



RACIAL DIFFERENCES IN URINARY
AMINO ACID EXCRETION IN MAN

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AJOVI B. SCOTT-EMUAKPOR
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ABSTRACT

RACIAL DIFFERENCES IN URINARY AMINO ACID EXCRETION IN MAN

By Ajovi B. Scott-Emuakpor

A population of healthy white and Negro boys who live together and take all their meals together has been studied for possible differences in their patterns of urinary amino acid excretion. Consistent differences between the boys are assumed to be genetic, except for the unlikely possibility that previous experience influences urinary amino acid excretion patterns. An average of nine fasting urine samples (taken first thing in the morning) was taken from each of forty-two boys of each race. The urines were subjected to two-dimensional paper chromatography and the relative density was measured for a series of fourteen spots on each chromatogram. The consistency of pattern for each individual is sufficiently great, that many of the boys could be ruled out as donors of a randomly chosen chromatogram.

Although there are no clear-cut racial differences between the white and the Negro boys in their pattern of urinary amino acid excretion, histidine and ornithine tend

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to be denser spots for whites, and glutamine and beta-amino-isobutyric acid (BAIB)--in BAIB "non-excretors"--tend to be denser for Negroes.

These differences can be used in making a probability statement about the race of the donor of a urine sample.

RACIAL DIFFERENCES IN URINARY AMINO ACID
EXCRETION IN MAN

by

Ajovi B. Scott-Emuakpor

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To Papa (who left), Mother
Bro. Lese, Brothers and Sisters
and
to Prof. Harman M. Slatis and Family

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INTRODUCTION

Several studies on urinary amino acid excretion following Harris's "high" and "low" excretor theory for beta-amino-isobutyric acid (Harris, 1953-54) have been carried out with little success. Sutton and Vanderberg (1953), Berry (1953) and Gartler et al. (1955) found that the excretion of glycine was under the control of a single autosomal recessive gene. At the same time, these same authors found the distinction made between "high" and "low" excretors of taurine by Dent and Harris (1951) relatively unsatisfactory. However, only very few studies have been made of racial differences in excretion patterns. Sutton and Clark (1955) found various differences between Chinese and Caucasians living in Michigan. In their study, the Chinese excreted significantly more beta-amino-isobutyric acid (BAIB), lysine, leucine, histidine, tyrosine and uric acid than the Caucasians. The Caucasians on the other hand excreted more of two unidentified substances that have color reactions with acid bromocresol green (BCG acid) and basic bromocresol green (BCG basic) than the Chinese. McEvoy-Bowe and Lugg (1961) also found differences between Caucasians, Chinese, and Malays living in and near Singapore. They found that the Chinese sample excreted low cystine as compared to the others and the

Senoi (a native Malay group) sample excreted low alanine and glutamine compared to the others. They also found notably higher glycine:tartrate ratios in the Caucasian data than in the others. The distribution of BAIB was reasonably different in the three groups as well. The Senoi sample had the most "high" excretors and the Chinese the fewest excretors. Gartler et al. (1957) found more BAIB excretors among the Apache Indians than among Caucasians, with "Black Caribs" intermediate. None of these racial studies, however, controlled sources of variability such as diet, age and sex, which probably produced some of the differences that have been observed.

The present study was designed to search for possible racial differences in amino acid excretion patterns between American Negroes and Caucasians. All the known sources of variability were reasonably controlled. The boys chosen for this study were between 13 and 18 years old. They live together in an unsegregated training school for socially delinquent boys. Both racial groups are largely from socio-economically inadequate homes, though there are probably systematic differences between the races. Thus we cannot eliminate the (very unlikely) possibility that previous experience affects the pattern of excretion. All the boys were on the same diet, individuals rarely supplemented this diet in any way, and rules forbid the exchange of food items between boys, so that patterns of food consumption are roughly comparable for all individuals.

REVIEW

Mammalian urine is a complex mixture of substances. Consequently, an analysis of urine for its various constituents is also a complex process. Some of the substances present in urine, such as urea and creatinine, are excreted by all humans in quantities proportional to body mass and to the rate of metabolism. Others such as methyl-histidine do not form regular constituents of urine. They are excreted only after certain foods have been taken. It has been shown by Datta and Harris (1951) that methyl-histidine is a break-down product of anserine which is a prominent constituent of vertebrate muscle, and hence it is excreted in large amounts following a meal of meat. Yet other substances are excreted in highly variable amounts and in this group belong most of the free amino acids.

A variety of amino acids is excreted in the urine of man. The relative amounts of each amino acid is characteristic of the individual, his state of health and his diet. The variations in the amounts of amino acid excreted may be due to two major factors. They may be due to the concentration of some amino acid in the blood, and they may be due to the height of renal threshold. Whatever the case, it has generally been presumed that these patterns are

under genetic control, but only a few examples of this control have been identified. The ability to excrete BAIB in large amounts is controlled by a common recessive gene (Harris, 1953), but there is an overlap between "high" and "low" excretors so that many people are in the intermediate region (Gartler et al., 1957). Persons who excrete relatively large amounts of certain other amino acids have also been described, and these appear to be genetically determined, but such individuals are physically and/or physiologically abnormal in most cases and are also rare. In this group are the excretors of phenylalanine (Medes, 1932), histidine (Ghadimi et al., 1961, 1962), tryptophan (Tada et al., 1963), cystine (Fox et al., 1964, and Berry, 1959), methionine (Hooft et al., 1964), ornithine (Russel et al., 1962), proline (Schafer et al., 1962, and Scriver et al., 1961), and glutamic acid (Menkes et al., 1962). An autosomal recessive gene is said to control the excretion of all of the above amino acids except glutamic acid which is believed to be under the control of a sex-linked recessive gene. Disease conditions, such as cirrhosis and hepatitis, are also associated with high amino acid excretion (Kirsner et al., 1949, 1950).

A considerable amount of disagreement exists as to whether the regular pattern of excretion of urinary amino acids is affected by diet. Eckhardt and Davidson (1948, 1949) feel that variations in the pattern are as

a result of differences in urine preservation rather than by differences in the amount of food ingested. In support of this Fowler et al., (1956) argued that what might be excessive excretion by one criterion may be normal or even below normal by another. Berry (1953) suggested that urinary constituents are only slightly under dietary influence, and that most of the variance observed between individuals is genotypically conditioned.

Age and sex have also been shown to affect the excretion pattern of urinary amino acids. Jagenburg (1959) found that age has only slight but characteristic effects on the amino acid pattern. He found that the excretion of ethanolamine and taurine were very high in the first 24 hours of life. Proline and hydroxy-proline were prominent in infants and not in adults, while histidine and methyl-histidine were excreted, in infancy, only in very small amounts and sometimes were absent. Glycine, in Jagenburg's study, seemed to be excreted in greater amounts during the 6th day of life than at any of the other age periods studied. Sex has been found to have somewhat less profound effects under normal physiological conditions. However, during pregnancy it has been found that histidine is considerably elevated (Honda, 1923; Voge, 1929; and Langley, 1941). Further studies by Lawrie (1947) and Christensen et al., (1957) have shown that apart from elevated histidine, pregnant women also exceed males in the excretion of free lysine, arginine, serine, threonine, tyrosine and tryptophan.

MATERIALS AND METHODS

The subjects were divided into four groups, each consisting of members of a different living unit. From each unit, ten or eleven boys of each race were chosen for study. Boys with a large amount of American Indian ancestry (determined visually) were excluded, as were light-pigmented Negroes, so that the "Caucasian" sample was almost entirely of European ancestry and the "Negro" sample was of predominantly African ancestry. Individuals on drug therapy were also excluded. Urine samples were taken from all boys in a living unit so that excluded individuals did not realize that they were not part of the sample.

Early morning urine samples (fasting samples) were taken from each group on ten mornings within a ten or eleven day period.

Whatman 3mm chromatographic paper was used, and 100 μ l of the urine was slowly placed in a spot and dried with warm air using a hair drier. The chromatographic system used is shown in Figure 1. The rack is made of stainless steel and consists of two flat square plates connected by four rods that are fastened to the plates with nuts (as described by Block, Durrum and Zweig, 1958). The Whatman paper is cut to be slightly larger than the size of the

square plates and four holes are punches at its four corners. The paper sheets are then placed on the rack by stacking them on the rods through the holes. The sheets are separated from each other by 1-inch spacers made of glass tubing. Ten sheets of Whatman paper were usually stacked on each rack. When placed in a chromatography jar, the steel racks were raised off the bottom of the jar by glass rods, and the papers extended below the racks into the solvent.

