969 Do genes determine sex? Nature 221:446-448.

Mittwoch, U., J.D.A. Delhanty and F. Beck
1969 Growth of differentiating tests and ovaries. Nature 224:1323-1325.

Morrison, D.F.
1976 Multivariate Statistical Methods. Second Edition. New York: McGraw Hill.

Morton, N.E., C.S. Chung and M.Mi.
1967 Genetics of interracial crosses in Hawaii. In: Monographs in Human Genetics, 3. L. Beckman and M. Hauge eds. Basel: Karger.

Mukherjee, D.P.
1966 Inheritance of total numbers of triradii or fingers, palms, and soles. Annals Human Genetics 29:349-355.

Mulvihill, J.J. and D.W. Smith
1969 The genesis of dermatoglyphics. Journal of Pediatrics 75:579-589.

Nance, W.E., M. Nakata, T.D. Paul and Yu Pao-lu
1974 The use of twin studies in the analyses of phenotypic traits in man. In: Congenital Defects: New Directions in Research. D. Janerich, R. Skalko and I. Porter eds. New York: Academic Press.

Nanda, R.
1974 Effect of vitamin A on the potentially of rat palatal processes to fuse in vivo and in vitro. Cleft Palate Journal 11:123-133.

Nanda, R. and D. Romeo
1975 Differential cell proliferation of embryonic rat palatal processes as determined by incorporation of tritiated thymidine. Cleft Palate Journal 12:436-443.

Neel, J.V.
1958 A study of major congenital defects in Japanese infants. American Journal of Human Genetics 10:398-445.

Nie, N.H., C.H. Hull, J.G. Jenkins, K. Steinbrenner, and D.H. Bent
1975 Statistical Package for the Social Sciences. New York: McGraw-Hill.

Niswander, J.D. and M.S. Adams
1968 Major malformations in relatives of oral cleft patients. Acta Genetica 18:229-240.

Niswander, J.D., M.V. Barrow and G.J. Bingle
1975 Congenital malformations in the American Indian. Social Biology 22:203-215.

Niswander, J.D. and C.S. Chung
1965 The effects of inbreeding on tooth size in Japanese children. American Journal of Human Genetics 17:390-398.

Niswander, J.D. and W. Wertelecki
1973 Congenital malformation among offspring of epileptic women. Lancet 1:1062.

Ohler, E.A. and H. Cummins
1942 Sexual differences in breadths of epidermal ridges on fingertips and palms. American Journal of Physical Anthropology 29:341-362.

Okajima, M.
1975 Development of dermal ridges in the fetus. Journal of Medical Genetics 12:243-250.

Oliveira, L.T.
1978 Dermatoglyphics of University of Tennessee Students: Effects of Parental Age and Birth Order. Ph.D. Dissertation, University of Tennessee, Knoxville.

Oorthuys, A.M.A. and W.H. Doesburg
1974 Dermatoglyphics in Down's syndrome IV. Evaluation of the use of dermatoglyphic discriminants after the topological classification of constituent characters. Clinical Genetics 5:395-405.

Pannbacker, M.
1968 Congenital malformations and cleft lip and palate. Cleft Palate Journal 5:334-339.

Parker, C.E., J. Mavalwala, R. Koch, A. Hatashita, A. Derencsenyi
1972 The syndrome associated with the partial deletion of the long arms of chromosome 18 (18q-). California Medicine 117: 65-71.

Parsons, P.A.
1964 Finger-print pattern variability. Acta Genetica Et Statistica Medical 14:201-211.

Parsons, P.A.
1973 Maternal age and dermatoglyphic asymmetry in man. Human Heredity 23:226-229.

Pashayan, H., D. Pruzansky and S. Pruzansky
1971 Are anticonvulsants teratogenic? Lancet 2:702-703.

Passarge, E., H.C. Altrogge and R.A. Rüdiger
1970 Human chromosomal deficiency: the 4p-syndrome. Humangenetik 10:51-57.

Pateria, H.N.
1974 Genetic basis of a-b, b-c, and c-d ridge counts on human palms. American Journal of Physical Anthropology 40:171-172.

Penrose, L.S.
1949 The Biology of Mental Defect. First Edition. London: Sidgwick and Jackson.

1954 The distal triradius t on the hands of parents and sibs of mongol imbeciles. Annals of Human Genetics 19:10-38.

1963 Finger-prints, palms and chromosomes. Nature 197:933-938.

1967 Finger-print patterns and the sex chromosomes. Lancet 1:298-300.

1966 Dermatoglyphic patterns in large acrocentric trisomy. Journal of Mental Deficiency Research 10:1-18.

1969a Dermatoglyphics in trisomy 17 or 18. Journal Mental Deficiency Research 13:44-59.

