12-1991

A Study of the Correlation between Alcoholism and Fingerprint Patterns

Miyo Yokota
Western Michigan University

A STUDY OF THE CORRELATION BETWEEN ALCOHOLISM AND FINGERPRINT PATTERNS

by

Miyo Yokota

A Thesis
Submitted to the
Faculty of the Graduate College
in partial fulfillment of the
requirements for the
Degree of Master of Arts
Department of Anthropology

Western Michigan University
Kalamazoo, Michigan
December 1991

A STUDY OF THE CORRELATION BETWEEN ALCOHOLISM AND FINGERPRINT PATTERNS

Miyo Yokota, M.A.

Western Michigan University, 1991

The study of 51 white male alcoholics and 50 white male nonalcoholics between the ages of 15 and 40 was undertaken to learn whether there are differences in the fingerprint patterns between the two groups. Previous research demonstrated that fingerprint patterns are genetically determined and influenced by the intrauterine environment. Fingerprint patterns, ridge counts, pattern intensity index, pattern type symmetry and ridge counts on whorls were studied. Both groups were compared by means of the Z statistics and chi-square tests. It was hoped that the differences observed in the fingerprint patterns of the two groups could be used as a diagnostic criterion to identify potential alcoholics. Unfortunately, no difference between the two groups was observed. Problems associated with this type of study are discussed and suggestions for additional research are presented.

ACKNOWLEDGEMENTS

I wish to express my great appreciation to my advisor and the chairperson of my thesis committee, Dr. Robert Sundick, for not only his critical comments and direction but also his assistance and generosity. I would also like to thank the members of my committee, Dr. Tal Simmons and Dr. John Crowell, for their patience, advice, assistance, and kindness, without which I could not have finished my project.

My special appreciation for help with my research and the supply of materials is expressed to Sgt. Marty Johnson of the Forensic Laboratory, Kalamazoo County Sheriff's Department; the faculty members of the Western Michigan University Department of Anthropology; and volunteers including students, faculty and staff of Western Michigan University.

Thanks to Sue Gibson for the task of final formatting for my thesis.

Finally, my deepest appreciation goes to my parents in Japan, Tadao and Kazuko Yokota, and Todd Crandell for their love, support, and encouragement throughout the course of my study.

Miyo Yokota

Order Number 1346547

A study of the correlation between alcoholism and fingerprint patterns

Yokota, Miyo, M.A.

Western Michigan University, 1991

U·M·I
300 N. Zeeb Rd.
Ann Arbor, MI 48106

TABLE OF CONTENTS

Table of Contents--Continued

LIST OF TABLES

LIST OF FIGURES

CHAPTER I

INTRODUCTION

Physicians, scientists, and laymen have probably been trying to understand the cause of alcoholism ever since alcoholic beverages became available. In the 19th century, Galton (1869/1962) became interested in the factors that determine human behavior and biology. He did not investigate alcoholism per se, but reviewed the issue of nature versus nurture in regard to many social and biological features in human populations. Alcoholism is one of those social or biological problems that have been attributed to nature in the past.

Most investigators today emphasize a genetic (natural) causation, although the data from recent studies are often contradictory. Cloninger, Bohman, and Sigvardsson (1981) examined the familial factor by studying alcoholism in 862 adopted individuals in Sweden. The adoptees and their biological and adoptive parents were categorized into one of four degrees of severity for alcoholism (none, mild, moderate, severe) according to their medical and criminal records. They concluded that (a) children of alcoholic biological parents are more likely to be alcoholic; (b) adopted children of alcoholic biological parents are more likely to drink at an early age than a

parents are more likely to drink at an early age than a nonalcoholic group; and (c) the daughters of alcoholic biological parents have higher anxiety and emotional instability than a nonalcoholic group, while the sons of alcoholic biological parents exhibit a higher frequency of alcohol abuse and criminality.

Studies on alcoholism using twins as subjects have also been undertaken. Kaij (1960) studied 174 groups of twins. The subjects were categorized into five classes based on medical records, criminal convictions, and personal examinations. He found that the concordance rate of alcoholism was 54.2% in monozygotic twins and 31.5% in dizygotic twins. The difference was statistically significant, thus reinforcing the idea of genetic linkage or cause.

Alcoholism has also been studied in relationship to genetic traits such as blood type and color blindness. Swinson (1972) focused on the correlation between blood groups and alcoholism in British populations. Studying 222 alcoholics and 6,510 nonalcoholics, he found that alcoholism was significantly decreased in patients whose blood type was A. In contrast, Nordmo (1959), studying 939 alcoholics and 4,774 nonalcoholics in a Mexican population, found an increased rate of alcoholism in people of blood type A in the population.

Cruz-Coke and Varela (1965) conducted color vision tests on 100 male alcoholic addicts and 633 male control individuals and found that 18 people (18%) in the alcoholic group were color blind, a condition which is associated with a recessive sex-linked gene, while 30 people (4.7%) in the control group had the same disease. This result was statistically significant. Cruz-Coke and Varela (1966) also examined color vision tests in 20 alcoholics and their families, and demonstrated that females whose fathers are alcoholic and color blind are themselves not alcoholic and color blind, but they are likely to pass those characteristics to their sons. Based on these two studies, Cruz-Coke and Varela (1966) suggested that alcoholism is a component of genetic polymorphism of a color blind gene (X linked recessive gene). Fialkow, Thuline, and Fenster (1966) examined color vision in 24 male and 22 female alcoholics. Forty-six percent of the males and 41% of the females were classified as color defective; however, when the researchers retested the same individuals in the same study (1966), half of the color defective individuals identified in the first test turned out to have normal color vision. Because of the latter results, they concluded that an alcoholic's color vision defects were due to a change in the alcoholic individual's metabolism and that alcoholism was not genetically determined.

A third type of alcoholism study involves a search for genetic markers. If genetic markers can be found which predict alcoholism, then individuals predisposed toward alcoholism can be identified. Based on the adoptee studies and twin studies, Begleiter, Porjesz, Bihari, and Kissin (1984) demonstrated through visual tests that there are brain wave differences between boys whose biological fathers are alcoholics and boys whose biological fathers are nonalcoholics. However, Begleiter et al. (1984) were not able to tell from their study whether the brain wave deficit in their subjects would lead to alcoholism without first conducting a longitudinal study of similar subjects. Polich, Burns, and Bloom (1988) also compared brain wave deficit between subjects with a familial history of alcoholism and controls with no history of alcoholism by auditory stimuli tests, and concluded that no significant differences were found between alcoholics and nonalcoholics.

Neurological research is given the most attention by scientists these days. Cloninger's neuro-genetic study (1987) summarized alcoholic influences on the basic stimulus response for three major brain functions consisting of behavioral activation, inhibition, and maintenance. In behavioral activation, alcohol interrupts dopamine cell activity which stimulates novelty seeking behavior or behavioral activation. In behavioral inhibition,

alcohol blocks the expression of behavioral inhibition required by operant conditioning in which a particular behavioral response is learned to prevent punishment or immorality. In the final function of behavioral maintenance, alcohol stops norepinephrine to help relieve individuals from tension.

Blum et al. (1990) examined one of three substances influencing brain stimulus. They studied 70 cadavers consisting of 35 alcoholics and 35 nonalcoholics and examined the D_2 dopamine receptor which affects the capacity of cells to absorb dopamine as one of the brain's chemical transmitters. In 77% of alcoholic cadavers and 28% of nonalcoholic cadavers, the researchers found the D_2 receptor gene mostly on the A-1 allele. Although no one knows how the D_2 receptor gene predisposes someone to alcoholism, they concluded the D_2 receptor gene might predict whether someone will be an alcoholic.

Gordis, Tabakoff, Goldman, and Berg (1990) criticized the Blum et al. (1990) D_2 dopamine study for three reasons: (1) the sample size was small; (2) the determination of whether the individual was an alcoholic or nonalcoholic was done after death, which is not reliable; and (3) although the D_2 dopamine gene receptor was found in 77% of alcoholics, what is the explanation of the other 23% of alcoholics without the D_2 dopamine receptor?

In addition, it is not clear how the samples in the study of Blum et al. (1990) were chosen. Bolos et al. (1990) also studied alcoholism and the D_2 dopamine receptor. They carefully defined their subject and control groups by a double-blind method and examined the D_2 dopamine gene by electrophoresis. Unlike Blum et al. (1990), they did not find any significant difference in the presence of the D_2 dopamine receptor gene between the subject and control groups.

The studies of Cloninger et al. (1981), Kaij (1960), and Swinson (1972) indicate alcoholism is genetically determined. Scientists assume that different genes are somehow involved and work differently amongst different groups of individuals. At the same time, those studies lead scientists to wonder why so many genes are related to alcoholism. It is also not known why one study shows significant differences while another does not.

The Begleiter et al. (1984) study did not show the causation of alcoholism, although it showed the association of alcoholism with brain wave deficit. Therefore, Begleiter believed that alcoholic disorders are environmental events because no specific alcoholism gene exists and no "pure" alcoholics were found (cited in Holden 1991:163). Some investigators believe alcoholism is environmentally determined, as opposed to genetically determined. Goodwin (1976) hypothesized that alcoholism

is sometimes based on culture and tradition. Every society tends to have its drug of choice as an intoxicating substance which many people like to use. In India and North Africa, cannabis has been used in agriculture as an oil, a medicine, an edible fruit, and as the source of a fiber. It also serves as the drug of choice in those regions, while in Judeo-Christian countries, alcohol is the drug of choice for relieving stress. Goodwin (1976) also suggested that some environmental believers theorize that children of alcoholics are more vulnerable to alcoholism than those of nonalcoholics because parental behavior affects the process of their children's development. Children observe and learn from their parent's drinking behavior and anti-social attitudes. However, these environmental factors do not explain what kind of vulnerability to alcohol abuse creates alcoholics and why only a small number of individuals in a society suffer from alcoholism.

Despite the abundance of theories which have been proposed to explain alcoholism, no conclusions have been reached.

I became interested in finding a new method for identifying alcoholics or those prone to alcoholism. With this in mind, I have studied the correlation of fingerprints with alcoholism. Fingerprints were first studied by Galton (1892/1965) in the 19th century.

Later, Cummins and Midlo (1943/1976) studied fingerprints. They demonstrated that dermatoglyphics are unique to each individual and don't change after they are formed in intrauterine life. By the sixth fetal week, volar pads appear and pattern differentiation starts between the third and fourth fetal months. Studies of fingerprint patterns in identical twins reveal that they are primarily genetically determined, but that there is a small intrauterine environmental influence. Holt (1968) studied the inheritance of total finger ridge counts in families and found that monozygotic twins are highly correlated, while dizygotic twin groups, sibling groups and parent-child groups are correlated to a lesser degree.

By the late 1960s, researchers learned that there is a correlation between certain genetically determined diseases and fingerprints. Holt (1968) demonstrated that compared to controls, individuals with Down's syndrome showed an increase of ulnar loops and a decrease of arches, whorls, and radial loops. Holt (1968) also found similar dermatoglyphic characteristics in the patients' families, even though they were phenotypically normal.

Since the 1980s, scientists have examined possible correlations between dermatoglyphics and such diseases as breast cancer, Alzheimer's disease, and alcoholism. Seltzer, Plato, and Fox (1990) demonstrated a strong correlation between breast cancer and the number of whorls.

Six or more whorls were found more commonly in breast cancer patients than in the control group. In the Bierman, Faith, and Stewart (1988) study, breast cancer patients showed a significant difference in the frequency of abnormal fingerprint patterns like "accidentals" and "transitionals," but the researchers did not find a higher frequency of multiple whorls.