The solvent was put in the air-tight chromatography jar (sealed with a greased glass plate) some two hours before the insertion of the rack in order to allow thorough equilibration. The first direction of chromatography was in a solvent 100 ml. butyl alcohol, 100 ml. methyl alcohol, 50 ml. deionized water, and 1 ml. glacial acetic acid. The second direction was in a solvent of 150 ml. butyl alcohol, 150 ml. acetone, 75 ml. deionized water, 10 ml. diethylamine and 0.25 ml. 1M ammonium hydroxide. Ascending chromatography was run in the first direction for 20 hours, dried for at least four hours and then run in the second direction for another 20 hours. After drying, the papers were briefly immersed in a solution of 0.75 gm. of ninhydrin in 600 ml. of acetone and then dried in a fumehood for 5 minutes, and transferred into an oven held between 70° to 80° for 10 minutes.

The density of each of 14 amino acid spots on the chromatograms was determined by measuring the amount of

light transmitted through its center. The readings were done on a crude photographic light meter consisting of a photo-electric cell and a pointer that moves over a logarithmic scale. A series of tests has shown that this method gives consistent results and that these results are not affected by a wide range of variations in the amount of urine used. However it is not possible to convert these results into measurements of the concentration of each amino acid in the urine. This method of reading the spots is as efficient as, if not more efficient than, the elution technique which is very widely used. The elution technique involves cutting out a spot, eluting its color with 20% alcohol, and reading the light transmittance in a spectrophotometer. Table 1 gives the results obtained by these two methods from 100 lambda of the same urine sample chromatographed three times. The two methods are highly correlated as testified by the correlation coefficients of 0.97348, 0.95830 and 0.97560 for the first, second and third runs respectively. Although it is known that the intensity of color in the amino acid-ninhydrin reaction is directly proportional to the amount of amino acid present in the reaction, the results are only interpretable indirectly from the relative density of the spots produced by this method of analysis, rather than in values directly related to their true concentrations in the urine.

The densest amino acid on a chromatogram was given an arbitrary value of 30, the next densest was given a value of 28, the next a value of 26, and so on. A total of 14 amino acids were measured in this way, so that the least dense spot received a value of 4. If two or more spots had the same density, they received the same value which in this system is always an integer (for example, if two spots were of equal density and were denser than any other spot on the chromatogram, each would get a value of 29, and the third densest spot would get a value of 26). If an amino acid appeared to be missing, a reading would be made at the point at which it would be expected. Except for the "lower histidine" spot, missing spots were rare.

Table 1.--Comparison between Elution technique and Photographic light meter readings as a means of reading the relative intensity of spots on a chromatogram.

Amino Acids	First Run		Second Run		Third Run	
	Elution & trans-mission	Light meter reading	Elution & trans-mission	Light meter reading	Elution & trans-mission	Light meter reading
Glutamic acid	88.2	13.6	87.0	13.5	88.0	13.5
Glutamine	81.5	10.3	82.0	10.1	80.0	10.3
Glycine	72.5	9.2	71.7	9.2	70.5	8.9
Serine	84.1	11.0	83.2	11.1	84.0	11.5
Taurine	86.0	11.8	87.0	12.0	85.0	12.0
Threonine	90.0	13.9	89.0	13.8	91.0	13.5
Histidine (up.)*	78.0	9.5	79.2	9.6	78.5	9.2
Histidine (low)	--	--	--	--	--	--
Ornithine	95.0	14.4	94.5	14.4	95.0	14.5
Alanine	92.5	14.0	94.5	14.2	92.2	14.0
Cysteine	96.2	14.6	95.0	14.5	97.0	14.5
Phenylalanine	97.5	15.0	98.1	15.0	97.5	15.0
Tryptophane	97.0	14.8	98.2	14.8	97.2	14.9
BAIB	98.0	15.6	98.2	15.5	98.0	15.8
Tyrosine**	--	--	--	--	--	--
Correlation Coefficient	0.97348		0.95830		0.97560	

*Two spots can be identified for histidine. The upper spot is always present, but the lower spot may or may not be present. No reason has been found for this strange phenomenon.

**The spot identified as tyrosine was not included in this part of the experiment. However, it is suspected to be a complex spot of several ninhydrin-positive compds. with Tyrosine as the major contributor.

RESULTS

Figure 3 shows the relative positions of the major spots at the time the colors are developed, and the chief amino acid that has been identified with each spot. Figure 2 is a picture of a typical chromatogram.

The raw data for most of the boys consist of ten observations (average: 9.13). In general, there was a good deal of consistency for each individual from day to day and often quite a lot of differentiation between individuals.

The 14 amino acids selected for this study are glutamine, glycine, serine, taurine, threonine, histidine, ornithine, alanine, cysteine, phenylalanine, tryptophan, beta-amino-isobutyric acid (BAIB), and tyrosine. The spot identified for tyrosine is probably a complex spot that includes a lot of other ninhydrin-positive compounds in the urine. Although the sample size is too small to allow any statement regarding "high" and "low" excretors to be valid, suggestive trends were observed. Table 2 gives the values of "high," normal" and "low" excretors for the various amino acids as computed from the data. Because the method of segregation of "high" and "low" excretors is a crude one by this technique, attention is drawn only to

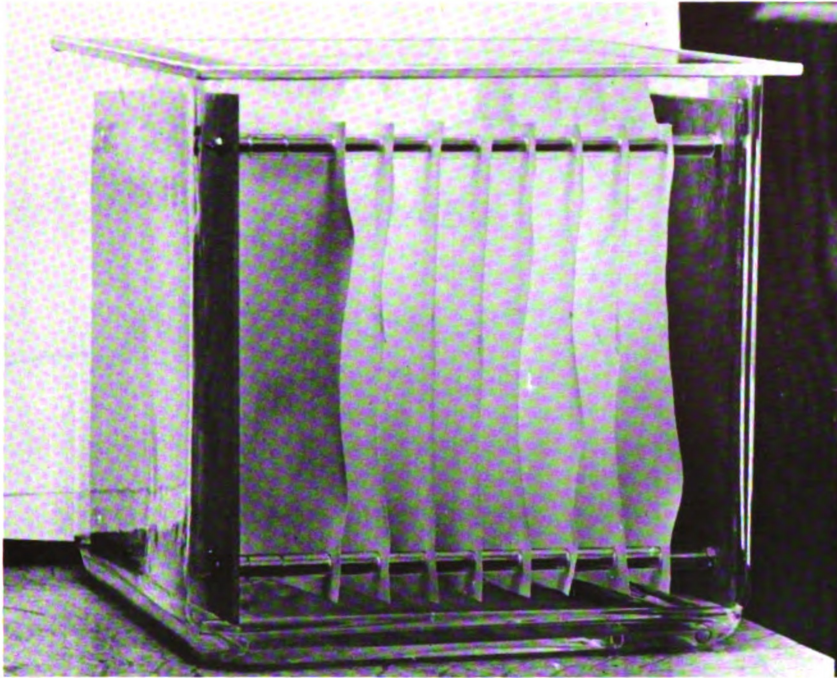


Figure 1.--Picture of chromatographic system.