1969b Dermatoglyphics. Scientific American 221:72-84.

1972 Dermatoglyphic patterns in a case of trisomy 8. Lancet 1:957.

Penrose, L.S. and D.S. Loesch
1967 A study of dermal ridge width in the second (palmar) interdigital area with special reference to aneuploid states. Journal of Mental Deficiency Research 11:36-42.

1970 Comparative study of sole patterns in chromosomal abnormalities. Journal of Mental Deficiency Research 14: 129-140.

1971 Dermatoglyphic patterns and clinical diagnosis by discriminant function. Annals of Human Genetics 35:51-60.

Perry, T.B. and F.C. Fraser
1972 Paternal age and congenital cleft lip and cleft palate. Teratology 6:241-246.

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

Philpot, John
Personal communication.

Piatkowska, E. and J. Sokokowski
1972 Palmar dermatoglyphics in cases of cleft primary and secondary palate. International Dermatoglyphics Association News Bulletin. June p. 12.

1973 Dermatoglyphics in primary and secondary cleft palate. American Journal Human Genetics 25:575-576.

Plato, C.C.
1970 Polymorphism of the C line of palmar dermatoglyphics with a new classification of the C line terminations. American Journal of Physical Anthropology 33:413-419.

Plato, C.C., J.J. Cereghimo, and F.S. Steinberg
1973 Palmar dermatoglyphics of Down's syndrome: revisited Pediatric Research 7:111-118.

Plato, C.C., W. Wertelecki, P.S. Gerald and J.D. Niswander
1971 Dermatoglyphics in the 18q-syndrome. Pediatric Reserach 5:64-69.

Pons, J.
1964 Genetics of the a-b ridge count on the human palm. Annals of Human Genetics 27:273-277.

Potrzebowski, P.W.
1974 A Family Study of Dermatoglyphics and Oral-Facial Clefts. Ph.D. Dissertation, University of Pittsburgh, Pittsburgh.

Preus, M. and F.C. Fraser
1972 Dermatoglyphics and syndromes. American Journal of Diseases of Children 124:933-943.

Priest, J.H., C. Verhulst and S. Sirkin
1973 Parental dermatoglyphics in Down's syndrome. A ten-year study. Journal of Medical Genetics 10:328-332.

Pruzansky, S.
1975 Anomalies of face and brain. Birth Defects: Original Article Series 11(7):183-204.

Pruzansky, S., M. Markovic and D. Buzdygan
1970 Twins with clefts. Acta Geneticae Medicae et Gemellologiae 19:224-229.

Ptacek, L.J., J.M. Opitz, D.W. Smith, T. Gerritsen and H.A. Waisman
1963 The Cornelia de Lange syndrome. Journal of Pediatrics 63:1000-1020.

Reed, T.E., D.S. Borgaonkar, P.M. Conneally, P.L. Yu, W.E. Nance and J.C. Christian
1970 Dermatoglyphic nomogram for the diagnosis of Down's syndrome. Journal of Pediatrics 77:1024-1032.

Reed, T., F.R. Sprague, K.W. Kang, W.E. Nance and J.C. Christian
1975 Genetic analysis of dermatoglyphics patterns in twins. Human Heredity 25:263-275.

Richards, I.D.G.
1969 Congenital malformations and environmental influences in pregnancy. British Journal of Preventive Social Medicine 23:218-225.

Rife, D.C.
1968a Finger and palmar dermatoglyphics in Seminole Indians of Florida. American Journal of Physical Anthropology 28:119-126.

1968b Finger and palmar dermatoglyphics in Puerto Ricans. The Anthropologist (Special Volume):133-143.

Roberts, D.F. and E. Coope
1975 Components of variation in a multifactorial character: a dermatoglyphic analysis. Human Biology 47:169-188.

Robinson, G.C., J.R. Miller, E.G. Cook and B. Tischler
1966 Broad thumbs and toes and mental retardation. Unusual dermatoglyphic observations in two individuals. American Journal of Diseases of Children 111:287-290.

Rodewald, A., H. Zankl, H. Wischerath and B. Borkowsky-Fehr
1977 Dermatoglyphic patterns in trisomy 8 syndrome. Clinical Genetics 12:28-38.

Ross, R.B.
1965 Cranial base in children with lip and palate clefts. Cleft Palate Journal 2:157-166.

Ross, R.B. and M.C. Johnston
1972 Cleft Lip and Palate. Baltimore: The Williams and Wilkins Company.