Since other investigators have been able to demonstrate a correlation between fingerprint patterns and behavioral disorders or genetic diseases, I hypothesized a correlation between alcoholism and fingerprint patterns. This hypothesis was based on the work of Kojic et al. (1977) who suggested that a positive relationship would be found between fingerprints and alcoholism. They examined the fingerprints of 118 Serbian males undergoing alcoholic treatment in a hospital and another 253 non-alcoholics. Simultaneously, researchers performed various blood tests on both groups examining ABO antigens, Rh phenotypes, Kell, Duffy, P blood group antigen, and others. Blood type A was found in more alcoholic individuals than any other ABO phenotype. This difference was statistically significant. Certain blood group antigens in the Lewis and Duffy system showed a statistical difference between nonalcoholics and alcoholics. Fingerprint patterns exhibited significant differences between the alcoholic groups and nonalcoholic groups. The

alcoholic group had a higher frequency of whorls and arches and a lower frequency of loops with a significant decrease of mean total ridge counts. Kojic et al. (1977) concluded that individuals predisposed to alcohol addiction exhibit different fingerprint patterns than normal people. Furthermore, they suggested that those characteristics would not only be valuable as genetic markers but could also be utilized in the early detection of alcoholism (Kojic et al. 1977).

The de Torok (1972) study also supports my hypothesis. He observed the ratio of the ABO blood groups and chromosome irregularity for alcoholism between a nonalcoholic group and an alcoholic group. His alcoholic group consisted of patients in alcoholic treatment, alcoholism connected organic brain syndrome (OBS) patients, and "dry" alcoholics who were arrested for alcohol related offenses and had not touched alcohol for at least five years. His nonalcoholic group contained both blacks and whites while his alcoholics were all white. Both groups consisted of males and females. He found that alcoholics have a slightly higher frequency of blood type A, although the frequency of each blood type was not significantly different and the frequency of a given blood type depends on different races and ethnic groups. Examining karyotypes from blood taken from alcoholic and nonalcoholic groups, de Torok found that chromosomal aberrations

such as 2n-1 (45) and 2n-2 (44) occur in greater frequency in alcoholics than in nonalcoholics. He also showed that severe chromosomal damage was found more often in OBS patients and treatment patients than in dry alcoholics. He concluded that different occurrences of chromosomal aberrations in alcoholics were due to metabolites, namely heavy intake of alcohol for a number of years. Although de Torok did not show the inheritance of chromosomal abnormalities, I believe that the inheritance of chromosomal aberration will occur if alcoholics are chronic heavy drinkers.

Further support for my hypothesis comes from Cloninger et al. (1981) who demonstrated the genetic inheritance of alcoholism by studying adoptees. As mentioned in the beginning of this chapter, they demonstrated adoptees who had no contact with their alcoholic biological parents had a higher occurrence of alcoholism than adoptees whose biological parents were nonalcoholic.

In conclusion, many scientists have been trying to demonstrate a relationship between fingerprints, genetics, and alcoholism, but they have not been totally successful. Since Cummins and Midlo (1943/1976) demonstrated that fingerprint patterns are formed during fetal life, most people believe dermatoglyphics are genetically determined. The association studies of dermatoglyphics with genetic diseases such as Down's syndrome and

Klinefelter's syndrome strongly supported a genetic theory. Based on Kojic et al. (1977) who showed a strong correlation between alcoholism and fingerprint patterns, de Torok (1972) who demonstrated that alcoholism causes anomalous chromosomes and Cloninger et al. (1981) who emphasized the genetic determination of alcoholism by adoptee samples, I hypothesized a correlation between alcoholism and fingerprints. In this study, I was interested in evaluating how alcoholism expresses itself in fingerprint patterns rather than seeking the cause of alcoholism. In addition, since there are few studies of the correlation between alcoholism and fingerprint patterns, finding a possible relation by examining not only fingerprint patterns, ridge counts and pattern intensity indices, but also ridge counts on whorls was also the purpose of this study. I hoped in this research to be able to find some correlation between alcoholism and fingerprint patterns that can be utilized as a screening or a diagnostic tool for alcoholics and potential alcoholics.

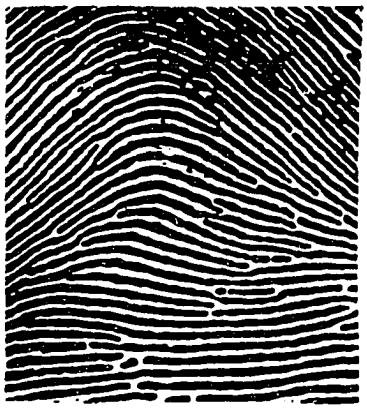
CHAPTER II

TERMINOLOGY

Since Cummins and Midlo (1943/1976) introduced the term "dermatoglyphics" (derma=skin, glyphe=carve) in 1926 as the study of skin ridges, this terminology has been universally accepted. The presence of skin ridges on fingers, palms and soles is a characteristic of primates including humans (Holt 1968). The skin ridges facilitate grasping and locomotor functions.

Many studies have shown fingerprints are unique to each individual. Even identical twins exhibit some differences between them. When fingerprints are taken from an individual, it is only the pattern of ridges on the distal phalanx of each finger that is important. These patterns are always categorized into one of three major types, consisting of arches, loops and whorls.

The arch is the most primitive pattern since it has no triradius. There are two kinds of arches, plain and tented. In the plain arch, the ridges enter from one side of the pattern and flow out to the other side with a rise or wave in the center (Figure 1a). A tented arch is the pattern in which most of the ridges enter from one



a. Plain Arch

b. Tented Arch

c. Loop

d. Plain Whorl

e. Central Pocket Whorl

Figure 1. Fingerprint Pattern Types.

Figure 1--Continued

f. Double Loop Whorl

g. Accidental Whorl

Source: Federal Bureau of Investigation. (1977). The Science of Fingerprints. Washington, DC: U.S. Government Printing Office, pp. 4-6.

side of the pattern and flow out to the other side with tented shape in the center (Figure 1b).

A loop is the pattern in which more than one ridge enters on each side, curves and flows out in the same direction as the entrance (Figure 1c). The loop has only one triradius. There are two kinds of loops. One is called an ulnar loop in which the loop opens on the ulnar side and the delta is located at the ulnar side. The other is called the radial loop in which the loop opens on the radial side with a delta on the radial side. Ulnar loops and radial loops were named after the anatomical position of the ulna and radius bones of the forearm. The final type of the pattern is the whorl, which is a circle shape pattern that has two triradii. The

four types of whorls are plain, central pocket, double loop, and accidental (Figure 1d-g).

A plain whorl has two triradii and at least one ridge forming a complete circle. The best way to distinguish a plain whorl from a central pocket whorl is to see if an imaginary line, drawn between the two triradii, touches and crosses the inner whorl pattern area in a plain whorl. In a central pocket whorl, the same imaginary line would not touch or cross the whorl patterns. A double loop whorl consists of two loops that have two triradii and two separate sets of shoulders. An accidental whorl, the rarest pattern, has more than two different patterns none of which are plain arches. It has more than two triradii.

A triradius is a point where ridges come together in three directions. It is shown in Figure 2. In loops, the location of the triradius on the ulnar or radial side of a finger determines whether the loop is a radial loop or an ulnar loop. Whorls have more than two triradii. The number of triradii is related to the calculation of Pattern Intensity Index which is described later.

The core is the inner point or center of fingerprint patterns (Figure 2). Usually in a loop, the core is shaped like a single straight line called a rod, a series of more than a pair of parallel rods, or just a tiny pinpoint. Arbitrary cores have the following rules.

When there are two rods or the innermost recurve contains no ending ridge, the core has to be the one located furthest from the triradius (Figure 3). When an even number of rods appears on a finger, the core will be the furthest member of the middle pair of rods. In a whorl, the core is the pinpoint of the circle or a hook-shaped ridge. The point of the core along with the triradius serves as the endpoints for containing ridges.

The Total Ridge Count (TRC) is the sum of ridge counts between the core and the triradius for a person's ten fingers. The ridge count does not include the core and the triradius (Figure 3). A straight line or an imaginary line can be drawn between the core and triradius to aid in counting ridges. TRC is an indicator of heredity because uniqueness of an individual's fingerprint patterns comes from TRC with the combination of one of the three patterns--arches, loops, or whorls.

The Pattern Intensity Index (PII) is the total number of triradii of a person's ten digits. The PII shows the difference and complexity of patterns between arches, loops, and whorls. In other words, arches have no triradii whereas loops have one triradii and whorls have two. PII provides a more quantitative value for statistical analysis. A high PII indicates that there are many complex whorl patterns while a low PII indicates a large number of primitive arch patterns.

Figure 2. Core and Triradius.

Source: Federal Bureau of Investigation. (1977). The Science of Fingerprints. Washington, DC: U.S. Government Printing Office, p. 5.

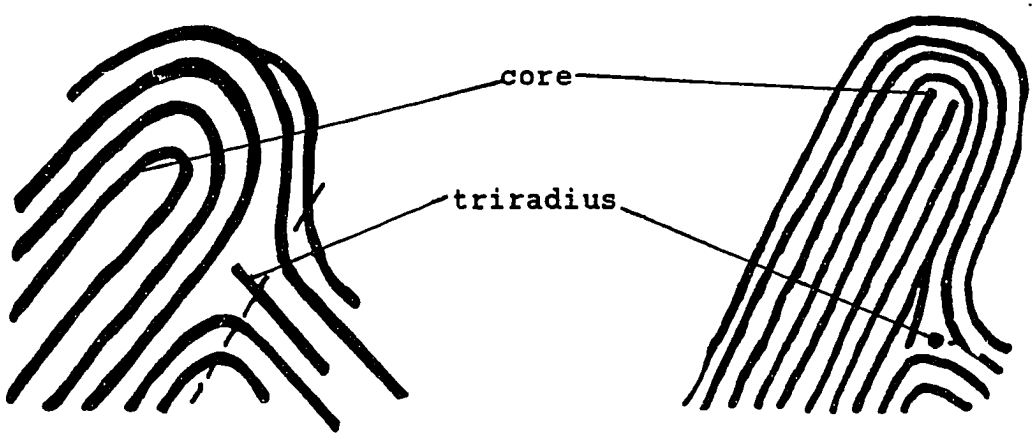


Figure 3. The Determination of Core.

Source: Federal Bureau of Investigation. (1977). The Science of Fingerprints. Washington, DC: U.S. Government Printing Office, pp. 13, 14.

CHAPTER III

MATERIALS AND METHOD

My fingerprint sample was obtained from 101 American white males consisting of 51 alcoholics and 50 nonalcoholics. Data collection was conducted between July and September, 1991, and the data were analyzed in September and October, 1991. The age range in both groups was between 18 and 40 years old. White males were studied because they were the easiest group for which to obtain data. My 51 subjects were culled from the arrest records of the Kalamazoo County Sheriff's Department in Kalamazoo, Michigan. I chose fingerprint records of individuals who had been arrested for alcohol related offenses two or more times. I hoped that my criterion of two or more alcohol related arrests would adequately distinguish between chronic alcoholics and occasional alcohol users.

My control individuals were nonalcoholic volunteers chosen from faculty members and students at Western Michigan University, Kalamazoo. The volunteers were screened in my survey in which I defined nonalcoholics in the following way:

1. They could not drink alcohol more than once per week.

2. They never had more than two drinks at a time.

3. They had never been arrested for an alcohol related offense.

4. They had never been treated for alcoholism.

The consent and survey form are in Appendices B and C, respectively.