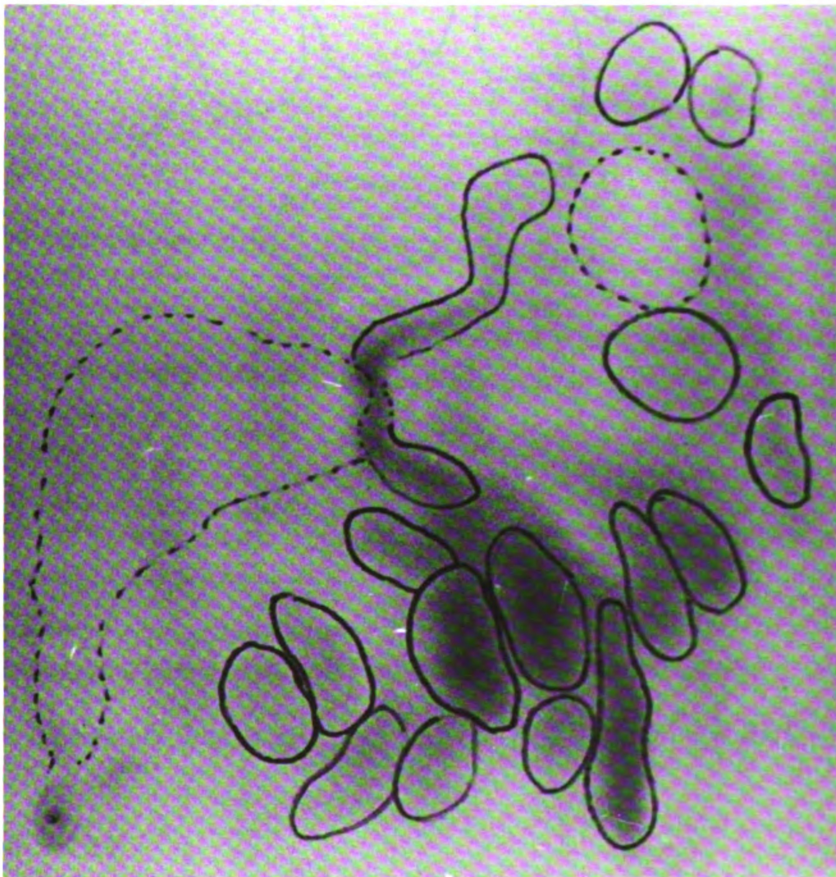


Figure 2.--Picture of a typical chromatogram.

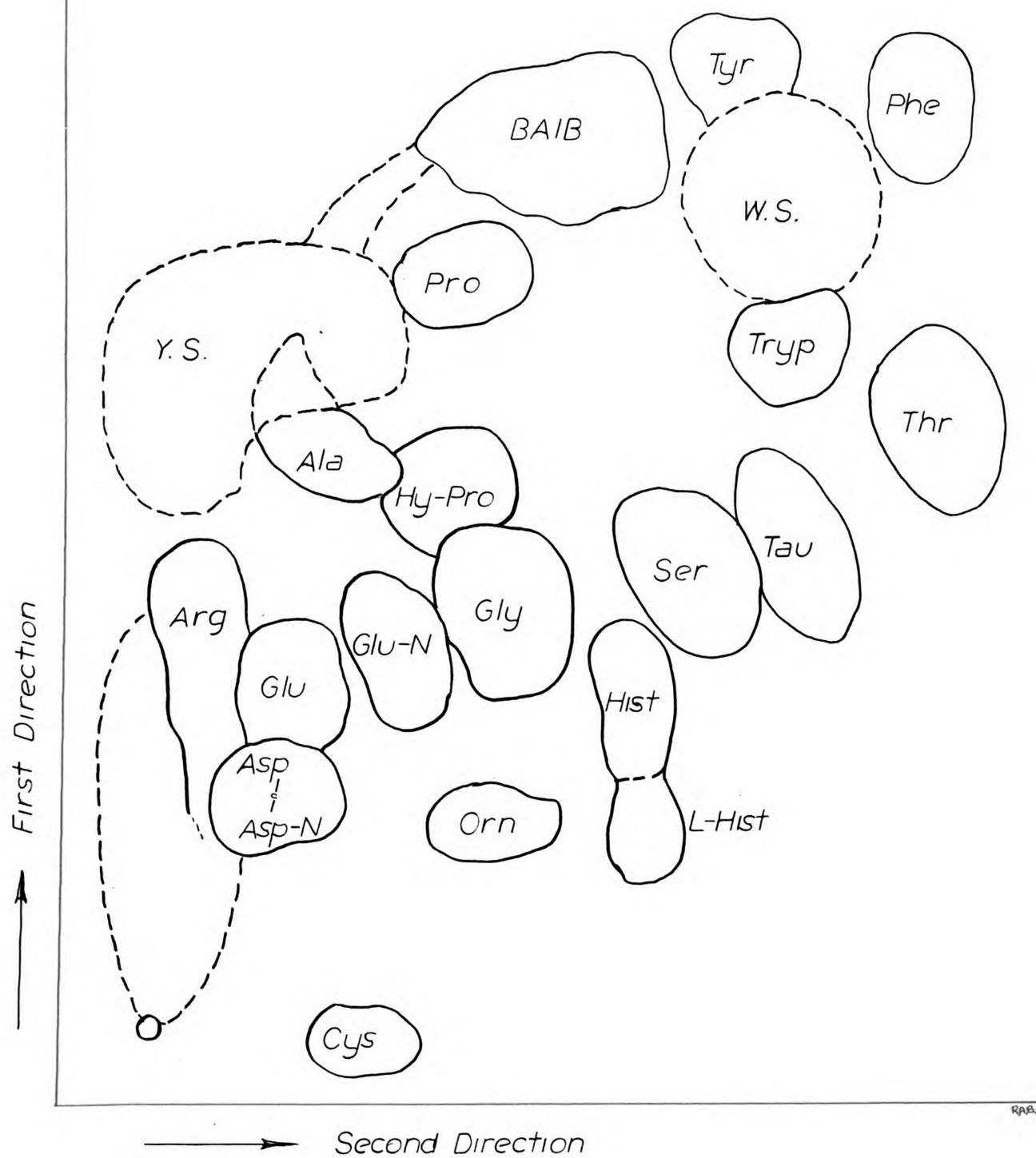


Figure 3.--Diagram showing relative position of amino acids.

Table 2.--"High," "Normal" and "Low" excretors for the individual amino acids.

Amino Acids	Mean Rank**	Absolute Numbers of Excretors*									
		White			Negro			Total			
		High	Normal	Low	High	Normal	Low	High	Normal	Low	
Glutamic acid	18.3	-	42	-	1	40	1	1	82	1	
Glutamine	26.5	-	42	-	-	42	-	-	84	-	
Glycine	29.8	-	42	-	-	42	-	-	84	-	
Serine	23.3	-	41	1	-	42	-	-	83	1	
Taurine	21.2	1	38	3	1	41	-	2	79	3	
Threonine	11.5	3	37	2	4	37	1	7	74	3	
Histidine	27.0	-	41	1	1	40	1	1	81	2	
Ornithine	14.0	8	30	4	6	27	9	14	57	13	
Alanine	15.4	2	34	6	2	38	2	4	72	8	
Cysteine	15.5	2	38	2	1	39	2	3	77	4	
Tryptophan	8.5	5	33	4	4	36	2	9	69	6	
Phenylalanine	10.0	-	41	1	3	36	3	3	77	4	
BAIB	8.9	3	39	-	4	38	-	7	77	-	

*"High" excretors are those whose values fall above the mean rank by, at least 3 units.

"Normal" excretors are those whose values are less than 3 units above and below the mean rank.

"Low" excretors are those whose values fall below the mean rank by, at least 3 units.

**The mean ranks are, in every case, the grand mean of the individual means; and the mean of an individual is the average of light values for about 9 urine samples.

possibilities and not to facts. In general, the relative density of each amino acid is comparable from person to person. A "high" excretor is defined as a person who is 3 units or more above the mean rank, and a "low" excretor is defined as a person who is 3 units or more below the mean rank. The data presented in Table 3 show that although we have possibly one "high" and one "low" excretor for glutamic acid among the Negroes, no white boy showed either pattern. Another possible Negro-white difference that is apparent from the data is in the excretion of ornithine and phenylalanine. There seems to be a shift in the trend of "high" and "low" ornithine excretors from more "high" than "low" excretors among the whites to more "low" than "high" excretors among the Negroes. For phenylalanine, whereas there were no "high" excretors among the whites, there were 3 out of 42 (7.1 per cent) "high" excretors among the Negroes. Interestingly enough, there were no unusually low excretors for BAIB. For "high" excretors of BAIB, there were 3 out of 42 (7.1 per cent) whites and 4 out of 42 (9.6 per cent) Negroes. Taking both groups together, we have 7 out of 84 (8.8 per cent) "high" excretors of BAIB, which value is in reasonable agreement with Harris's 9.6 per cent (Harris, 1953).

Two amino acids that tend to exchange positions in the two groups are glutamine and histidine. Among whites, histidine tend to be the second densest spot while

glutamine is the third densest spot. Among Negroes glutamine tends to occupy the second position while histidine is third. Table 3 gives the number of white and Negro urine samples that contain more glutamine than histidine, those that have the same amount of glutamine and histidine and those that have more histidine than glutamine. It was found that of the 394 urine samples analyzed for whites, only 75 (19.035 per cent) showed more glutamine than histidine while 300 (76.142 per cent) showed more histidine than glutamine. Among the Negroes, of the 373 samples analyzed, 175 (46.917 per cent) showed more glutamine than histidine, while 173 (46.381 per cent) showed more histidine than glutamine.