Ross, R.B. and W.K. Lindsay
1965 The cervical vertebrae as a factor in the etiology of cleft palate. Cleft Palate Journal 2:273-281.

Rostron, J.
1977 Multivariage studies on the genetics of dermal ridges. Annals of Human Genetics 41:199-203.

Rothhammer, F., G. Pereira, A. Camousseight and M. Benado
1971 Dermatoglyphics in schizophrenic patients. Human Heredity 21:198-202.

Safra, M.J. and G.P. Oakley
1976 Valium: an oral cleft teratogen. Cleft Palate Journal 13:198-200.

Saksena, P.N., P.C. Bajpai and S.K. Dube
1966 Evaluation of dermatoglyphics in mongolism. Indian Journal of Pediatrics 33:293-297.

Saldaña-Gardia, P.
1973 A dermatoglyphic study of sixty-four XYY males. Annals of Human Genetics 37:107-116.

1975 Dermatoglyphic findings in 54 triple-x females and a review of some general principles applying to the soles in sex chromosome aneuploidy. Journal of Medical Genetics 12:185-192.

Sánchez Cascos, A.
1964 Finger-print patterns in congenital heart disease. British Heart Journal 27:524-527.

Santiago, A.
1969 Classification of cleft lip and palate for machine record coding. Cleft Palate Journal 6:434-435.

Saxen, I. and A. Lahti
1974 Cleft lip and palate in Finland: incidence, secular, seasonal, and geographical variations. Teratology 9:217-224.

Schaumann, B. and M. Alter
1976 Dermatoglyphics in Medical Disorders. New York: Springer-Verlag.

Schaumann, B., J. Cervenka, and R.J. Gorlin
1974 Dermatoglyphics in Trisomy 8 Mosaicism. Humangenetik 24: 201-

Schilli, W., H. Hartmann, F. Härle, R. Kiefer
1969 A general description of 315 cleft lip and palate patients. Cleft Palate Journal 7:573-577.

Seal, H.
1964 Multivariate Statistical Analysis for Biologists, London: Methuen and Company LTD.

Shiono, H., J. Kadowaki and S. Kasahara
1969 Dermatoglyphics of Down's syndrome in Japan. Tahoku Journal of Experimental Medicine 99:107-113.

Siegel, M.I. and W.J. Doyle
1975a The differential effects of prenatal and postnatal audiogenic stress of fluctuating dental asymmetry. Journal of Experimental Zoology 191:211-214.

1975b Stress and fluctuating limb asymmetry in various aspects of rodents. Growth 39:3630369.

Siegel, M.I., W.J. Doyle and C. Kelley
1977 Heat stress, fluctuating asymmetry and prenatal selection in the laboratory rat. American Journal of Physical Anthropology 46:121-126.

Siegel, M.I. and H.H. Smookler
1973 Fluctuating dental asymmetry and audiogenic stress. Growth 37:35-39.

Silver, W.E.
1966 Dermatoglyphics and cleft lip and palate. Cleft Palate Journal 3:368-375.

Singh, S.
1968 A measure of asymmetry of finger ridge counts. Acta Genetica Et Statistica Medical 18:599-605.

1970 Inheritance of asymmetry in finger ridge counts. Human Heredity 20:403-408.

Smiley, G.R.
1972 A possible genesis for cleft palate formation. Plastic and Reconstructive Surgery 50:390-394.

Smiley, G.R., R.J. Vanek and A.D. Dixon
1971 Width of the craniofacial complex during formation of the secondary palate. Cleft Palate Journal 8:371-378.

Smith, D.W.
1970 Recognizable Patterns of Human Malformation. Philadelphia: W.B. Saunders Company.

Smith, G.F.
1966 A study of the dermatoglyphics in the de Lange syndrome. Journal of Mental Deficiency Research 10:241-254.

Sokal, R.R. and F.J. Rohlf
1969 Introduction to Biostatistics. San Francisco: W.H. Freeman and Company.

Soltan, H.C. and K. Clearwater
1965 Dermatoglyphics in translocation Down's syndrome. American Journal of Human Genetics 17:476-479.

Soulé, M.
1967 Phenetics of natural populations, II. Asymmetry and evolution in a lizard. American Naturalist 101:141-160.

Soulé, M. and B. Baker
1968 Phenetics of natural populations. IV. The population asymmetry parameter in the butterfly Coenonympha Tullia. Heredity 23:611-614.

Spence, M.A., R.C. Elston, K.K. Namboodiri and W.S. Pollitzer
1973 Evidence for a possible major gene effect in absolute finger ridge count. Human Heredity 23:414-421.