The control individuals were also screened for their family background. I eliminated those volunteers whose fathers or mothers had been treated for alcoholism because I hypothesized that alcoholism was genetically determined. On the other hand, one control individual whose nonbiological father was alcoholic was included because this case did not conflict with my hypothesis. In addition, I also included a few controls who did not know if either their biological fathers or mothers drank alcohol or not.

Since the fingerprints of the alcoholic subjects had previously been taken by the Kalamazoo County Sheriff's Department, I did not include individuals with missing or unreadable fingerprints because of bad impressions, scars, broken fingers, etc. Fingerprints in the control group were taken by me with a roll impression on a Comparison and Elimination Fingerprint Record and a black ink pad (Lightning Powder Company, Inc. Salem, OR). The patterns and the ridge counts were analyzed with a magnifying glass and a pointer.

The evaluation of fingerprints was based on the procedures in Cummins and Midlo (1943/1976), The Science of Fingerprints by the FBI (1977), and Holt (1968). Since few investigators have done research on the correlation between alcoholism and fingerprints, I tested many possible comparisons looking for significant differences, keeping in mind the effect of this search on the interpretation of any significant differences found.

The first comparison was of pattern types. Questionable patterns were evaluated by Sgt. Marty Johnson of the Forensic Laboratory, Kalamazoo County Sheriff's Department. The fingerprints of all participants were classified into arch count, ulnar loop count, radial loop count, and whorl count. I compared the distribution for each pattern and the mean count for each pattern between the subject and control groups. The pattern frequencies for each digit on both hands were also observed between the two groups.

The second comparison was of pattern intensity index. After the numbers of triradii for the ten fingers per person were added up, the mean pattern intensity index for the two groups was compared with a Z test.

The third observation was of the ridge counts. Since previous ridge count studies showed correlations with the inheritance of certain diseases and the inheritance in families (Cummins and Midlo 1943/1976; Holt

1968), the ridge counts were important in this study. I used the larger ridge count of the two ridge counts on whorls for the ridge count analysis because most scientists use this methodology (Holt 1968:40-41).

I also followed Holt's directions for drawing an imaginary line in whorl patterns (Figure 4) (Holt 1968: 19-21). In a spiral whorl, two imaginary lines are attached to the core. In a double loop whorl, each imaginary line is drawn between the triradii and the closest core of each loop. When whorls are symmetrical, the right imaginary line from right triradius goes to the top of the core while the left imaginary line goes to the bottom of the core. The two means of Total Ridge Count (TRC) in the subject and control groups were compared with a Z test. The difference in mean ridge counts for each digit was also examined between the subject and control groups.

Pattern type symmetry was also compared. Each pair of the same digit fingers for the right and left hands in an individual was classified separately. I recorded a "match" if the right and left finger patterns were the same and a "no match" if the finger patterns were different. The frequency distributions after the classification were compared with chi-square tests between the two groups.

Symmetrical Shape

Spiral Shape

Double Loop Shape

Figure 4. Imaginary Lines in Whorl Patterns.

Source: Federal Bureau of Investigations. (1977). The Science of Fingerprints. Washington, DC: U.S. Government Printing Office, pp. 5, 49.

The final analysis was on the ridge counts of the whorls. This was done in two ways. First, I counted everybody including those who had no whorl on any finger; and second, I counted only those who had a whorl on at least one finger. Both analyses used a Z statistic to compare the means between the subject and control groups.

CHAPTER IV

RESULTS

The data were analyzed on a VAX 8650 computer at Western Michigan University, Kalamazoo, utilizing the Minitab software program (System 7.2, 1989).

The fingerprints were analyzed beginning with pattern types, then proceeding to Pattern Intensity Index (PII) and ridge counts, pattern type symmetry, and concluding with ridge counts on whorls.

The Patterns

Table 1 reports the classification of all participants by arch count, ulnar loop count, radial loop count, and whorl count. Figure 5 indicates the distribution of pattern types for the subject and control groups. Thirty-four individuals in the subject group exhibited no arches and 33 individuals in the control group had no arches. Table 2 summarizes the mean count for each pattern and its estimated standard error. It also presents the Z values and p values for comparison of the control and subject means.

The comparison of means for each pattern in Table 2 shows no significant difference between the subjects and

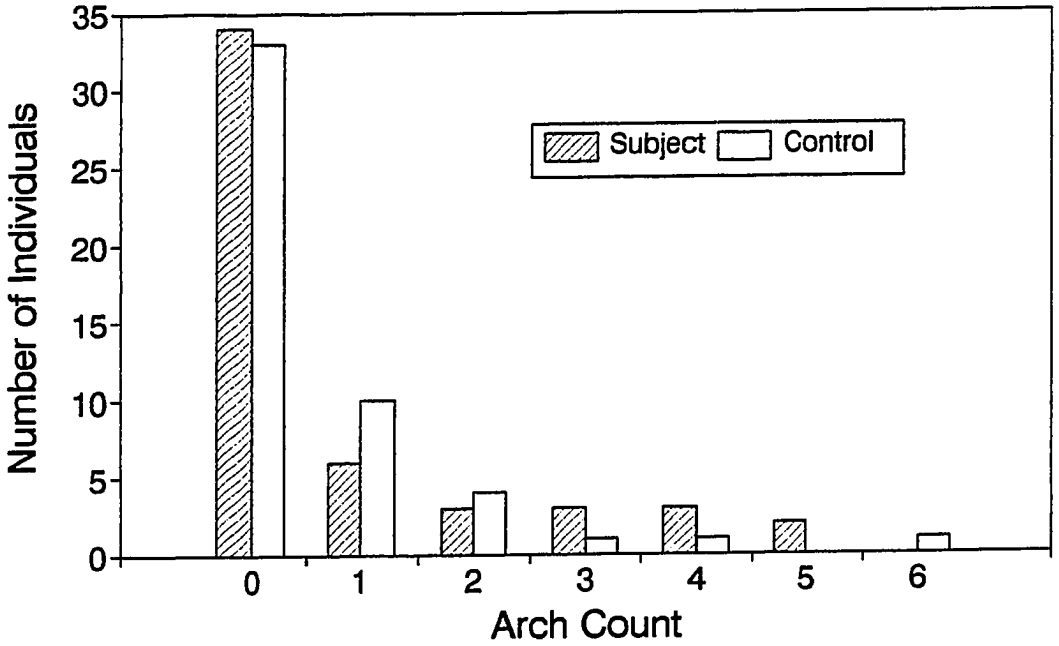
Table 1

The Classification of All Participants by Arch Count, Ulnar Loop Count, Radial Loop Count, and Whorl Count

Group[a]	0	1	2	3	4	5	6	7	8	9	10
					Number of Arches						
Subject	34	6	3	3	3	2	0	0	0	0	0
Control	33	10	4	1	1	1	0	0	0	0	0
					Number of Ulnar Loops						
Subject	1	1	1	3	1	7	13	13	4	3	4
Control	4	1	1	2	3	8	7	8	12	6	0
					Number of Radial Loops						
Subject	38	10	3	0	0	0	0	0	0	0	0
Control	31	15	4	0	0	0	0	0	0	0	0
					Number of Whorls						
Subject	14	4	10	8	6	3	1	3	1	0	1
Control	10	10	10	6	4	3	2	1	1	1	2

[a]Subject N=51; Control N=50.

the controls. For each major pattern type, a chi-square test was used to compare the distribution of counts in the subject and control groups. Since many of the cells in Table 1 have a frequency of 5 or less, certain columns of each subtable had to be combined to carry out the analyses. The columns of 8, 9, and 10 whorl counts were



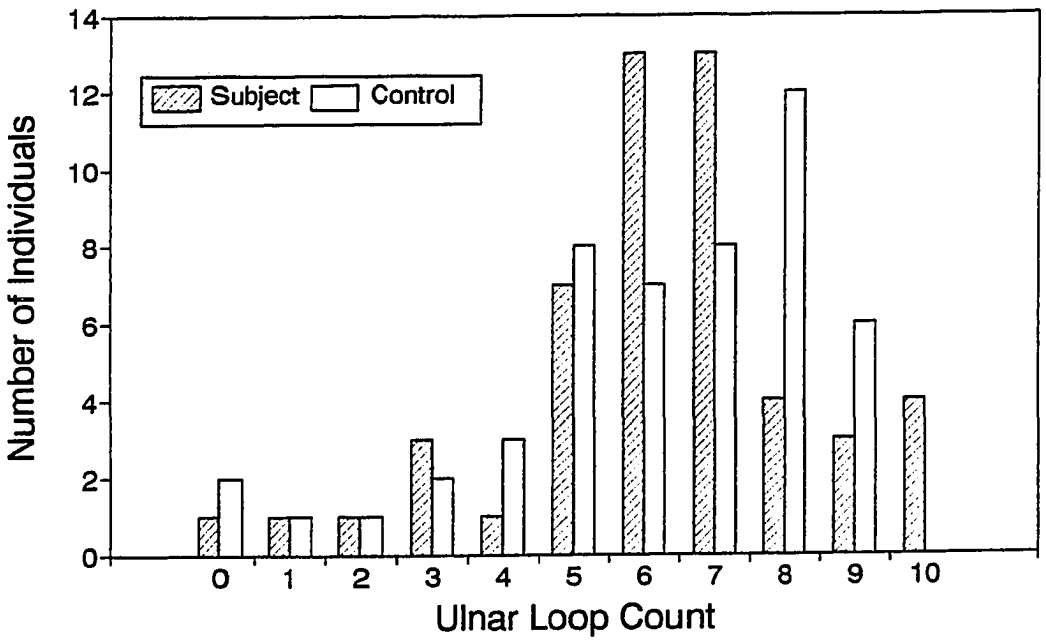
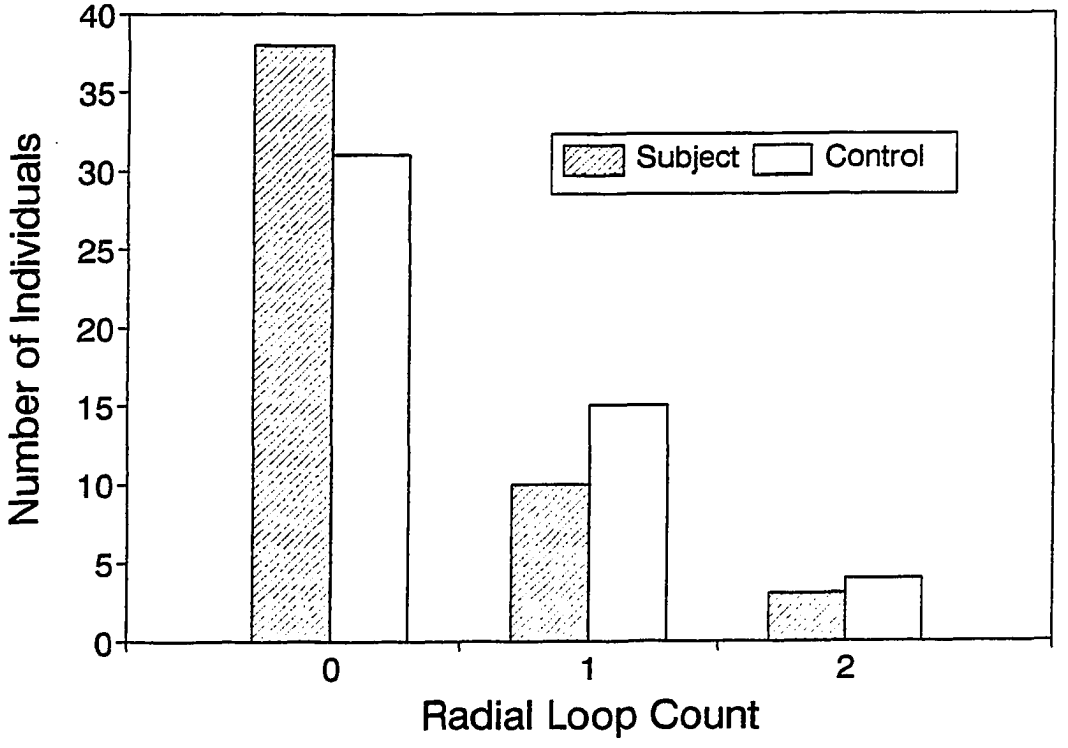


Figure 5. The Distribution of Pattern Types.