The unweighted means of the results for each boy were used as the information subjected to analysis in this study. The overall racial means for the relative density of spots are presented in Table 4.

The results as presented in Table 4 do not show any clear distinction between the races. However, there are minor differences that are helpful in distinguishing boys of the two races. The major difference is that glutamine is relatively denser on the chromatograms of Negroes than on those of whites. The mean for Negroes, 26.871, is only 0.714 above the mean for whites, 26.157. The standard deviations, 0.655 and 0.541, respectively, are almost as large as this difference which indicates that a relatively

Table 3.--A table showing the number of urine samples that contain more glutamine than histidine for both races.

Races	Number of Urine Samples	More glutamine than histidine	Tie	More histidine than glutamine
Whites	394	75	19	300
Negroes	373	175	25	173

Table 4.--Overall Racial Means for the Relative Density of Spots.

Amino Acids	White	Negro	Differences (white-Negro)
Glutamic acid	18.3	18.3	0
Glutamine	26.2	26.9	-0.7
Glycine	29.8	29.8	0
Serine	23.2	23.5	-0.3
Taurine	21.1	21.3	-0.2
Threonine	11.4	11.7	-0.3
Histidine (Up.)	27.3	26.7	+0.6
Histidine (Lo.)	9.0	8.9	+0.1
Ornithine	14.7	13.4	+1.3
Alanine	15.2	15.7	-0.5
Cysteine	15.6	15.3	+0.3
Tryptophan	8.5	8.5	0
Phenylalanine	10.1	10.0	+0.1
BAIB	8.5	9.3	-0.8
Tyrosine	20.0	19.7	+0.3

large proportion of each race will fall into the mode set by the other race. If the midpoint of the two races, 26.514, is chosen as the decision point, 26.2 per cent of Negroes will be below this point and so will be misclassified, while 23.8 per cent of the whites will be above this point and will be misclassified, as shown in Figure 4. An analysis of variance for glutamine is shown in Table 5. Individual variability within each race has F equal to 10.1860, which is significant at the 1 per cent level while a test of homogeneity between the races has F equal to 9.6207 which also is significant at the 1 per cent level. This means that although individuals of the same race differ significantly from each other, the races also differ significantly. Therefore it appears that Negroes excrete relatively greater amounts of glutamine than do whites, although the two distributions overlap. Ornithine, which shows a greater mean difference between the races, showed individual variability within each race to be significantly different at the 1 per cent level, but showed that both races constituted a homogeneous population.

A discriminant analysis has been performed to determine the best formula for distinguishing members of the two races. A series of analyses found that the best discrimination was obtained from the data for glutamine (GLN), serine (SER), threonine (THR), and cysteine (CYS). The discriminant function is:

Table 5.--Analysis of Variance for glutamine.

Sources of Error	D.F.	S.S.	M.S.S.	F
Races (R)	1	111.6	111.6	R/P = 9.6207*
People (P)	82	949.6	11.5805	P/S = 10.1860*
Sample (S)	683	776.5	1.1369	
Total	766			

D.F. is Degree of freedom.

S.S. is Sums of squares.

M.S.S. is Mean sums of squares.

*Significant at 1 per cent level.

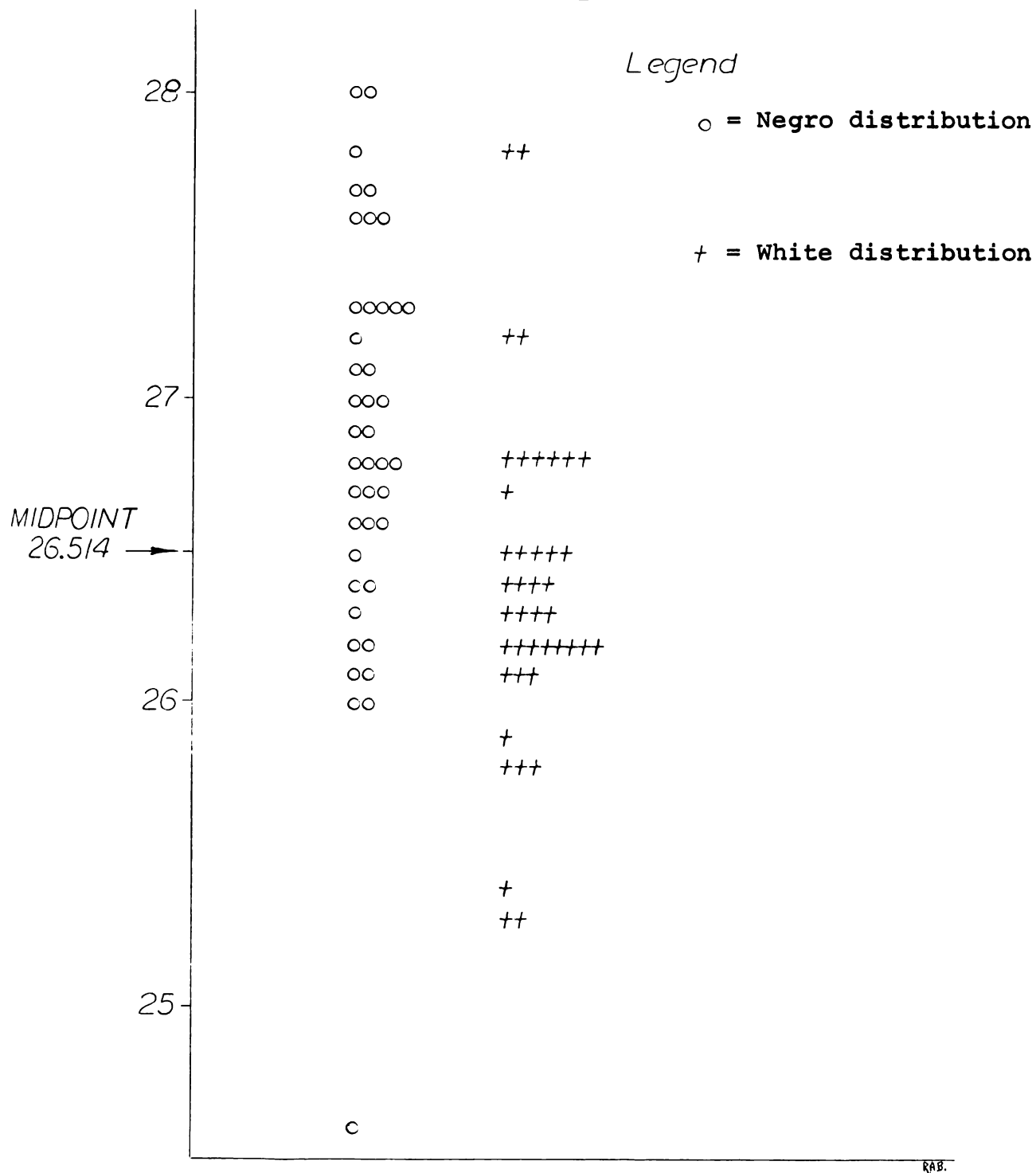
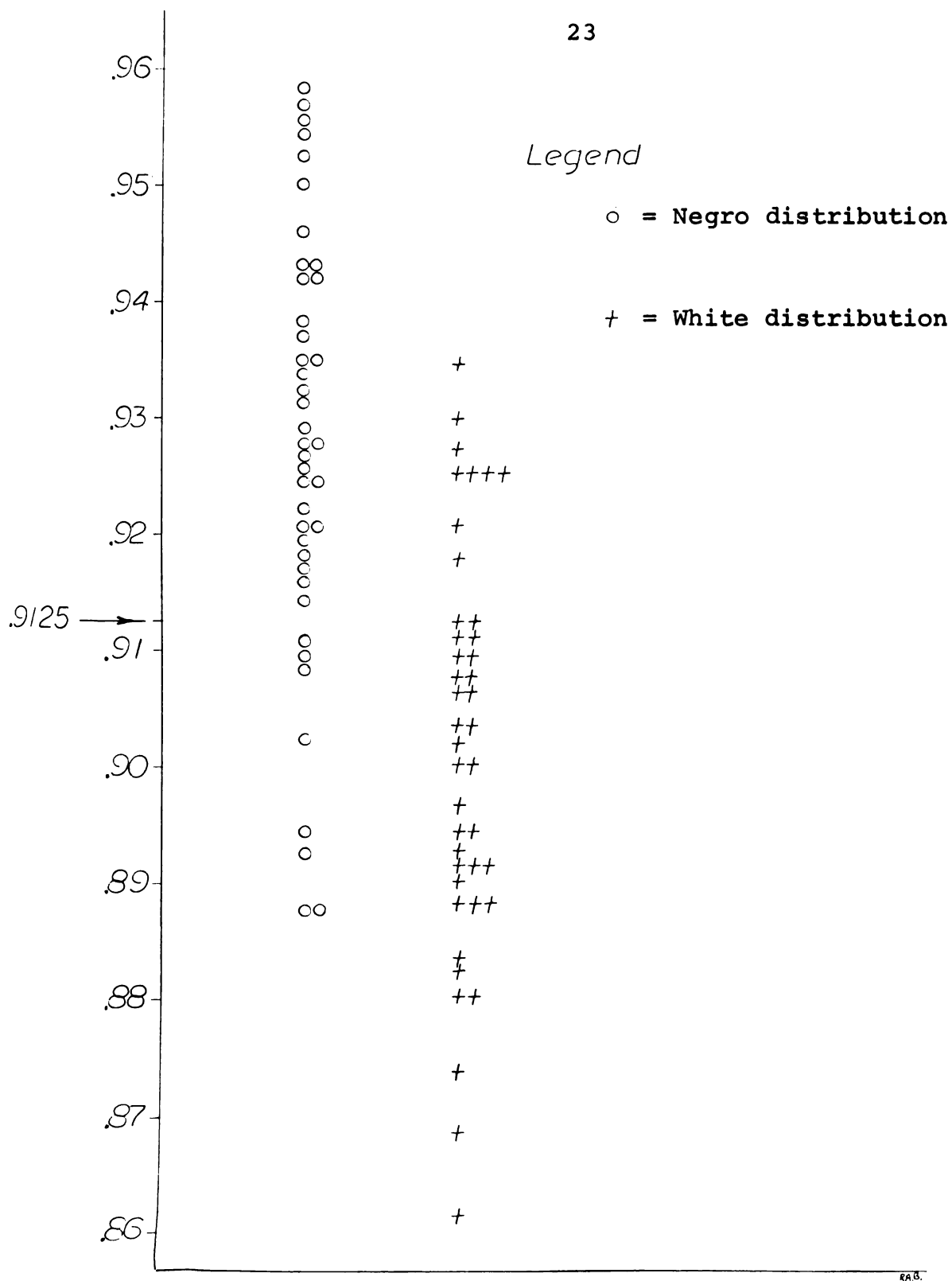