Spriestersbach, D.C., D.R. Dickson, F.C. Fraser, S.L. Horowitz, B.J. McWilliams, J.L. Paradise, and P. Randall
1973 Clinical research in cleft lip and cleft palate: the state of the art. Cleft Palate Journal 10:113-165.

Spriestersbach, D.C., B.R. Spriestersbach and K.L. Moll
1962 Incidence of clefts of the lip and palate families with children with clefts and families with children without clefts. Plastic and Reconstructive Surgery 29:392-401.

Stevenson, A.C., H.A. Johnston, M.I.P. Stewart and D.R. Golding
1966 Congenital malformations. A report of a study of series of consecutive births in 24 Centres. World Health Organization, Supplement to Vol. 34:1-127.

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

Šubrt, I. J. Červenka, M. Krecek
1966 Cytogenetic study of cleft lip and palate. Cleft Palate Journal 3:362-367.

Tanaka, K., H. Fujino, Y. Fujita, H. Tashiro and Y. Sanui
1969 Cleft lip and palate: some evidences for the multifactorial trait and estimation of heritability based upon Japanese data. Japanese Journal of Human genetics 14:1-9.

Tanaka, K., H. Fujino, H. Tashiro and Y. Sanui
1967 Recurrent risks of cleft lip and palate among relatives with special considerations of sex and racial differences. Japanese Journal of Human Genetics 12:141-149.

Tatsuoka, M.M.
1971 Multivariate Analysis: Techniques for Educational and Psychological Research. New York: John Wiley and Sons.

Thoday, J.M.
1956 Balance, heterozygousity and developmental stability. Cold Springs Harbor Symposium in Quantitative Biology 21:318-326.

1958 Homeostasis in a selection experiment. Heredity 12: 401-415.

Thompson, J.S. and M.W. Thompson
1965 Genetics in Medicine. Philadelphia: W.B. Saunders.

Tolarová, M.
1970 Isolated cleft palate: a genealogic study using codified genealogic questionnaire. Cleft Palate Journal 7:476-483.

Trasler, D.G. and F.C. Fraser
1963 Role of the tongue in producing cleft palate in mice with spontaneous cleft lip. Developmental Biology 6:45-60.

Time-position relationships with particular reference to cleft lip and cleft palate. In: Handbook of Teratology, Vol. 2. J.G. Wilson and F. C. Fraser, eds., New York: Plenum Press, pp. 271-292.

Tuncbilek, E., M. Atasu and B. Say
1972 Dermatoglyphics in trisomy 8. Lancet 2:821.

Uchida, I.A., J.R. Miller and H.C. Soltan
1964 Dermatoglyphics associated with the XXYY chromosome complement. American Journal of Human Genetics 16:284-291.

Uchida, I.A., K. Patau and D.W. Smith
1962 Dermal patterns of 18 and D, trisomics. American Journal of Human Genetics 14:345-352.

Uchida, I.A. and H.C. Soltan
1963 Evaluation of dermatoglyphics in medical genetics. Pediatric Clinics of North America 10:409-422.

Usoev, S.S.
1972 Dermatoglyphics in children suffering from congental defects, with or without chromosomal aberrations. Genetika 8:165-168.

Van de Geer, J.P.
1971 Introduction to Multivariate Analysis for the Social Sciences. San Francisco: W.H. Freeman and Company.

Van der Woulde, A.
1954 Fistula Labii Inferioris Congenita and its association with cleft lip and palate. American Journal of Human Genetics 6:244-256.

Van Valen, L.
1962 A study of fluctuating asymmetry. Evolution 16:125-142.

Veau, V.
1931 Division palantine. Anatomie Chirurgie-Phonoetique. Paris: Masson et Cie.

Veldman, D.J.
1967 Fortran Programming for the Behavioral Sciences. New York: Holt, Rinehart and Winston.

Vij, S. and R. Kanagasuntheram
1971 Cleft palate and associated anomalies in a 30 mm C.R. length human embryo. Cleft Palate Journal 8:409-414.

Vormittag, W. and M. Weninger
1972 XXXY Klinefelter-Syndrome. Humangenetik 15:327-333.

Waddington, C.H.
1957 The Strategy of the Genes. New York: Macmillan.

Walker, B.E. and A. Patterson
1974 Induction of cleft palate in mice by tranquilizers and barbituates. Teratology 10:159-164.

Walker, N.F.
1957 The use of dermal configurations in the diagnosis of mongolism. Journal of Pediatrics 50:19-26.

1958 The use of dermal configurations in the diagnosis of mongolism. Pediatric Clinics of North America 5:531-543.

Walker, N.F., D.H. Carr, F.R. Sergovich, M.L. Barr and H.C. Soltan
1963 Trisomy 21 and 13-15/21 translocation chromosome patterns in related mongol defectives. Journal of Mental Deficiency Research 7:150-163.