Figure 5--Continued



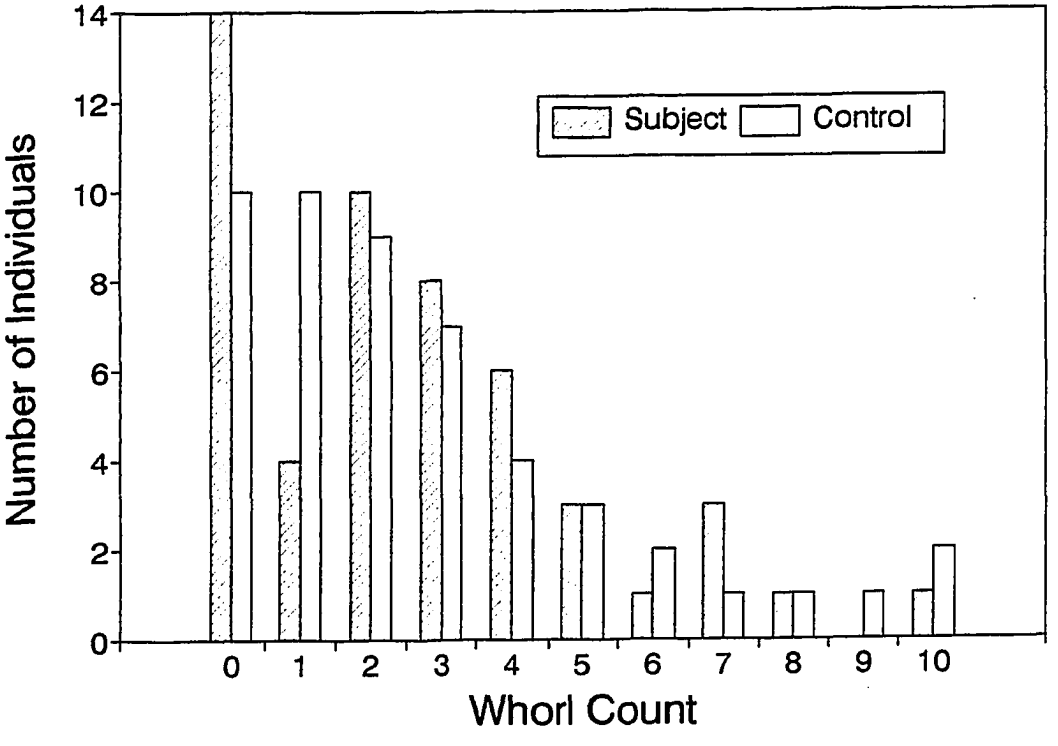


Table 2

Data Summary of the Mean Count for Four Major Patterns

Pattern Types	Sample Groups[a]	Mean	SE Mean[b]	Z Value	p Value
Arch	Subject	0.843	0.205	0.845	0.40
	Control	0.620	0.166		
Ulnar Loop	Subject	6.255	0.301	0.169	0.87
	Control	6.180	0.325		
Radial Loop	Subject	0.3137	0.325	-1.193	0.24
	Control	0.4600	0.0816		
Whorl	Subject	2.588	0.339	-0.264	0.795
	Control	2.720	0.375		

[a] Subject N=51; Control N=50.
[b] SE Mean: Estimated Standard Errors of Mean.

combined into one column. The p values of the chi-square tests for each pattern are: p=0.5724 for arches (χ^2=3.842, df=5); p=0.2872 for ulnar loops (χ^2=7.379, df=6); p=0.40 for radial loops (χ^2=1.843, df=2); and p=0.678 for whorls (χ^2=3.991, df=6). Therefore, the count distributions for the four major pattern types did not show any significant differences between subject and control groups.

Table 3 shows the pattern type frequencies for each finger on both hands. The comparisons of the pattern frequencies for each digit between subjects and controls were conducted with chi-square tests. According to the *p* values in Table 3, only left digit III showed a difference that approached the standard significance level of $p=0.05$ ($p=0.0629$). The other digits did not show significant differences.

In summary, the pattern analyses for the comparisons of the pattern counts showed no significant differences between subject and control groups. In addition, the comparison of pattern frequency distributions between subject and control groups for each finger also revealed no significant differences.

Pattern Intensity Index (PII)

Pattern intensity indices for all ten digits were totalled to arrive at a total PII for each person. The histograms in Figure 6 show the frequencies of the total PII in the subject group and the control group. Five individuals in the subject group had their total PII between 5 and 7; 14 individuals in the subject group had their total PII between 13 and 15.

The mean values of total PII for the two groups were compared with a *Z* statistic. The mean for the subject group was 11.706 with an estimated standard error of

Table 3

Pattern Type Frequencies for Each Digit

Finger	Groups[a]	Arch	Ulnar Loop	Radial Loop	Whorl	p Value
RI	Subject	1	29	0	21	0.9930
	Control	1	29	0	20	
RII	Subject	9	20	8	14	0.9480
	Control	11	18	7	14	
RIII	Subject	5	38	1	7	0.7569
	Control	3	39	0	8	
RIV	Subject	1	28	0	22	0.5434
	Control	1	22	0	27	
RV	Subject	2	44	0	5	0.1461
	Control	0	40	0	5	
LI	Subject	2	33	0	16	0.9593
	Control	2	31	0	17	
LII	Subject	12	16	8	15	0.5787
	Control	7	16	14	13	
LIII	Subject	10	32	1	16	0.0629
	Control	3	42	0	17	
LIV	Subject	0	35	0	16	0.7787
	Control	1	32	0	17	

Table 3--Continued

Finger	Groups[a]	Arch	Ulnar Loop	Radial Loop	Whorl	p Value
LV	Subject	1	45	0	5	0.9995
	Control	1	44	0	5	

[a]Subject N=51; Control N=50.

0.474, while the mean for the control group was 11.840 with an estimated standard error of 0.460. Although the control group had a slightly higher mean, this did not indicate a significant difference between the subject and control groups (p=0.77).

Ridge Counts

The histograms in Figure 7 show the distribution of total ridge counts (TRC) in the subject and control groups. The mean TRC in the subject group was 118.41, whereas that in the control group was 121.72. The estimated standard errors for these means were 6.61 and 6.02, respectively. The two means for the subject and control groups were compared with a Z statistic and the resulting p value was 0.73. Therefore, there was clearly no significant difference between the two groups in mean total ridge count.

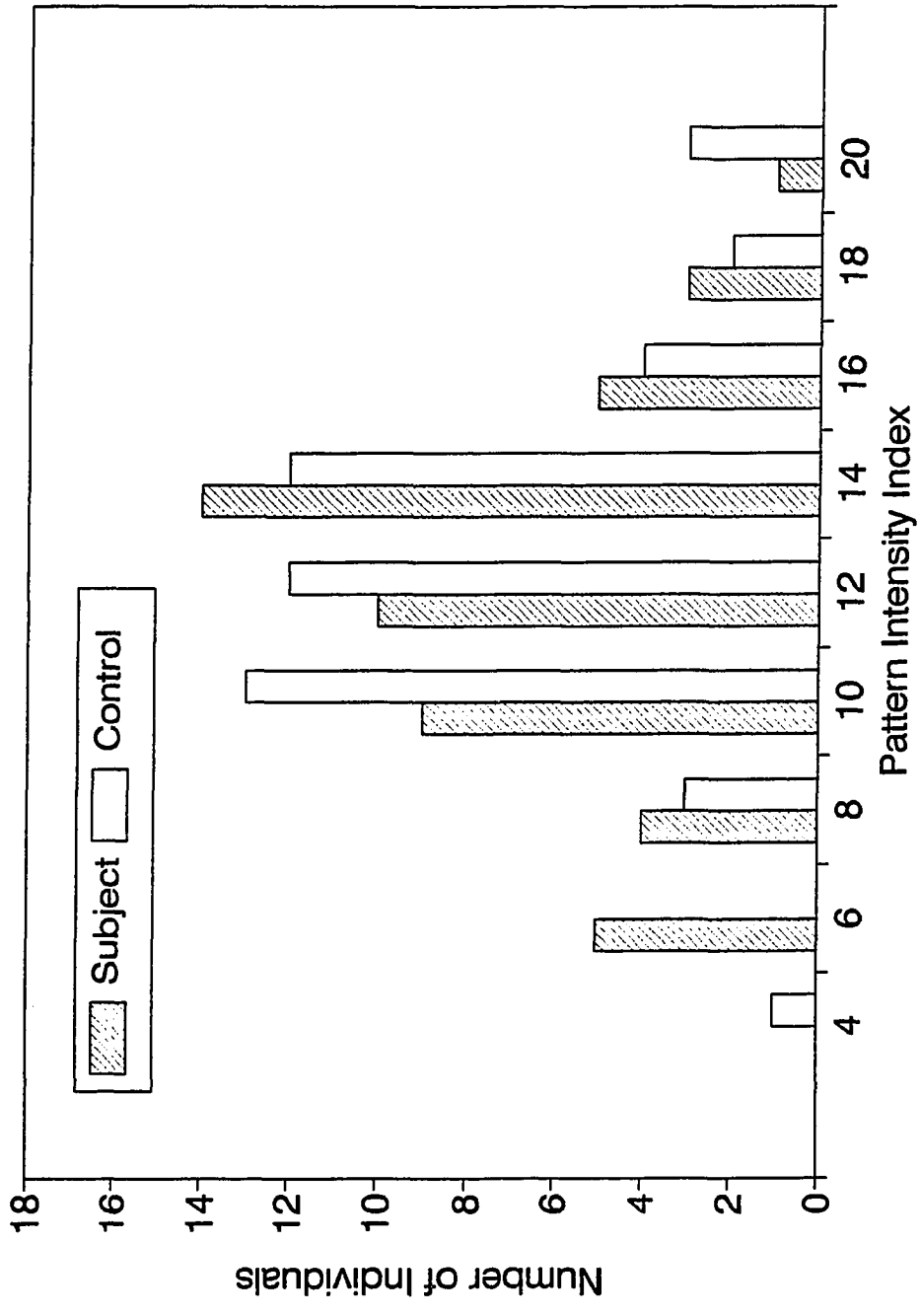


Figure 6. Pattern Intensity Index.