Figure 4.--Distrubution of Negro and White glutamine values.

$$0.03123 \text{ GLN} + 0.00479 \text{ SER} + 0.00262 \text{ THR} - 0.00360 \text{ CYS}$$

With this function, the mean value for Negroes is 0.92729 and that for whites is 0.90147, which is a difference of 0.02582. The standard deviations with each race are 0.01376, and 0.01687, respectively. Figures 5 and 6 represent a distribution of Negro and white discriminant values. Using a dividing point of 0.9127, 21.4 per cent of the Negroes will be below this point and hence will be misclassified as whites while the same percentage of whites will be above this point and will be misclassified as Negroes.



Legend

- = White distribution
 — = Negro distribution

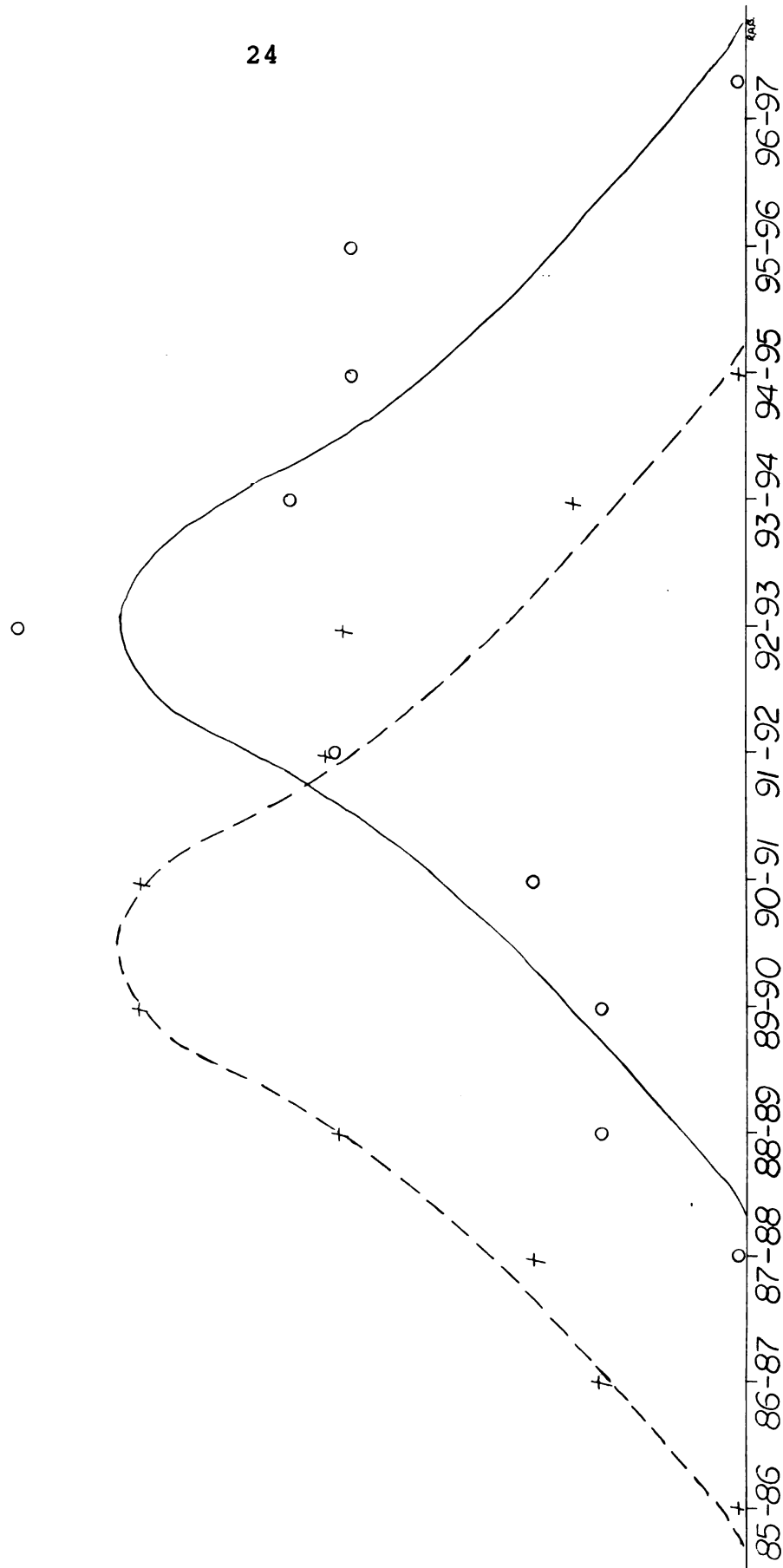


Figure 6.--Distribution of Negro and White discriminant factors.

DISCUSSION

The purpose of this study was to determine if racial differences in urinary amino acid excretion can be detected between whites and Negroes living under identical conditions. The subjects were healthy adolescent boys, which gave the group a great deal of uniformity except for the racial difference.

The chromatographic system that was used was chosen because it provides a large amount of data relative to the effort involved. It was possible for a single individual to analyze twenty urine samples per day as a routine procedure.

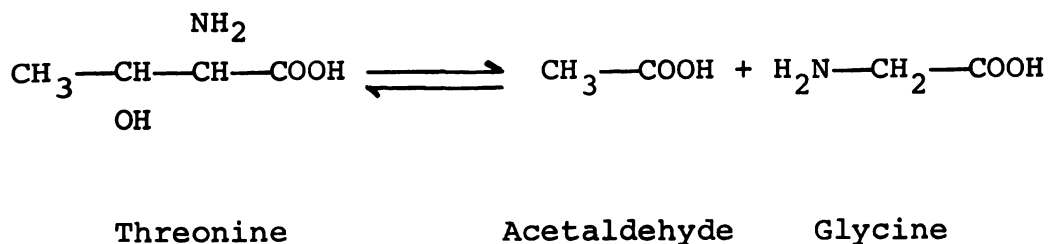
High excretion of an amino acid may be as a result of one or more of several factors:

1. It may be due to overproduction of the amino acid.
2. It may be due to the inability of the system to utilize the particular amino acid.
3. It may be due to very low renal threshold.