Walker, N.F. and H.M. Johnson
1965 Comparative studyes of the dermatoglyphics of Italian patients with Down's syndrome. Proceedings of the International Congress of Scientific Study of Mental Retardation 2:767.

Warburton, D., O.J. Miller
1967 Dermatoglyphic features of patients with a partial short arm deletion of a B-group chromosome. Annals of Human Genetics 31:189-207.

Webb, R.S.
1977 Sexual Differences, Fluctuating Asymmetry and Their Implications in the Growth Rate and Developmental Stability of the Permanent Dentition and the Primary Dermal Ridges of the Fingers and Palms. M.A. Thesis, University of Tennessee, Knoxville.

Weninger, M., G. Aue-Hauser and V. Schieber
1976 Total finger ridge-count and the polygenic hypothesis: a critique. Human Biology 48:713-725.

Wilson, J.G.
1977a Current Status of Teratology. In: Handbook of Teratology, Vol. 1. J.G. Wilson and F.C. Fraser, eds. New York: Plenum Press pp. 47-74.

1977b Embryotoxicity of drugs in man. In: Handbook of Teratology, Vol. 1. J.G. Wilson and F.C. Fraser, eds. New York: Plenum Press pp. 309-355.

Wittwer, B.
1967 Dermatoglyphics in cheilognathopalatoschisis. Lancet 2:617.

Woolf, C.M.
1963 Paternal age effect for cleft lip and palate. American Journal of Human Genetics 15:389-393.

1971 Congenital cleft lip, a genetic study of 496 propositi. Journal of Medical Genetics 8:65-71.

Woolf, C.M. and A.D. Gianas
1976 Congenital cleft lip and fluctuating dermatoglyphic asymmetry. American Journal of Human Genetics 28:400-403.

1977 A study of fluctuating asymmetry in the sibs and parents of cleft lip propositi. American Journal of Human Genetics 29:503-507.

Woolf, C.M., R.M. Woolf and T.R. Broadbent
1963 A genetic study of cleft lip and palate in Utah. American Journal of Human Genetics 15:209-215.

1964 Cleft lip and heredity. Plastic and Reconstructive Surgery 34:11-14.

Wolf, U., H. Reinwein, R. Porsch, R. Schröter and H. Baitsch
1965 Defizienz an den Kurzen Armen eines Chromosoms Nr.4. Humangenetik 1:397-413.

Zajczkowska, K.
1969 Palmar dermatoglyphics in patients with Down's syndrome and in their parents. Polish Medical Journal 8:1477-1482.

Zilberman, Y.
1973 Observations on the dentition and face in clefts of the alveolar process. Cleft Palate Journal 10:230-238.

APPENDIX

Table 34. Clinic and Control Sample Means and Standard Deviations by Sex for 36 Finger and Palmar Ridge-Counts

Variable	Males Mean	Males S.D.	Females Mean	Females S.D.
	Controls			
	n=102		n=102	
L5R	14.27	5.54	12.60	5.25
L5U	1.47	4.15	2.05	4.60
L4R	17.05	6.43	15.10	6.55
L4U	4.97	7.51	4.49	6.94
L3R	11.93	6.59	11.28	5.98
L3U	2.30	5.57	2.56	6.08
L2R	7.37	6.97	7.25	6.53
L2U	6.36	8.16	5.65	7.47
L1R	17.70	5.33	15.46	5.30
L1U	2.80	6.16	4.54	7.13
R5R	13.98	5.35	12.76	5.41
R5U	1.90	4.31	1.40	3.77
R4R	16.73	6.86	15.21	6.59
R4U	6.83	7.74	5.50	7.31
R3R	10.97	6.20	11.76	5.42
R3U	3.13	6.63	2.11	5.74
R2R	7.28	7.11	8.88	6.97
R2U	7.45	9.00	7.07	7.98
R1R	20.07	5.84	18.19	5.97
R1U	6.50	7.94	5.66	7.56
LCD	36.71	7.81	36.78	8.33
LBC	27.74	5.94	27.93	6.05
LAB	41.54	6.67	42.91	5.61
RCD	37.99	7.23	38.05	6.49
RBC	28.01	6.41	28.76	5.74
RAB	41.12	5.76	42.15	6.38
LDRad	2.83	6.04	3.01	6.40
LCUln	2.94	5.30	2.63	4.99
LCRad	2.11	3.59	2.29	3.48
LDAccess	0.52	1.81	0.37	1.37
LAAccess	0.08	0.56	0.05	0.36
RDRad	1.39	4.42	1.88	5.46
RCUln	3.01	5.86	4.40	6.99
RCRad	4.53	5.06	4.08	4.85
RDAccess	0.45	1.45	0.44	2.29
RAAccess	0.33	1.34	0.10	0.61