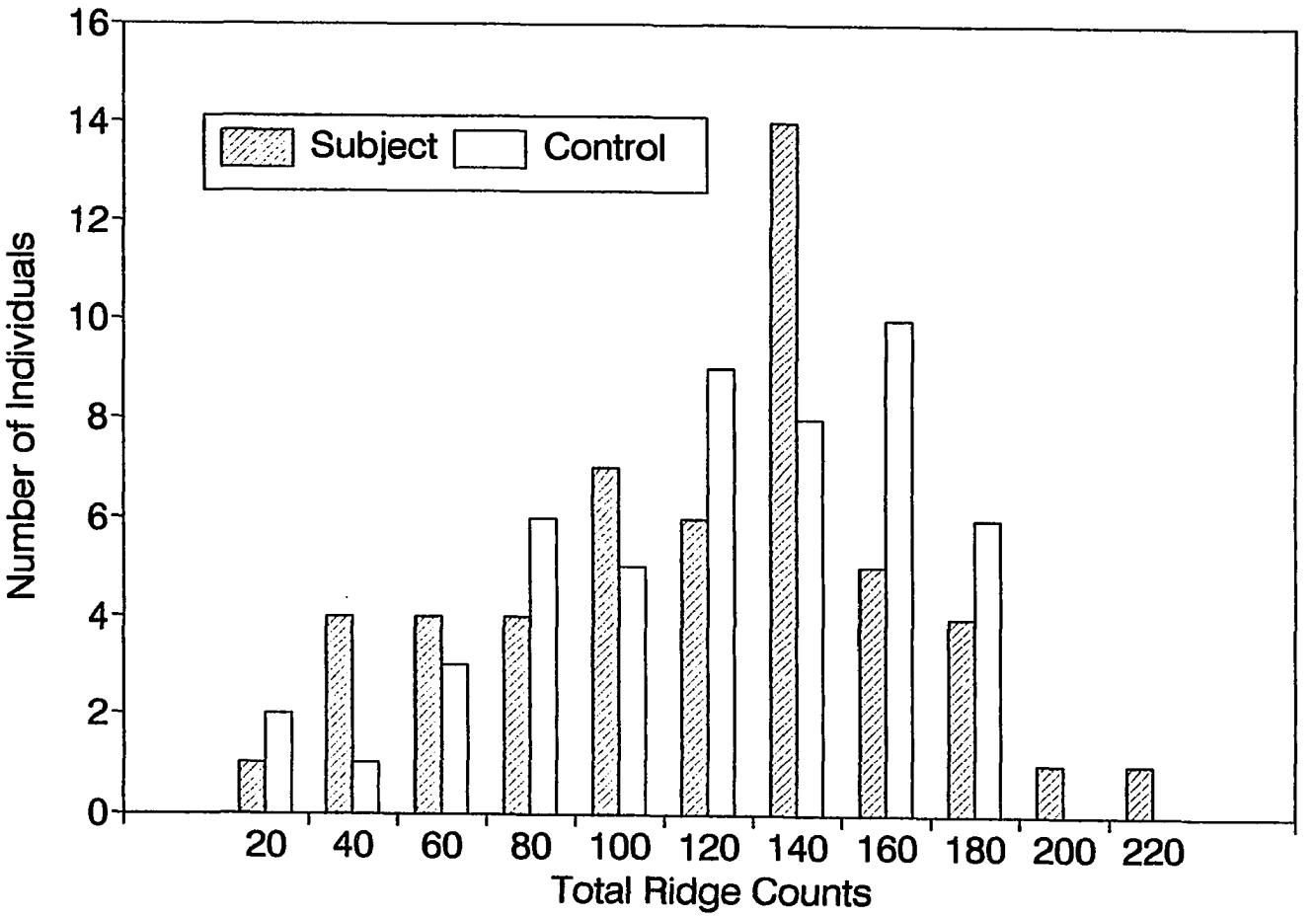


Figure 7. The Distribution of Total Ridge Counts.

For each digit, the difference in mean ridge count between the subject and control groups was examined by computing a Z statistic. Table 4 summarizes the data including the means, standard deviations, estimated standard errors of the means, Z values and p values for the 10 analyses. In each case, the mean TRC is similar for the control and subject groups. As the p values in Table 4 show, no significant differences were found in the comparison of mean TRC for each digit.

Pattern Type Symmetry

To look for possible differences in symmetry between the two groups, each pair of fingers (LI and RI; LII and RII, etc.) was considered separately. Table 5 shows a summary of the data. For any given pair of fingers, an individual was classified according to pattern type if the two fingers were of the same pattern type (a "match"); otherwise, the individual was classified as "no match." For example, 16 people in the subject group had whorls on both the right thumb and left thumb.

The frequency distributions for the two groups were compared with chi-square tests to see if there were differences in the types of symmetry. As mentioned in the pattern comparisons, the columns which contain frequencies of 5 or less were combined before proceeding with the analyses. For instance, in digit I, radial

Table 4

Data Summary of the Mean Ridge Count for Each Finger

Finger	Groups	Mean	SE Mean[a]	SD	Z Value	p Value
RI	Subject	18.824	0.809	5.778	0.63	0.54
	Control	18.100	0.817	5.776		
RII	Subject	9.59	1.02	7.31	0.13	0.91
	Control	9.40	1.03	7.28		
RIII	Subject	9.373	0.848	6.056	-0.734	0.485
	Control	10.200	0.742	5.249		
RIV	Subject	13.137	0.810	5.786	-1.117	0.25
	Control	14.380	0.863	5.394		
RV	Subject	11.196	0.750	5.355	-1.319	0.20
	Control	12.500	0.645	4.564		
LI	Subject	15.294	0.904	6.457	0.846	0.40
	Control	14.220	0.893	6.313		
LII	Subject	8.353	0.930	6.639	-0.235	0.83
	Control	8.660	0.919	6.502		
LIII	Subject	8.824	0.903	6.452	-0.945	0.3421
	Control	9.960	0.794	5.613		
LIV	Subject	12.588	0.890	6.345	-0.418	0.68
	Control	13.060	0.697	4.930		

Table 4--Continued

Finger	Groups	Mean	SE Mean[a]	SD	Z Value	p Value
LV	Subject	11.039	0.679	4.850	-0.239	0.83
	Control	11.260	0.631	4.462		

[a] SE Mean: Estimated Standard Errors of Mean

Table 5

Summary of Pattern Type Symmetry

Finger		Arch	Ulnar Loop	Radial Loop	Whorl	No Match
I	Subject	1	28	0	16	6
	Control	1	22	0	14	13
II	Subject	5	9	3	8	26
	Control	4	9	4	9	24
III	Subject	3	28	1	5	14
	Control	1	34	0	4	11
IV	Subject	0	26	0	15	10
	Control	0	19	0	14	17
V	Subject	0	42	0	4	5
	Control	0	39	0	4	7

The frequency distributions for the two groups were compared with chi-square tests to see if there were differences in the types of symmetry. As mentioned in the pattern comparisons, the columns which contain frequencies of 5 or less were combined before proceeding with the analyses. For instance, in digit I, radial loops were combined with ulnar loops. In addition, arches were combined with loops since the pattern intensity index for arches is closer to that for loops than to that for whorls.

The p values for each digit are: $p=0.1831$ for digit I; $p=0.9838$ for digit II; $p=0.6024$ for digit III; $p=0.2312$ for digit IV; and $p=0.8045$ for digit V.

In examining symmetry, no significant difference was found between controls and subjects.

Ridge Counts on Whorls

In this section of the study, the variable of interest was the total ridge count for those fingers whose pattern type was whorl. Thus an individual with no whorls was assigned a TRC on whorls of 0. Two analyses were done. The first analysis included all participants in the study. Figure 8 shows the distribution of the TRC on whorls in the subject and control groups. The means are 44.06 and 46.64 with estimated standard errors of 6.41 and 6.55 for the subjects and controls,

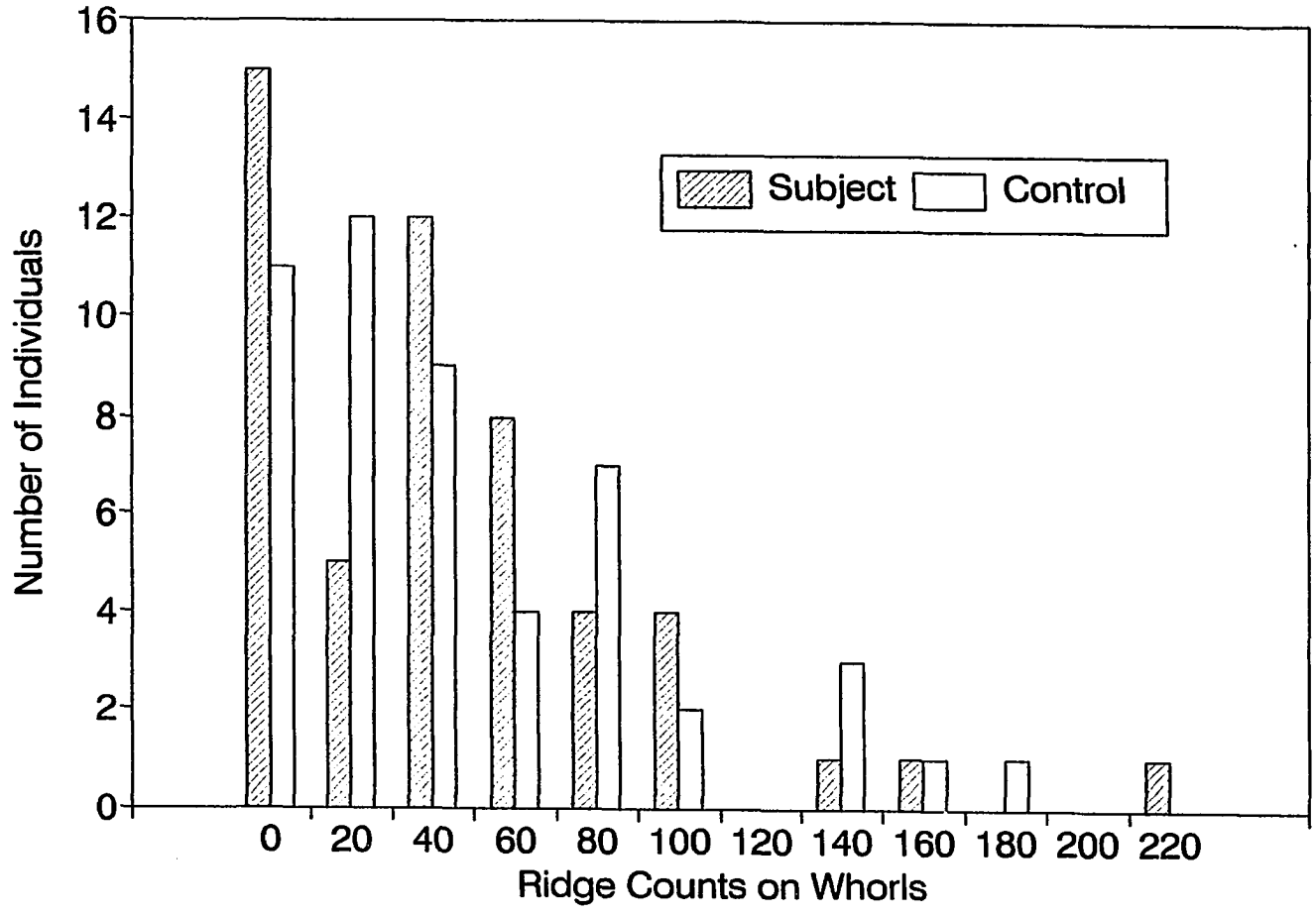


Figure 8. The Distribution of Ridge Counts on Whorls.

respectively. The standard Z test did not show any significant differences in mean TRC on whorls between the two groups ($p=0.79$).

The second analysis included only those participants who had at least one whorl. For subjects, the mean was 62.42, the median was 51.50, and the standard deviation was 42.62; whereas, for controls the mean was 58.30, the median was 47.00, and the standard deviation was 44.68.

The first quartiles in the subject group and the control group were 36.25 and 19.00, respectively, which suggests a difference in the distribution of TRC on whorls between the two groups. However, no significant difference in mean TRC on whorls between the two groups ($p=0.609$) was found.