The first and second reason will concentrate the amino acid in the blood, while the third reason will cause the urine to have too much of the amino acid without necessarily being too high in the blood. The genetic

interpretation that can be given to high excretion of an amino acid will depend upon the cause. If it is due to an enzyme defect, a single gene (which may be autosomal or sex-linked and may be recessive, semi-dominant or dominant) is often said to be responsible. On the other hand, if the defect is traced to renal malfunction, except if familial studies prove it to be genetic, it may be pathologic.

Interestingly enough, of the seven people who were classified as "high" excretors of threonine, six of them showed trends of excreting moderately higher amounts of glycine when expressed in terms of its light value relative to the other amino acids and not in terms of its rank. The reason for this is easy to explain from the biochemical relationship between glycine and threonine. It is known that in the liver and kidney of mammals, the enzyme threonine aldolase (in the presence of a cofactor, pyridoxal phosphate) catalyses the following reversible reaction.



By this method of analysis (the ranking of light values), it was not possible to pick out "high" excretors of glycine. However, it would require a more quantitative analysis and thorough enzyme assays to confirm the presence of such a relationship between glycine and threonine in the urine and to determine the position of the primary cause.

There are no striking differences in excretion patterns between the two races. However, the minor difference in the relative amounts of glutamine in the urine can be used to make a probability statement about the race of the donor of a given urine sample. Among the 30 per cent of the chromatograms that have glutamine as the densest or second densest spot, the probability that the chromatogram comes from a Negro donor is 0.72, while among the six per cent of the chromatograms that do not have glutamine among the three densest spots, the probability is of the same magnitude that the donor is white. Similarly, among the 62 per cent of the chromatograms that have histidine as the densest or second densest spot, the probability that the chromatogram comes from a white donor is 0.64.

The probability of guessing correctly about the race of an individual is increased by taking the average of several chromatograms, and this is slightly improved by a discriminant function analysis which combines information on the relative densities of glutamine, serine,

threonine and cysteine. The addition of information on the relative densities of the other amino acids did not increase the power of this method in separating the two races.

The difference reported here may not be due to a major gene effect. It may be a result of the genetic background of the individuals; in which case, it is a result of many genes.

The racial differences that this study was designed to establish were not distinctly obvious. However, it was slightly obvious that the pattern of excretion of glutamine was different enough to separate the races, which separation is made slightly better by a discriminant analysis. The racial studies on urinary amino acid excretion by Sutton and Clark (1955), Gartler et al. (1956-57), and McEvoy-Bowe and Lugg (1961) failed to show this racial difference. This difference may be made even more obvious if a much larger population is considered.

REFERENCES

- Berry, H. K. Variations in urinary excretion patterns in a Texas population. Amer. J. phys. Anthropol. 11, 559, 1953.
- . Further studies on individual urinary and salivary amino acid patterns. Univ. Texas publ. No. 5109, 157, 1951.
- Berry, H. K., and Cain, L. Individual urinary excretion patterns of young children. Univ. Texas publ. No. 5109, 165, 1951.
- Berry, H. K. Cystinuria in mentally retarded siblings with a typical osteogenesis imperfecta. A.M.A. J. dis. Child. 97, 196, 1959.
- Block, R. J., Darrum, E. M., and Zweig, G. A manual of paper chromatography and paper electrophoresis. Academic Press, Inc., N.Y., 1958.
- Christensen, P. J., Date, J. W., Schonheyder, F., and Volqvartz, K. Amino acids in blood plasma and urine during pregnancy. Scand. J. clin. Lab. Invest. 9, 54, 1957.
- Datta, S. P., and Harris, H. Dietary origin of urinary methyl-histidine. Nature 168, 296, 1951.
- Dent, C. E., and Harris, H. The genetics of cystinuria. Ann. Eugen. (Lond.) 16, 60, 1951.
- Eckhardt, R. D., and Davidson, C. S. Urinary excretion of amino acids following the rapid injection of a solution of amino acids in man. J. clin. Invest. 27, 727, 1948.
- . Urinary excretion of amino acids by a normal adult receiving diets of varied protein content. J. biol. Chem. 177, 687, 1949.
- Fowler, D. I., Norton, P. M., Cheung, M. W., and Pratt, E. L. Observations on the urinary amino acid excretion in man: the influence of age and diet. Arch. Biochem. 68, 452, 1957.

- Fox, M., Thier, S., Rosenberg, L., Kiser, W., and Segal, S. Evidence against a single renal transport defect in cystinuria. New Eng. J. Med. 270, 556, 1964.
- Gartler, S. M., Firschein, I. L., Gidaspow, T. Some genetical and anthropological considerations of urinary beta-aminoisobutyric acid excretion. Acta Genet. (Basel) Suppl. No. 6, 435, 1956-57.
- Gartler, S. M., Dobzhansky, T., and Berry, H. K. Chromatographic studies on urinary excretion patterns on monozygotic and dizygotic twins. II. Heritability of the excretion rates of certain substances. Amer. J. hum. Genet. 7, 108, 1955.
- Ghadimi, H., Partington, M. W., and Hunter, A. A familial disturbance of histidine metabolism. New Eng. J. Med. 265, 221, 1961.
- _____. Inborn error of histidine metabolism. Pediatrics 29, 714, 1962.
- Harris, H. Familial studies on the urinary excretion of beta-amino isobutyric acid. Ann. Eugen. (Lond.), 18, 43, 1953-54.
- _____. Aminoaciduria in man. Third International Congress of Biochemistry (abstract), 1955.
- Hoof, C., Timmermans, J., Snoeck, J., Antener, I., Oyaert, W., and Van den Hende, Ch. Methionine malabsorption in a mentally defective child. Lancet ii, 20, 1964.
- Honda, M. Untersuchung des Harns gravider Frauen. J. Biochem. (Japan) 2, 351, 1923 (abstract).
- Jagenburg, R. The urinary excretion of free amino acids and other amino compounds by the human. Scand. J. clin. Lab. Invest. suppl. 43, vol. 11, 1959.
- Kirsner, J. B., Sheffner, A. L., and Palmer, W. L. Studies on amino acid excretion in man. III: Amino acid levels in plasma and urine of normal men fed diets of varying protein contents. J. clin. Invest. 28, 716, 1949.
- Kirsner, J. B., Sheffner, A. L., Palmer, W. L., and Bergeim, O. Amino acids in plasma and urine of patients with hepatitis before and after a single infusion of protein hydrolysate. J. Lab. clin. Med. 36, 735, 1950.

- Langley, W. D. Urinary histidine. Determination of histidine in normal and in pregnancy urines. J. biol. Chem. 137, 254, 1941.
- Lawrie, N. R. The excretion of l(-) tyrosine in urine. Biochem. J. 41, 41, 1947.
- McEvoy-Bowe, E., and Lugg, J. W. H. A direct quantitative paper chromatography of amino acids and application to the urinary excretion of some human ethnic groups. Biochem. J. 80, 616, 1961.
- Modes, G. A new error tyrosine metabolism: tyrosinosis, the intermediary metabolism of tyrosine and phenylalanine. Biochem. J. 26, 917, 1932.
- Menkes, J. H., Alter, M., Steigleder, G. K., Weakley, D. R., and Sung, J. A. A sex-linked recessive disorder with retardation of growth, peculiar hair, and focal cerebral and cerebellar degeneration. Pediatrics 29, 764, 1962.
- Russel, A., Levin, B., Oberholzer, V. G., and Sinclair, L. Hyperammonaemia: a new substance of an inborn enzymatic defect of the biosynthesis of urea. Lancet ii: 699, 1962.
- Schafer, I. A., Scriver, C. R., and Efron, M. L. Familial hyperprolinemia, cerebral dysfunction, and renal anomalies occurring in a family with hereditary nephropathy and deafness. New Eng. J. Med. 267, 51, 1962.
- Scriver, C. R., Schafer, I. A., and Efron, M. L. New renal tubular amino acid transport system and a new hereditary disorder of amino acid metabolism. Nature 192, 672, 1961.
- _____. Evidence for a renal tubular amino acid transport system common to glycine, L-proline and hydroxy L-proline. J. clin. Invest. 40, 1080, 1961.
- Sutton, H. E., and Clark, P. J. A biochemical study of Chinese and Caucasoids. Amer. J. phys. Anthropol. 13, N.S. 53, 1953.
- Sutton, H. E., and Vanderberg, S. G. Studies on the variability of human urinary excretion pattern. Hum. Biol. 25, 318, 1953.
- Tada, K., Ito, H., Wada, Y., and Arakawa, T. Congenital tryptophanuria with dwarfism. Tohoku J. Exper. Med. 80, 118, 1963.
- Voge, C. A simple chemical test for pregnancy. Brit. med. J. 2, 829, 1929.