Table 34 (Continued)

Variable	Males Mean	Males S.D.	Females Mean	Females S.D.
	CL±P Probands			
	n=48		n=40	
L5R	13.21	5.15	11.38	6.12
L5U	0.25	1.73	0.40	1.60
L4R	15.25	6.56	13.30	8.22
L4U	3.06	6.71	4.18	7.15
L3R	9.94	6.19	11.00	7.93
L3U	2.56	6.06	2.75	6.38
L2R	5.85	6.98	6.98	6.96
L2U	6.67	7.20	5.13	8.38
L1R	17.02	6.03	13.18	6.79
L1U	5.73	7.87	4.48	7.57
R5R	12.92	5.95	11.33	6.33
R5U	1.17	2.93	1.08	3.53
R4R	14.88	7.20	14.25	8.17
R4U	5.67	6.89	4.95	6.97
R3R	9.19	6.53	9.68	7.34
R3U	2.58	6.33	2.13	4.82
R2R	6.63	7.18	6.40	6.91
R2U	6.67	8.16	5.40	7.83
R1R	19.17	6.93	15.73	7.61
R1U	5.81	8.00	4.25	7.39
LCD	36.13	7.31	33.95	9.50
LBC	27.25	5.77	27.90	5.14
LAB	41.98	4.81	42.95	5.85
RCD	37.13	5.97	34.70	8.70
RBC	28.27	5.58	27.73	5.80
RAB	41.23	5.30	41.68	5.91
LDRad	1.13	3.47	3.73	5.39
LCUln	2.44	4.21	2.53	4.80
LCRad	2.08	3.17	1.43	2.78
LDAccess	0.23	0.99	0.35	1.55
LAAccess	0.13	0.73	0.10	0.63
RDRad	1.79	5.28	2.18	4.90
RCUln	1.73	4.15	3.40	6.95
RCRad	5.71	5.29	2.73	3.89
RDAccess	0.06	0.43	1.48	3.71
RAAccess	0.52	1.65	0.00	0.00

Table 34 (Continued)

Variable	Males Mean	Males S.D.	Females Mean	Females S.D.
	CL±P Siblings			
	n=21		n=22	
L5R	13.19	5.34	11.46	5.71
L5U	0.81	2.60	0.41	1.92
L4R	15.86	5.67	12.59	7.08
L4U	4.81	6.48	4.96	7.22
L3R	11.95	5.47	9.50	7.39
L3U	2.33	5.44	3.32	6.00
L2R	5.00	5.71	7.96	7.48
L2U	8.24	7.62	3.68	6.44
L1R	11.43	5.03	15.32	6.20
L1U	5.57	7.48	3.73	6.81
R5R	12.86	5.27	11.55	6.33
R5U	0.71	2.39	1.73	3.97
R4R	15.86	6.01	13.50	6.31
R4U	6.05	6.90	4.82	7.40
R3R	11.71	4.69	10.50	6.67
R3U	2.14	5.71	2.96	6.00
R2R	5.00	6.03	6.27	7.35
R2U	7.76	6.11	7.64	8.63
R1R	19.29	5.08	17.14	5.83
R1U	8.10	7.92	3.73	7.33
LCD	35.95	6.01	35.46	9.02
LBC	27.62	5.84	24.59	6.91
LAB	40.24	6.12	40.91	4.12
RCD	38.48	4.16	37.09	6.67
RBC	27.86	6.69	25.91	5.66
RAB	39.81	6.11	39.86	5.25
LDRad	1.19	3.46	2.18	4.32
LCUln	4.24	5.28	2.82	4.43
LCRad	1.91	3.33	1.46	2.43
LDAccess	0.05	0.22	0.55	1.79
LAAccess	0.00	0.00	0.00	0.00
RDRad	0.00	0.00	2.50	5.72
RCUln	4.52	6.49	2.05	3.67
RCRad	3.71	4.24	2.73	4.58
RDAccess	0.43	1.43	1.27	3.17
RAAccess	0.14	0.66	0.23	1.07

Table 34 (Continued)