CHAPTER V

DISCUSSION

This study of fingerprints, pattern intensity index, total ridge counts, pattern type symmetry, and ridge counts on whorls did not demonstrate any correlation between fingerprints and alcoholism. Although only one pattern type frequency analysis for left digit III showed a difference approaching the $p=0.05$ significant level, I believe that this difference may have happened by chance because nothing else except this result showed a significant difference. The interpretation of these results is hard to define because of the complexity that is seen in the inheritance of fingerprint patterns. Meier (1980) summarized the inheritance of dermatoglyphics from three points of view.

Some scientists, such as Slatis, Katznelson, and Bonne-Tamir (1976) and Uchida, Miller, and Soltan (1964), believe in a monogenic theory in which a single allele affects dermatoglyphic patterns. Slatis et al. (1976) examined fingerprint patterns from 571 individuals from the "Habbanite" community in Israel. The Habbanites, who used to live in Yemen, immigrated into Israel and formed an isolated community in the 1950s, because there was no

interbreeding with the local Moslems or the rest of the Jewish community. Because family size in the Habbanites was large, pedigrees were traceable to grandparents of the oldest living family members. Under these conditions, using the Mendelian theory, Slatis et al. (1976) examined the frequency of each pattern type (arch, ulnar loop, radial loop, and whorl) on each finger and pattern sequences on both hands and compared those of each individual with those of his/her family members. They concluded that the basic fingerprint pattern sequence is all ulnar loops and that a variety of genes cause deviation from this pattern sequence. A dominant gene for radial loops is seen on the index finger and a dominant gene for arches is seen on thumbs. However, since genes code for various information, one problem with the monogenetic theory is proving what single gene affects dermatoglyphics. Although some investigators (Rignell 1987; Uchida et al. 1964) demonstrated that an abnormal chromosome affects certain dermatoglyphic features, the monogenic theorists have not been able to attribute the normal inheritance of dermatoglyphics to any specific gene.

Other scientists state that the formation of dermatoglyphics is related to the intrauterine environment. Babler (1978) found that humans spontaneously aborted within 11 weeks have a higher frequency of arches than in elective abortions. Yet, Babler's results may come from

severe chromosomal aberrations in the fetus that led to the spontaneous abortion, because Holt (1968) showed that patients with Trisomy 17 or 18 are likely to have a greater number of arches. Jones, Smith, Ulleland, and Streissguth (1973) also demonstrated that babies of chronic alcoholic mothers are likely to have unusual dermatoglyphics. Because Holt (1968) shows that reciprocal translocations also happen between a father and his children, the inheritance and formation of dermatoglyphics are not always influenced by mothers.

A third explanation for dermatoglyphic patterns is a polygenic theory. In the polygenic theory, some scientists believe dermatoglyphic patterns are influenced by various genes. Due to problems with the monogenic theory and some controversial evidence in regard to the environmental theory, most scientists believe in the polygenic theory. Holt (1968) examined the correlation coefficient of the total ridge count for familial pairs such as parent-parent, parent-child, sibling, monozygotic twin, and dizygotic twin pairs. She showed that the correlation coefficient for monozygotic twin pairs was much higher ($\underline{r}$=0.95) than any other familial pair. She concluded that total ridge count was controlled by multiple genes because the hereditary composition of monozygotic twins was much more similar than that of any other pair. However, polygenic theorists have not proven what kinds

of specific genetic information affect dermatoglyphics, either.

In addition, as patients with Trisomy 17 or 18 are likely to have a greater number of arches (Holt 1968), the occurrence of arches is higher in alcoholics than in controls (Kojic et al. 1977). The question is whether the higher number of arches in these two different studies are related to each other or not. In another study, white breast cancer patients in the United States show a higher incidence of whorls than people with no risk for cancer (Seltzer et al. 1990), but this does not mean that populations with higher frequencies of whorls, such as Japanese, Tibetans and Eskimos (Jantz 1974), have a higher risk of cancer.

Thus, association studies of dermatoglyphics are related to theoretical problems both at the individual level by certain genetic features and at the population level by features such as race, ethnicity and sex. American samples, such as that studied in my research are especially difficult to use for proving associations between alcoholics and dermatoglyphics due to the large number of immigrants and hybrids in the population. I believe Americans are more heterogenic than homogeneous populations such as Japanese, Australian aborigines, and American Indians.

In summary, this study was affected by the following problems. First, the sample size was small (subjects N=51, controls N=50). Second, the samples in this research came from two different environments. The subjects were obtained from the Kalamazoo County Sheriff's Department, while the controls were students, faculty, or staff from Western Michigan University. The population that the two groups came from may have been very different from one another in spite of the fact that a white male population was chosen in each case. The ethnic origin of the two groups could have differed significantly, which could have led to the lack of any significant results in my study.

Furthermore, because the subject group consisted of individuals arrested by the Kalamazoo County Sheriff's Department a while ago, there was no way that information on the parents could be obtained. In the control group, I had to rely on individual honesty to learn through my survey whether they were alcoholic or not and whether or not their parents were alcoholic. This reliance on the subject's description of his own alcoholism or nonalcoholism and that of his parents could have created inaccuracies in my data.

A major problem hindering research on alcoholism and dermatoglyphics is the definition of alcoholism. Psychological, sociological, and physiological dependence on

alcohol leads scientists to develop and utilize various definitions of alcoholism in their research (Kaij 1972; National Council on Alcoholism, Criteria Committee 1972). Some scientists define alcoholism sociologically by observing the criminal behavior of alcoholics. Other researchers define alcoholism from a physiological point of view by examining such alcohol-related diseases as cirrhosis of the liver and mental deterioration, while a third group of researchers might define alcoholism by psychological aspects which involve a dependency on alcohol for the relief of emotional problems (e.g., anger, depression, fatigue, etc.). The various definitions of alcoholism make it difficult to determine the criteria to be utilized in a study such as my research.

Statistical methodology can also be a problem in fingerprint studies. While my study did not show any significant difference, Kojic et al. (1977) showed a statistically significant difference between alcoholics and nonalcoholics in their dermatoglyphic study. Their chi-square statistics indicated highly significant differences in proportions of arches, loops, and whorls between control and subject groups. However, a major problem was that Kojic et al. (1977) did not fully explain the data they presented. Because of this, it is impossible to confirm their statistical methodology and results. In addition, when they were looking for physio-

logical differences by blood tests between nonalcoholics and alcoholics, their experimental unit was the individual person. However, in examining dermatoglyphics, they considered each finger independently and ignored any statistical dependence between each individual and their fingers. For example, they stated that 26.1% of the total number of fingers in the nonalcoholic group (N=258) and 30.9% of the total number in the alcoholic group (N=118) had whorls (Kojic et al. 1977). Their chi-square analysis for whorls showed 8.98. When I recalculated their data with a chi-square analysis, my chi-square was 9.448. This discrepancy may be due to the fact that there were not 10 fingerprints for each participant in their study. If the fingers for one individual do not represent 10 independent pieces of information, their methodology is inappropriate. Since the purpose of a fingerprint study is to determine the relationship between fingerprints and an individual person regarding a disease such as alcoholism, breast cancer, etc., the experimental unit has to be a person, not a finger. For this reason, I believe the statistically significant differences in Kojic et al. (1977) are suspect. My research methodology was based on Seltzer et al. (1990) who carefully treated people as the experimental units in their study, where they looked at the number of whorls for each individual.

A related statistical problem for fingerprint studies is whether, during fetal life, each finger has an independent probability of exhibiting a certain pattern or whether the patterns on each of the 10 fingers are related to one another. As Cummins and Midlo (1943/1976) show, there is a high frequency of the same fingerprint patterns for homologous pairs of digits, but this high frequency is very difficult to predict if all fingers have independent probabilities. In another study, Seltzer et al. (1990) demonstrated that, in comparison with controls, breast cancer patients have a higher frequency of whorls on digits II, III, and IV. Again, this raises the question of whether or not the pattern types of adjacent fingers are determined independently of each other.

Regarding the question of experimental unit and the probability of a certain pattern occurring for each finger, most scientists, including Kojic et al. (1977), Plato, Cereghino, and Steinberg (1975), and Holt (1968:27), seem to treat all fingers independently. They analyzed pattern frequency for the total number of each pattern type by total number of fingers. Instead, as Chapter IV showed, I observed the total number of each pattern type per person. Thus, considering these issues is important in the choice of statistical methodologies, because the results can be significantly affected.

The last problem is the interpretation of fingerprints. Various people judge transitional patterns differently. In the case of a transitional pattern between an arch and a loop, some call it an arch, while others call it a loop. In addition, ridge counts may vary from 1 to 5 because some people include thin lines or small dots in the ridge counts. Therefore, those relative observations which affect frequencies of patterns, ridge counts and pattern intensity index produce different results.

In summary, my research did not show any significant difference between alcoholics and nonalcoholics. However, this does not mean that alcoholism is not genetically determined. If alcoholism is caused solely by external environmental factors, alcohol intake in adulthood would not affect fingerprint patterns. However, if alcoholism is considered to be entirely environmentally determined, then some evidence for this should be presented.

Based on what I have learned, I would suggest that additional research be conducted and that the following variables be better controlled: (a) Subjects should be chosen from a discrete homogeneous population; (b) the subjects should come from similar environmental backgrounds; (c) a larger sample size than mine should be studied; (d) the concept of alcoholism should be clearly

defined; and (e) there should be a careful selection of the statistical methods. Further studies should also examine palm prints to see if they exhibit any difference between alcoholic and nonalcoholic populations.

CHAPTER VI

CONCLUSIONS

The analysis of fingerprint patterns conducted in this study indicated that no significant differences exist between alcoholics and nonalcoholics for any of the variables investigated. The interpretation of these results must proceed with caution, however, as numerous other studies have shown that many factors may confound the analysis. Included among those factors would be (a) the statistical methodology, (b) the definition of alcoholism, (c) subject reliability and honesty in reporting whether they are alcoholic or not, and (d) the interpretation of the fingerprint patterns themselves. This study also was not designed to determine whether any variation which might be found in the fingerprint patterns of individuals or their offspring is caused by environmental (intrauterine) or genetic factors.

While this research does not provide any conclusive results the literature and this study suggest that further work should be done to investigate the relationship of fingerprint patterns to alcoholism. In order to determine whether there is any intrauterine environmental effect of alcoholism on fingerprint patterns, a sample of

children whose mothers would admit to having consumed alcohol during pregnancy should be fingerprinted. Those fingerprints should be compared to the prints of children whose mothers claim that they did not consume alcohol during the period of pregnancy. Because United States public health policy recommends that pregnant women not consume alcohol, a planned study in which pregnant women are advised to consume alcohol for the purpose of the study could not be done. Instead the investigator would have to conduct a retrospective study in which women who have delivered their children would be questioned as to whether or not they had consumed any alcohol during their pregnancy. In a study of this nature there is an excellent chance that the woman being questioned might not tell the truth about whether she had consumed alcohol. It would also be difficult to ascertain the quantity, the type of alcohol and the trimester of pregnancy in which the alcohol was consumed. In spite of these problems the study should be done to determine the intrauterine environmental effect on the development of fingerprint patterns. Another problem that could be investigated is to see whether the children of parents (male and/or female) who are chronic alcoholics show different fingerprint patterns than children whose parents are not alcoholics.

If these studies demonstrate that alcoholism is in some way genetically determined, and furthermore that

there is a relationship between fingerprint patterns and alcoholism, then the analysis of fingerprint patterns in young individuals could be utilized to ascertain the probability that an individual would become an alcoholic in adult life. Advance knowledge of this possible genetic predisposition for alcoholism might be useful in the treatment of those individuals.