ADDITIONAL REFERENCES

- Allan, J. D., Cusworth, D. C., Dent, C. E., and Wilson, V. K. A disease, probably hereditary, characterized by severe mental deficiency and a constant gross abnormality of amino acid metabolism. Lancet i:182, 1958.
- Berry, H. K., Cain, L., and Rogers, L. L. A study of the urinary excretion patterns of six human individuals. Univ. Texas publ. No. 5109, 150, 1951.
- Clarkson, T. W., and Kench, J. E. Urinary excretion of amino acids by man absorbing heavy metals. Biochem. J. 62, 361, 156.
- Datta, S. P., and Harris, H. Urinary amino acid patterns of some mammals. Ann. Eugen. (lond.) 18, 107, 1953.
- Dent, C. E. Detection of amino acids in urine and other fluids. Lancet 2, 637, 1946.
- _____. A study of the behavior of some sixty amino acids and other ninhydrin-reacting substances on phenol-"collidine" filterpaper chromatograms, with notes as to the occurrence of some of them in biological fluids. Biochem. J. 43, 169, 1948.
- _____. Clinical applications of amino acid chromatography. Scand. J. clin. Lab. Invest. 10, suppl. 31, 122, 1957.
- Dingham, H., and Wright C. A multivariate analysis of amino acid excretion. J. Men. Def. Research 8(1), 77, 1964.
- Dustin, J. P., Moore, S., and Bigwood, E. J. Chromatographic studies on the excretion on the excretion of amino acids in early infancy. Metabolism 4, 75, 1955.
- Eckhardt, R. D., Cooper, A. M., Faloon, W. W., and Davidson, C. S. The urinary excretion of amino acids in man. N.Y. Acad. Sci. Trans. 10, 284, 1948.

- Evered, D. F. The excretion of amino acids by the human. A quantitative study of ion-exchange chromatography. Biochem. J. 62, 416, 1956.
- Gartler, S. M. A family study of urinary beta-aminoisobutyric acid excretion. Amer. J. hum. Genet. 8, 120, 1956.
- Jagenburg, R. Amino acids and peptides in infant urine. Acta chem. scand. 9, 1047, 1955.
- Miller, S., Ruttinger, V., and Macy, I. G. Urinary excretion of ten amino acids by women during the reproductive cycle. J. biol. Chem. 209, 795, 1954.
- Moore, S., and Stein, W. H. A modified ninhydrin reagent for the photometric determination of amino acids and related compounds. J. biol. Chem. 211, 907, 1954.
- Mueller, G. C., Bowman, G., Herranen, A. Desalting amino acid solutions by ion exchange. Analyt. Chem. 27, 1357, 1955.
- Nasset, E. S., and Tully, R. H. Urinary excretion of essential amino acids by human subjects fed diets containing proteins of different biological value. J. Nutr. 44, 477, 1951.
- Norton, P. M., Pratt, E. L., and Hasselmeier, E. Urinary amino acid excretion by premature infants. Amer. J. dis. Child. 88, 659, 1954.
- Rubenstein, M., and Wolff, S. M. Amino acid excretion in familial mediterranean fever. Arch. Internal Med. 113(3), 409, 1964.
- Salisbury, P. F., Dunn, M. S., and Murphy, E. A. Apparent free amino acids in deproteinized plasma of normal and uremic persons. J. clin. Invest. 36, 1227, 1957.
- Sandler, M., and Pare, C. M. B. Starvation amino aciduria. Lancet 1, 494, 1954.
- Slatis, H. M. Normal variations in excreted amino acids in man. Proc. XI International Congress of Genetics, 1963 (abstract).

- Smith, I. Colour reactions on paper chromatograms by a dipping technique. Nature 171, 43, 1953.
- Soupart, P. Urinary excretion of free amino acids in normal adult men and women. Clin. chim. Acta 4, 265, 1959.
- Sutton, H. E. A further study of urinary excretion patterns in relation to diet. Univ. Texas publ. No. 5109, 173, 1951.

APPENDICES

Appendix A.1.--Table of Means.

Negroes	GLU.	GLU-N.	GLY.	SER.	TAU.	THREO.	U-HIST.	L-HIST.	ORN.	ALA.	CYST.	TRYP.	PHE.	BAIB	TYR.
Battle	17.3	26.8	30.0	23.8	21.8	14.6	27.2	--	14.1	15.1	14.9	15.1	13.0	8.2	--
Brown	20.0	26.3	30.0	24.4	22.0	13.5	26.5	--	12.1	15.3	15.1	9.5	12.6	7.0	--
Cunning-															
ham	17.3	26.2	29.2	23.4	21.3	15.5	28.3	--	11.7	12.4	14.2	11.7	9.3	8.4	--
Gillis	17.8	27.0	30.0	23.0	23.5	9.5	26.7	--	15.6	17.2	13.8	9.0	10.0	6.1	--
Hayes,A.	18.0	24.6	30.0	24.3	23.1	15.6	28.0	--	13.6	14.1	10.3	11.6	12.3	10.0	--
Jones	21.9	26.4	28.9	22.9	19.6	11.0	28.7	--	11.6	13.9	14.8	9.8	14.1	10.1	--
Morgan	17.3	27.1	29.1	23.3	21.1	11.6	28.0	--	15.5	18.2	12.8	7.8	13.9	8.0	--
Neal	19.8	26.8	30.0	23.1	22.2	10.3	27.2	--	15.8	16.4	14.8	10.2	10.3	6.9	--
Smith	20.9	26.6	29.4	23.5	21.6	10.9	27.4	--	13.3	15.1	17.3	11.0	9.5	10.1	--
Spears	19.9	26.8	30.0	23.2	23.1	11.0	26.4	--	8.0	13.9	15.6	11.0	11.2	13.1	--
Walker	16.1	26.7	30.0	23.1	22.3	17.1	27.3	--	12.3	15.5	12.6	8.2	9.5	7.5	--
Antwine	21.0	27.0	30.0	23.0	21.0	12.0	26.6	9.5	11.6	16.5	14.8	9.0	7.1	8.5	22.0
McCloud	15.6	26.9	29.3	23.4	20.3	9.7	27.6	16.6	15.3	15.6	11.7	11.4	7.4	6.9	19.4
Hartson	16.8	26.0	28.0	23.4	20.8	12.4	30.0	18.2	12.0	16.2	12.6	8.2	6.2	7.2	14.8
Benson	17.7	27.3	30.0	23.9	19.6	9.4	25.7	10.6	18.9	16.7	12.6	10.6	8.1	7.0	22.0
Boyd	18.9	28.0	30.0	23.4	20.6	8.9	26.0	10.7	10.6	15.1	14.4	14.3	8.9	8.3	22.0
Harman	16.9	27.1	30.0	23.3	21.5	11.4	25.9	12.5	10.6	17.8	14.8	10.4	8.3	7.6	18.4
Johnson	19.1	27.6	30.0	23.8	20.4	12.9	26.2	7.5	8.8	17.9	15.0	9.2	6.5	13.1	21.2
McDaniel	16.9	27.3	30.0	23.4	21.0	10.3	25.8	5.6	11.6	16.6	13.8	8.4	7.2	20.5	19.7
Morton	20.3	26.4	30.0	23.6	19.9	12.0	24.7	11.0	16.7	14.0	15.0	9.9	6.4	8.1	21.9
Ross	19.1	26.9	30.0	23.6	21.2	11.5	26.9	10.1	10.4	15.5	15.8	9.1	8.8	9.1	19.4
West	17.1	26.1	30.0	25.5	25.7	13.9	21.7	5.6	9.0	11.2	14.5	14.3	12.0	11.3	26.0
Adkins	17.7	26.6	29.4	21.6	20.0	7.8	27.5	8.5	22.4	13.4	17.0	6.8	11.7	7.4	18.8
Anderson	16.4	27.3	30.0	23.0	21.5	10.4	26.1	7.4	10.9	19.3	16.8	7.5	12.5	9.1	21.0
Britton	18.6	27.7	30.0	24.4	21.4	11.7	25.3	4.7	7.4	14.4	17.3	7.5	12.0	15.6	25.6

Appendix A-1.--Continued.