Variable	Males Mean	Males S.D.	Females Mean	Females S.D.
	CL±P Parents			
	n=45		n=74	
L5R	13.27	6.58	13.16	5.66
L5U	0.53	2.64	0.73	2.69
L4R	15.38	7.30	15.38	7.30
L4U	3.31	6.70	3.20	6.33
L3R	10.60	7.01	10.51	6.87
L3U	3.22	6.63	2.80	5.93
L2R	6.78	6.71	7.28	7.56
L2U	7.40	8.86	7.19	7.82
L1R	16.22	7.08	13.97	6.54
L1U	4.93	7.66	4.60	7.10
R5R	14.13	6.20	13.49	5.19
R5U	1.69	3.85	0.97	2.90
R4R	16.29	7.89	16.80	7.47
R4U	7.16	7.99	5.15	6.94
R3R	10.42	5.99	11.16	6.31
R3U	2.20	6.09	1.78	4.52
R2R	6.71	6.85	7.78	7.01
R2U	8.40	9.02	6.66	8.51
R1R	19.87	6.78	17.16	6.55
R1U	6.91	8.99	5.24	8.07
LCD	36.38	7.37	35.05	7.47
LBC	27.29	4.83	26.04	6.18
LAB	42.07	4.78	40.96	5.50
RCD	38.16	4.89	37.12	6.90
RBC	28.04	5.16	26.31	6.14
RAB	42.78	5.16	39.69	4.62
LDRad	2.98	5.51	2.51	4.64
LCUln	3.58	6.39	2.65	4.15
LCRad	2.27	3.33	2.27	3.99
LDAccess	0.47	1.46	0.18	1.06
LAAccess	0.13	0.66	0.00	0.00
RDRad	2.11	5.09	1.43	4.28
RCUln	3.36	6.38	2.64	5.25
RCRad	4.80	5.21	4.80	5.57
RDAccess	0.18	1.05	0.74	2.40
RAAccess	0.47	1.60	0.10	0.81

Table 34 (Continued)

Variable	Males Mean	Males S.D.	Females Mean	Females S.D.
		CP Probands		
	n=15		n=14	
L5R	12.47	5.74	12.57	6.94
L5U	0.93	2.58	1.71	3.85
L4R	14.00	7.13	14.79	9.99
L4U	3.13	6.50	5.86	8.12
L3R	11.00	7.87	9.79	7.92
L3U	0.00	0.00	2.43	4.85
L2R	8.33	8.02	8.21	6.54
L2U	3.67	6.71	4.00	7.69
L1R	17.27	7.69	11.50	7.56
L1U	5.67	8.90	5.07	7.82
R5R	14.40	6.05	14.36	6.03
R5U	1.67	3.24	3.36	6.37
R4R	15.13	6.33	15.71	9.36
R4U	4.87	7.01	8.50	8.04
R3R	10.40	8.77	10.71	7.03
R3U	2.33	6.68	1.36	3.46
R2R	7.87	6.62	9.07	7.31
R2U	5.07	8.15	8.14	8.90
R1R	18.07	4.82	14.21	6.39
R1U	8.47	8.99	6.71	8.80
LCD	34.40	6.75	34.71	7.02
LBC	25.67	6.42	27.00	5.52
LAB	40.00	5.95	43.29	3.60
RCD	37.33	6.06	34.86	6.92
RBC	25.20	6.25	29.71	5.72
RAB	39.87	6.51	41.64	4.01
LDRad	3.60	4.54	3.71	6.51
LCUln	2.67	3.96	2.36	3.03
LCRad	0.13	0.52	0.86	1.75
LDAccess	0.00	0.00	1.00	2.99
LAAccess	0.00	0.00	0.00	0.00
RDRad	4.93	7.47	2.21	5.32
RCUln	1.00	2.30	2.93	4.57
RCRad	1.93	2.76	4.86	5.61
RDAccess	0.27	1.03	0.29	1.07
RAAccess	0.27	1.03	0.00	0.00

Table 34 (Continued)