Appendix A

Letter of Approval From the Human Subjects Institutional Review Board (HSIRB)

Human Subjects Institutional Review Board

Kalamazoo, Michigan

WESTERN MICHIGAN UNIVERSITY

Date: July 18, 1991

To: Miyo Yokota

From: Mary Anne Bunda, Chair

Re: HSIRB Project Number 91-06-19

This letter will serve as confirmation that your research protocol, "The Correlation Between Alcoholism and Fingerprint Patterns" has been approved after full review by the HSIRB. The conditions and duration of this approval are specified in the Policies of Western Michigan University. You may now begin to implement the research as described in the approval application.

You must seek reapproval for any change in this design. You must also seek reapproval if the project extends beyond the termination date.

The Board wishes you success in the pursuit of your research goals.

xc: R. Sundick, Anthropology

Approval Termination: July 18, 1992

Appendix B

Consent Form

Subject # _____

The Consent Form

I am a graduate student in anthropology studying the correlation between alcoholism and fingerprint patterns for my masters thesis. The cause of alcoholism is not totally understood, but it is believed by some investigators to have a genetic basis and by others a cultural basis or a combination of the two. I am attempting in my research on fingerprint patterns to determine what role genetics plays in alcoholism.

Previous research has demonstrated a correlation between fingerprint patterns and particular genetically determined diseases such as Down's syndrome, Klinefelter's syndrome, etc. I propose to test the hypothesis that there is a correlation between alcoholism and fingerprint patterns.

I have already collected fingerprints from a sample of white males in the age range of 18-40, known to suffer from alcoholism. I now need to obtain fingerprints from a control population of comparable nonalcoholic and would like your permission to include you in this study.

I want to emphasize that the data I collect on this survey and your fingerprints will be for my use on this project only. Your survey form and fingerprints will be secured n a locked cabinet on the WMU campus. I will not record your name or any other identifying characteristic to protect your confidentiality. You will be assigned a subject number as a means of identification.

Please, remember, you have no obligation to participate in this project, if you don't wish to do so.

Since it is imperative that my control group contains nonalcoholic individuals only, my definition of alcoholism follows:

1. You regularly drink alcohol more than once a week.
2. When you drink alcohol, you have more than two drinks.
3. You have been arrested for drinking problems.
4. You have been treated for alcoholism.
5. You consider yourself an "alcoholic."
6. You have taken this survey before.

If you fit into one or more of the above categories, please don't participate in this survey and study, as it will have a detrimental effect on my research project.

Under the conditions mentioned above, if you still agree to participate in my research and give me permission to fingerprint you, please sign and date this release below.

I agree to participate in this research and complete the survey form.

_____ _____

Signature Date

(Please tear off)

If you decide in the next few days that you should not have participated in the study because you have second thoughts about meeting my definition for a control subject, please call me at 387-3970 and tell me to remove your survey and fingerprints from the study. Your identifying number is _____.

Thank you for your consideration.

Miyo Yokota

Appendix C

Survey Form

Survey on Alcohol Abuse

1. In what country were you born? 1. _____

2. In what country were you raised? 2. _____

3. What year were you born in? 3. _____

The following questions (#4-13) refer to your biological parents only.

4. In what country was your biological mother born? 4. _____

5. Do you know whether your biological mother drinks alcohol? 5. ____ Yes
____ No...Please move to question #9

6. Does your mother drink alcohol at least once a week? 6. ____ Yes
____ No

7. When your mother drinks alcohol, does she have more than one drink? 7. ____ Yes
____ No

8. Has your mother been treated for alcoholism? 8. ____ Yes
____ No

9. In what country was your biological father born? 9. _____

10. Do you know whether your biological father drinks alcohol? 10. ____ Yes
____ No...Please move to question #14

11. Does your father drink alcohol at least once a week? 11. ____ Yes ____ No

12. When your father drinks alcohol, does he have more than one drink? 12. ____ Yes ____ No

13. Has your father been treated for alcoholism? 13. ____ Yes ____ No

The following questions (#14-23) refer to the mother and father your grew up with, if they are not your biological parents (nonbiological parents are stepparents, foster parents, aunts, uncles, etc.).

14. In what country was your nonbiological mother born? 14. _____

15. Do you know whether your nonbiological mother drinks alcohol? 15. ____ Yes ____ No...Please move to question #14

16. Does your nonbiological mother drink alcohol at least once a week? 16. ____ Yes ____ No

17. When your nonbiological mother drinks alcohol, does she have more than one drink? 17. ____ Yes ____ No

18. Has your nonbiological mother been treated for alcoholism? 18. ____ Yes ____ No

19.	In what country was your nonbiological father born?	19. _____
20.	Do you know whether your nonbiological father drinks alcohol?	20. ____ Yes ____ No...Please move to question #24
21.	Does your nonbiological father drink alcohol at least once a week?	21. ____ Yes ____ No
22.	When your nonbiological father drinks alcohol, does he have more than one drink?	22. ____ Yes ____ No
23.	Has your nonbiological father been treated for alcoholism?	23. ____ Yes ____ No
24.	Do you have any biological siblings?	24. ____ Yes ____ No
25.	If you answered yes in question #24, please check if she/he (or they) are "alcoholic" or not by my definition. (A-E refer to individual siblings.)	25. A.___Yes/ ___No B.___Yes/ ___No C.___Yes/ ___No D.___Yes/ ___No E.___Yes/ ___No
26.	Do you have any nonbiological siblings?	26. ____ Yes ____ No
27.	If you answered yes in question #26, please check if she/he (or they) are "alcoholic" or not by my definition.	27. A.___Yes/ ___No B.___Yes/ ___No C.___Yes/ ___No D.___Yes/ ___No E.___Yes/ ___No

Thank you for your cooperation.

Appendix D

Data for Pattern Types, Total Ridge Counts, Pattern Intensity Index in Subject and Control Groups

	Arch		Loop		Whorl					
Sub-ject#	PA	TA	UL	RL	CPW	PW	DLW	AC	TRC	PI
1			3			7			150	17
2			6		1	3			134	14
3			10						147	10
4		1	5			2	2		91	13
5	5		5						35	5
6		1	6	1		2			116	11
7			6	1		3			165	13
8			7			3			158	13
9	4		6						40	6
10			7			3			149	13
11			6			2	2		144	14
12			9			1			146	11
13		1	7			2			127	9
14			10						59	10
15		1	7	1		1			102	10
16			6	2		1	1		110	12
17			7		2	1			124	13
18			3	2		4	1		143	15
19			7		3				166	13
20	1		6	1		2			103	11
21						10			219	20
22			7		3				99	13
23			5			4	1		169	15
24	1	2	7						58	7
25			8			2			175	12
26			7			3			184	13

	Arch		Loop		Whorl					
Sub-ject#	PA	TA	UL	RL	CPW	PW	DLW	AC	TRC	PI
27			3			6	1		199	17
28			7			3			131	13
29			9	1					84	10
30			6	2		2			95	12
31	2		8						40	8
32			10						70	10
33			2			7	1		189	18
34			6		2	2			133	14
35			7	1	1	1			127	12
36			10						138	10
37			9		1				136	11
38	3	1	6						45	6
39	1	2	5				2		71	9
40	2		5	1		2			114	10
41			6		3	1			147	14
42	4		6						59	6
43			4		2	3	1		149	16
44			8	1		1			97	11
45			7	1		1	1		149	12
46		1	1	1	2	5			99	16
47			5		1	4			177	15
48		3	7						52	7
49		2	8						73	8
50		5	5						17	5
51			6			3	1		135	14

	Arch		Loop		Whorl					
Con-trol#	PA	TA	UL	RL	CPW	PW	DLW	AC	TRC	PI
1			9	1					66	10
2		1	5		3		1		106	13
3			2		1	7			172	18
4			9	1					141	10
5		1	8	1					70	9
6			7	2			1		133	11
7			6	1		2	1		163	13
8			5		1	4			170	15
9		1	9						112	9
10	1		8	1					37	9
11					1	9			181	20
12			9		1				134	11
13		1	8	1					61	9
14			8	2					95	10
15			7		1		2		139	13
16		1	5	2		2			68	11
17			1			8	1		166	19
18			8		2				118	12
19	1		4		2		3		111	14
20			7			1	2		122	13
21			9	1					85	10
22			5		1	2	1		151	13
23	5	1	4						12	4
24		2	8						88	8
25			7		2	1			161	13
26			8			2			81	12

	Arch		Loop			Whorl				
Con-trol#	PA	TA	UL	RL	CPW	PW	DLW	AC	TRC	PI
27			3		3	3	1		169	17
28			4			6			107	16
29		1	7	1		1			108	10
30	1	3	5				1		26	7
31						8	2		165	20
32			6			2	2		167	14
33		2	6	1		1			118	9
34		2	6			2			110	10
35			8			2			114	12
36			9			1			135	11
37			7			3			180	13
38			6			4			179	14
39			3	1		6			163	16
40			5			4	1		147	15
41		1	8		1				118	10
42			7	1			2		125	12
43		2	6	1		1			71	9
44		1	6	1	2				90	11
45		3	5	1	1				87	8
46			8	1	1				144	11
47			5	2		2	1		173	13
48			8			1	1		158	12
49			7			2	1		165	13
50			8			1	1		132	12

Appendix E

Ridge Counts on Each Digit in Subject and Control Groups

Subject #	R1	R2	R3	R4	R5	L1	L2	L3	L4	L5	TRC Total
1	15	17	13	17	16	16	13	14	17	12	150
2	11	7	16	20	11	5	10	20	20	14	134
3	17	17	10	16	15	16	14	10	18	14	147
4	16	2	3	14	9	15	0	2	18	12	91
5	7	0	0	0	13	3	0	0	2	10	35
6	20	17	5	15	10	19	0	10	10	10	116
7	23	18	8	20	11	24	16	9	20	16	165
8	22	15	16	19	11	22	13	14	17	9	158
9	17	0	1	3	6	10	0	0	3	0	40
10	18	13	18	16	11	13	14	17	17	12	149
11	18	16	17	13	10	16	20	11	10	13	144
12	21	15	14	17	15	17	12	13	13	9	146
13	25	11	16	19	11	0	5	17	11	12	127
14	17	3	6	3	5	10	2	3	3	7	59
15	22	0	10	8	8	18	8	3	16	9	102
16	24	9	14	5	6	19	9	17	3	4	110
17	21	8	11	16	6	15	22	6	13	6	124
18	29	20	1	11	17	20	12	2	12	19	143
19	23	21	14	19	14	17	17	16	16	9	166
20	18	14	8	14	6	15	3	0	18	7	103
21	30	21	20	21	22	28	16	21	24	16	219
22	16	1	11	14	10	15	2	9	10	11	99
23	16	18	18	20	15	17	13	17	20	15	169
24	6	2	0	15	0	4	0	6	16	9	58
25	29	19	19	12	15	25	17	8	13	18	175
26	26	18	14	17	17	28	15	18	16	15	184