Variable	Males Mean	Males S.D.	Females Mean	Females S.D.
	CP Siblings			
	n=10		n=5	
L5R	13.60	4.48	13.00	2.74
L5U	0.00	0.00	2.60	5.81
L4R	14.80	6.03	16.80	4.60
L4U	3.90	5.02	3.80	5.31
L3R	7.30	8.23	7.80	4.87
L3U	1.60	3.24	1.60	3.58
L2R	6.60	7.00	11.80	2.68
L2U	3.90	5.76	3.60	8.05
L1R	17.70	6.04	11.80	4.09
L1U	4.10	7.43	0.00	0.00
R5R	13.90	5.00	14.20	3.03
R5U	2.40	5.06	2.20	4.92
R4R	16.20	6.43	15.60	3.51
R4U	6.80	8.32	5.00	7.28
R3R	10.30	7.51	10.40	6.27
R3U	3.40	7.26	0.00	0.00
R2R	7.00	6.60	11.80	2.95
R2U	4.40	9.32	0.00	0.00
R1R	20.70	5.35	12.40	6.19
R1U	6.10	8.32	2.60	4.34
LCD	35.00	5.93	30.40	5.03
LBC	27.10	5.32	29.00	4.30
LAB	41.00	5.16	41.20	5.45
RCD	36.70	4.99	36.40	3.65
RBC	27.10	6.24	28.40	6.73
RAB	39.60	6.24	41.00	5.24
LDRad	3.20	5.33	7.20	7.26
LCUln	1.30	3.47	0.00	0.00
LCRad	2.00	2.54	0.60	1.34
LDAccess	0.00	0.00	0.00	0.00
LAAccess	0.00	0.00	0.40	0.89
RDRad	4.10	6.74	4.00	8.94
RCUln	1.10	2.33	1.60	3.58
RCRad	4.50	5.36	5.00	5.43
RDAccess	0.00	0.00	0.00	0.00
RAAccess	0.90	2.85	0.00	0.00

Table 34 (Continued)

Variable	Males Mean	Males S.D.	Females Mean	Females S.D.
		CP Parents		
	n=18		n=18	
L5R	13.72	7.04	13.11	5.75
L5U	1.50	4.37	0.78	3.30
L4R	15.28	9.42	16.17	7.25
L4U	7.11	9.29	4.44	8.23
L3R	12.61	7.83	11.67	6.33
L3U	4.39	7.99	0.94	2.88
L2R	7.06	6.24	7.78	6.73
L2U	7.94	9.80	6.00	8.09
L1R	14.17	8.37	15.89	4.92
L1U	6.61	8.64	2.67	5.43
R5R	14.72	6.00	13.11	3.32
R5U	2.72	4.50	0.67	2.83
R4R	16.11	10.04	16.11	5.73
R4U	9.50	9.15	5.50	8.29
R3R	11.39	8.03	12.28	6.18
R3U	5.17	7.70	1.00	4.00
R2R	5.83	6.42	8.61	7.63
R2U	7.17	9.33	5.94	7.83
R1R	14.61	8.38	19.28	4.43
R1U	8.44	8.77	4.67	6.91
LCD	35.28	9.51	36.39	7.24
LBC	26.39	6.73	27.94	5.81
LAB	41.94	5.36	42.61	4.49
RCD	36.22	7.71	38.44	6.28
RBC	28.39	6.86	29.50	6.95
RAB	41.33	6.91	42.33	3.84
LDRad	1.78	5.22	3.33	5.72
LCUln	2.06	4.21	2.61	4.49
LCRad	2.28	3.79	2.17	3.60
LDAccess	0.28	0.96	0.00	0.00
LAAccess	0.22	0.94	0.00	0.00
RDRad	0.00	0.00	1.78	5.19
RCUln	3.56	7.00	3.89	6.69
RCRad	4.89	5.89	4.78	5.29
RDAccess	0.89	2.93	0.00	0.00
RAAccess	0.67	1.65	0.00	0.00

VITA

Douglas William Owsley was born July 21, 1951, in Sheridan, Wyoming. He graduated from Laramie Senior High School in 1969 and entered the University of Wyoming, receiving a Bachelor of Science degree in Zoology in 1973. The following fall, Douglas enrolled at the University of Tennessee, Knoxville, and was awarded the Master of Arts degree in December 1975. He was awarded a Graduate Teaching Assistantship for the University of Tennessee, Department of Anthropology, during the years 1973-1974, 1974-1975, and 1975-1976. Work on his doctorate was continued with the aid of a University of Tennessee Graduate School Fellowship in 1976-1977, and a Hilton A. Smith Graduate Fellowship in 1977-1978. A Ph.D. degree in Anthropology, specializing in physical anthropology, was received in December 1978. As part of this doctoral program, Douglas completed coursework and/or research at Arizona State University, Tempe, the University of Arizona, Tucson, and Meharry Medical College, Nashville.

Douglas has a lecturer appointment with the University of Tennessee Department of Anthropology and a Hilton A. Smith Post-Doctoral Research Fellowship for the year 1978-1979. The research concerns a paleodemographic and osteological analysis of prehistoric and protohistoric American Indian skeletons from the Northern Plains.

Douglas is a member of the Tennessee Anthropological Association, the American Association of Physical Anthropologists, Plains Anthropologists and the International Dermatoglyphics Association.

He is a member of the following honor societies: Omicron Delta Kappa, Phi Beta Kappa, Phi Kappa Phi and Sigma Xi.

He is married to the former Susanne Irene Davies of Lusk, Wyoming and has a daughter, Hilary Lynn.