Subject #	R1	R2	R3	R4	R5	L1	L2	L3	L4	L5	TRC Total
27	24	19	20	18	19	22	20	16	21	20	199
28	18	15	8	11	12	19	10	13	14	11	131
29	21	2	6	11	9	17	4	3	3	8	84
30	19	11	3	10	6	19	6	5	9	7	95
31	14	0	0	2	5	6	5	1	4	3	40
32	13	1	7	7	9	8	1	11	9	4	70
33	26	16	15	21	17	23	17	16	19	19	189
34	24	12	1	17	13	17	12	12	12	13	133
35	17	7	12	14	10	17	13	12	13	12	127
36	23	14	12	17	12	16	10	12	11	11	138
37	20	7	13	16	14	17	5	12	18	14	136
38	18	0	0	2	6	9	0	0	1	9	45
39	25	0	2	5	8	22	0	0	6	3	71
40	18	1	8	17	18	20	0	0	19	13	114
41	18	7	4	21	16	9	13	16	19	14	137
42	13	0	0	5	18	9	0	0	1	13	59
43	18	17	7	16	21	14	9	9	18	20	149
44	16	9	13	13	3	13	3	7	10	10	97
45	23	9	12	16	19	13	11	13	18	15	149
46	12	0	12	14	6	16	8	6	18	7	99
47	24	19	15	17	17	24	14	11	18	18	177
48	17	0	6	6	3	14	0	0	3	3	52
49	15	3	6	9	5	16	0	0	4	15	73
50	0	5	2	5	0	0	0	0	3	2	17
51	17	13	11	16	15	13	12	12	15	11	135

Control	R1	R2	R3	R4	R5	L1	L2	L3	L4	L5	TRC Total
1	12	3	6	6	5	7	2	7	11	7	66
2	22	12	5	18	19	15	0	6	8	11	116
3	21	18	17	17	18	17	18	19	14	13	172
4	18	13	11	17	13	14	12	10	17	16	141
5	12	0	3	9	6	13	2	4	14	7	70
6	18	5	14	14	15	2	0	12	10	12	102
7	21	17	11	21	20	18	8	13	19	15	163
8	24	17	14	14	16	22	13	17	16	17	170
9	23	5	9	18	12	18	11	11	13	13	133
10	10	2	3	5	6	0	3	1	3	4	37
11	22	17	15	24	13	22	18	17	16	17	181
12	13	16	12	12	17	8	13	12	14	17	134
13	9	0	5	7	5	9	4	8	9	5	61
14	11	6	11	7	14	13	1	9	13	10	95
15	20	15	13	11	11	23	21	12	2	11	139
16	13	5	2	11	4	11	4	0	14	4	68
17	19	20	17	14	14	18	16	18	16	14	166
18	14	2	13	17	10	10	12	13	16	11	118
19	21	0	10	18	15	17	7	2	2	19	111
20	18	15	12	14	11	18	7	3	12	12	122
21	14	5	7	15	6	7	2	4	12	13	85
22	23	15	14	18	13	19	5	14	16	14	151
23	0	0	0	0	5	0	3	2	2	0	12
24	16	0	3	13	12	14	0	5	10	15	88
25	23	14	17	13	18	10	17	16	22	11	161
26	26	1	5	1	8	20	3	7	6	4	81

Control #	R1	R2	R3	R4	R5	L1	L2	L3	L4	L5	TRC Total
27	31	16	13	15	14	20	21	13	16	10	169
28	14	7	7	11	12	13	10	10	13	10	107
29	14	0	13	17	14	5	2	15	19	9	108
30	7	0	0	4	4	3	0	0	4	4	26
31	17	15	16	18	13	17	16	18	18	17	165
32	19	15	15	22	18	19	15	11	16	17	167
33	17	7	0	17	13	18	0	8	17	13	110
34	18	0	9	17	17	11	0	10	13	15	110
35	14	8	9	13	14	11	10	10	14	11	114
36	18	15	15	14	12	19	12	10	12	8	135
37	26	18	14	18	19	22	18	17	13	15	180
38	24	12	21	19	17	19	14	21	18	14	179
39	19	22	11	17	13	18	21	12	18	12	163
40	17	22	12	18	17	13	11	9	14	14	147
41	22	11	9	15	11	16	10	0	13	11	118
42	23	5	1	19	14	25	9	8	8	13	125
43	11	0	6	15	5	7	8	0	13	6	71
44	20	0	10	7	7	15	2	10	13	6	90
45	21	0	7	13	7	13	0	7	16	3	87
46	19	20	14	22	18	5	8	9	14	15	144
47	30	10	17	19	16	24	7	18	21	11	173
48	20	12	12	21	17	15	11	12	21	17	158
49	18	17	17	20	16	16	15	18	15	13	165
50	23	15	13	14	11	22	11	10	7	7	133

Appendix F

Ridge Counts on Whorls in Subject and Control Groups

Subject #	Total RC on Whorls	No. of Whorls	Subject #	Total RC on Whorls	No. of Whorls
1	105	7	27	140	7
2	76	4	28	37	3
3	0	0	29	0	0
4	63	4	30	38	2
5	0	0	31	0	0
6	39	2	32	0	0
7	59	3	33	158	8
8	49	3	34	54	4
9	0	0	35	27	2
10	49	3	36	0	0
11	64	4	37	16	1
12	15	1	38	0	0
13	0	0	39	47	2
14	0	0	40	38	2
15	22	1	41	60	4
16	43	2	42	0	0
17	51	3	43	92	6
18	105	5	44	10	1
19	52	3	45	36	2
20	32	2	46	79	7
21	219	10	47	96	5
22	34	3	48	0	0
23	85	5	49	0	0
24	0	0	50	0	0
25	31	2	51	54	4
26	72	3			

Control #	Total RC on Whorls	No. of Whorls	Control #	Total RC on Whorls	No. of Whorls
1	0	0	27	132	7
2	71	4	28	73	6
3	142	8	29	17	1
4	0	0	30	7	1
5	0	0	31	165	10
6	18	1	32	68	4
7	60	3	33	17	1
8	76	5	34	30	2
9	0	0	35	27	2
10	0	0	36	18	1
11	181	10	37	66	3
12	16	1	38	96	4
13	0	0	39	96	6
14	0	0	40	73	5
15	67	3	41	13	1
16	25	2	42	48	2
17	149	9	43	11	1
18	33	2	44	17	2
19	78	5	45	13	1
20	37	3	46	22	1
21	0	0	47	73	3
22	80	4	48	41	2
23	0	0	49	48	3
24	0	0	50	38	2
25	44	3	51	54	4
26	46	2			

BIBLIOGRAPHY

Babler, W.
1978 Prenatal Selection and Dermatoglyphic Patterns. American Journal of Physical Anthropology 48(1):21-27.

Begleiter, H., B. Porjesz, B. Bihari, and B. Kissin
1984 Event-Related Brain Potentials in Boys at Risk for Alcoholism. Science 225:1493-1496.

Bierman, H., M. Faith, and M. Stewart
1988 Digital Dermatoglyphics in Mammary Cancer. Cancer Investigation 6(1):15-27.

Blum, K., E. Noble, P. Sheridan, A. Montgomery, T. Ritchie, P. Jagadeeswaran, H. Nogami, A. Briggs, and J. Cohn
1990 Allelic Association of Human Dopamine D_2 Receptor Gene in Alcoholism. The Journal of the American Medical Association 263:2055-2060.

Bolos, A., M. Dean, S. Lucas-Derse, M. Ramsburg, G. Brown, and D. Goldman
1990 Population and Pedigree Studies Reveal a Lack of Association between the Dopamine D_2 Receptor Gene and Alcoholism. The Journal of the American Medical Association 264:3156-3160.

Cloninger, C., M. Bohman, and S. Sigvardsson
1981 Inheritance of Alcohol Abuse. Archives of General Psychiatry 38:861-868.

Cloninger, R.
1987 Neurogenetic Adaptive Mechanisms in Alcoholism. Science 236:410-416.

Cruz-Coke, R., and A. Varela.
1965 Colour-Blindness and Alcohol Addiction. The Lancet 7426(2):1348.

Cruz-Coke, R., and A. Varela.
1966 Inheritance of Alcoholism. The Lancet 7476: 1282-1284.

Cummins, H., and Midlo, C.
1976 Fingerprints, Palms and Soles. South Berlin, MA: Research Publishing Co., Inc. The original was published in 1943.

de Torok, D.
1972 Chromosomal Irregularities in Alcoholics. Annals of the New York Academy of Sciences 197: 90-100.

Federal Bureau of Investigation
1977 The Science of Fingerprints. Washington, D.C.: U.S. Government Printing Office.

Fialkow, P., H. Thuline, and L. Fenster.
1966 Lack of Association Between Cirrhosis and the Common Types of Color Blindness. The New England Journal of Medicine 275:584-587.

Galton, F.
1962 Hereditary Genius. Cleveland, OH: The World Publishing Company. The original was published in 1869.

Galton, F.
1965 Fingerprints. New York: Da Capo Press. The original was published in 1892.

Goodwin, D.
1976 Is Alcoholism Hereditary? New York: Oxford University Press.

Gordis, E., B. Tabakoff, D. Goldman, and K. Berg.
1990 Finding the Gene(s) for Alcoholism. The Journal of the American Medical Association 263 (15):2094-2096.

Holden, C.
1991 Probing the Complex Genetics of Alcoholism. Science 251:163-164.

Holt, S.
1968 The Genetics of Dermal Ridges. Springfield, IL: Charles C. Thomas Publisher.

Jantz, R.
1974 Multivariate Analysis of Dermatoglyphic Variation in Man. Yearbook of Physical Anthropology 18:121-139.

Jones, K., D. Smith, C. Ulleland, and A. Streissguth.
1973 Pattern of Malformation in Offspring of Chronic Alcoholic Mothers. The Lancet 1:1267-1271.

Kaij, L.
1960 Alcoholism in Twins. Stockholm: Almqvist & Wiksell.

Kaij, L.
1972 Definitions of Alcoholism and Genetic Research. Annals of the New York Academy of Sciences 197:111-113.

Kojic, T., A. Dojcinova, D. Dojcinov, O. Stojanovic, S. Jakulic, N. Susakovic, and V. Gligorovic.
1977 Possible Genetic Predisposition for Alcohol Addiction. Advances in Experimental Medicine and Biology 85A:7-25.

Meier, R.
1980 Anthropological Dermatoglyphics: A Review. Yearbook of Physical Anthropology 23:147-178.

Minitab, Inc.
1989 Minitab software program 7.2 system. State College, PA: Minitab, Inc.

National Council on Alcoholism, Criteria Committee.
1972 Criteria for the Diagnosis of Alcoholism. Annals of Internal Medicine 77(2):249-258.

Nordmo, S.
1959 Blood Groups in Schizophrenia, Alcoholism, and Mental Deficiency. The American Journal of Psychiatry 116(1):460-461.

Plato, C., J. Cereghino, and F. Steinberg.
1975 The Dermatoglyphics of American Caucasians. American Journal of Physical Anthropology 42 (2):195-211.

Polich, J., T. Burns, and F. Bloom.
1988 P300 and the Risk for Alcoholism: Family History, Task Difficulty, and Gender. Alcoholism: Clinical and Experimental Research 12:248-254.

Rignell, A.
1987 Variation studies of fingerprints in XXY Klinefelter's syndrome. Hereditas 106:139-145.

Seltzer, M., C. Plato, and K. Fox.
1990 Dermatoglyphics in the Identification of Women Either with or at Risk for Breast Cancer. American Journal of Medical Genetics 37(4):482-488.

Slatis, H., M. Katznelson, and B. Bonne-Tamir.
1976 The Inheritance of Fingerprint Patterns. American Journal of Human Genetics 28:280-289.

Swinson, R.
1972 Genetic Polymorphism and Alcoholism. Annals of the New York Academy of Sciences 197:129-133.

Uchida, I., J. Miller, and H. Soltan.
1964 Dermatoglyphics Associated with the XXYY Chromosome Complement. American Journal of Human Genetics 16(3):284-291.