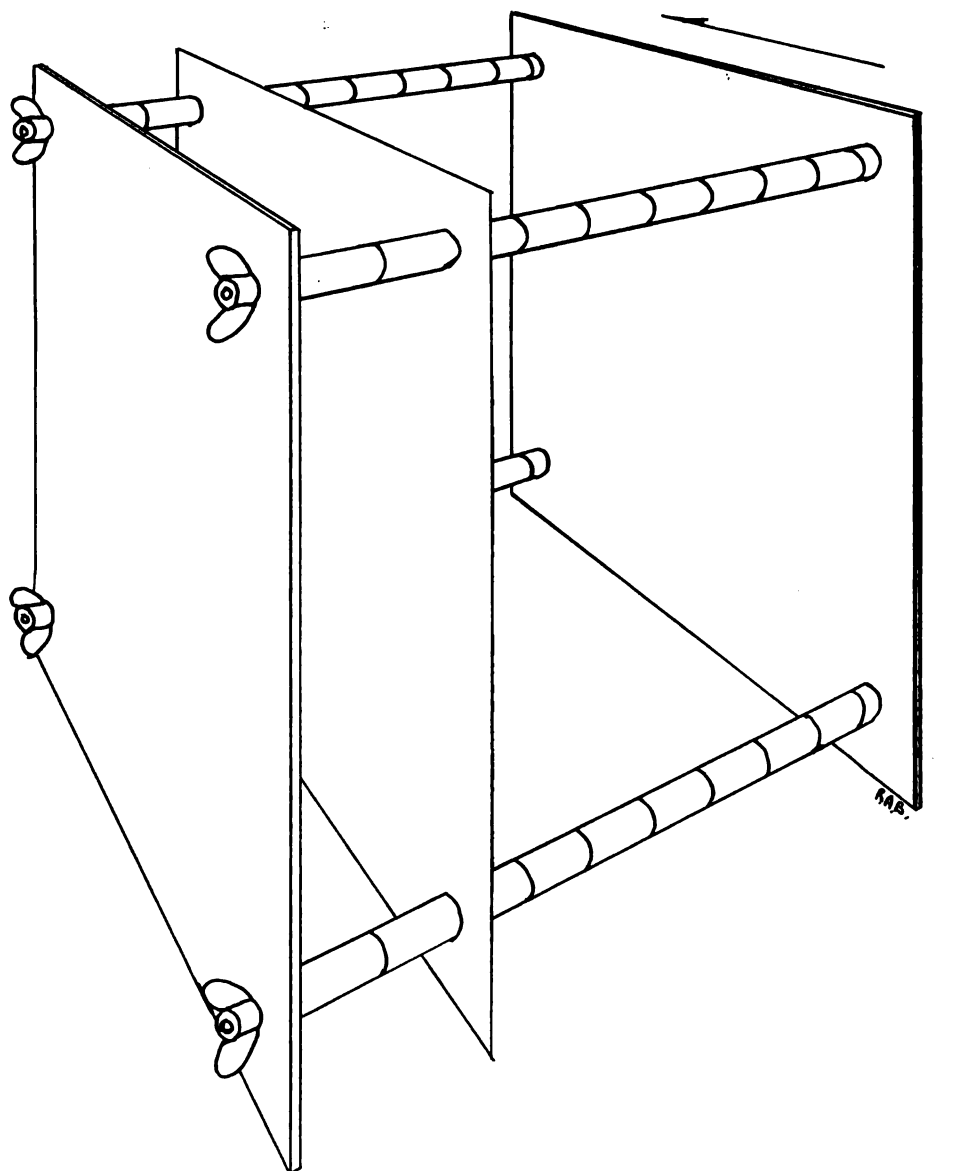
Negroes	GLU.	GLU-N.	GLY.	SER.	TAU.	THREO.	U-HIST.	L-HIST.	ORN.	ALA.	CYST.	TRYP.	PHE.	BAIB	TYR.
Brown, G.	14.8	26.5	30.0	23.4	19.0	14.0	27.5	5.9	20.6	15.7	14.2	6.6	10.6	9.2	19.2
Jones	19.3	26.7	30.0	22.6	23.2	12.4	27.3	5.1	12.3	14.5	17.8	6.8	10.9	9.2	20.2
Miller	19.4	27.6	30.0	23.5	20.0	11.5	26.4	6.0	11.4	14.6	18.6	8.3	9.3	10.9	23.5
Murphy	17.6	26.6	29.7	23.6	21.6	13.5	27.7	6.0	15.3	17.5	14.4	6.4	10.1	8.1	19.8
Reed	19.6	28.0	30.0	23.6	22.1	11.5	25.6	8.3	10.9	15.9	17.6	6.4	9.8	8.8	19.6
Reid	17.6	26.0	30.0	23.0	21.1	12.1	28.0	4.8	17.7	13.9	18.3	7.6	8.7	9.1	19.6
Snowden	19.5	27.0	30.0	22.4	21.1	10.9	27.0	4.4	16.4	17.2	16.8	6.0	10.8	8.8	18.4
Butler	19.8	27.8	30.0	24.0	21.5	12.5	26.3	4.0	11.3	14.6	16.4	6.8	12.5	11.1	18.9
Cannon	17.6	27.3	30.0	23.1	22.3	11.4	26.7	7.5	11.0	17.8	16.9	7.1	10.8	8.5	19.4
Cummings	15.5	26.1	30.0	22.5	20.0	11.0	27.4	11.4	22.4	15.8	13.4	5.0	8.6	8.5	15.5
Howard	18.1	26.7	30.0	23.1	20.8	12.3	27.1	13.9	12.6	16.4	13.4	5.1	7.5	11.2	16.8
Humphrey	15.8	27.2	30.0	24.0	20.6	9.7	26.3	10.2	17.6	17.1	14.2	5.9	10.2	9.2	17.3
McGee	17.9	26.8	30.0	23.0	20.2	10.3	27.0	12.2	10.9	16.7	15.9	5.7	9.3	10.4	18.8
McGore	16.3	27.6	30.0	23.8	19.2	11.7	25.3	11.6	11.3	19.7	17.5	5.8	10.6	7.4	17.0
Parkman	18.3	27.7	30.0	24.6	21.6	11.6	25.5	7.9	12.6	15.6	17.5	6.0	9.6	9.9	19.4
Sims	20.6	26.2	30.0	23.6	21.4	10.2	27.8	5.8	13.6	14.4	17.5	6.2	12.2	8.5	18.6
Turner	16.6	27.3	30.0	23.4	21.4	10.7	26.3	12.0	13.9	15.6	17.8	5.5	9.3	8.2	18.0
Sum	762.7	1128.6	1253.0	984.5	893.0	492.2	1122.9	275.5	561.4	660.3	640.5	358.7	419.6	392.1	612.2
Mean of Means	18.3	26.9	29.8	23.5	21.3	11.7	26.7	8.9	13.4	15.7	15.3	8.5	10.0	9.3	19.7

Appendix A-2.--Table of Means.

Whites	GLU.	GLU-N.	GLY.	SER.	TAU.	THREO.	U-HIST.	L-HIST.	ORN.	ALA.	CYST.	TRYP.	PRE.	BAIB	TYR.
Barnaby	19.8	26.0	29.1	23.8	21.8	15.4	28.9	--	14.0	14.3	11.6	9.7	12.7	7.1	--
Friend	20.9	25.1	29.8	22.9	17.9	12.1	28.2	--	14.6	12.9	17.6	10.8	11.3	6.9	--
Haynes	18.6	26.0	29.8	23.2	18.2	12.3	27.8	--	17.9	12.1	14.9	13.6	11.1	8.3	--
Howard	18.7	26.6	30.0	24.0	20.4	14.6	27.3	--	9.4	12.7	15.9	14.6	11.7	6.8	--
Liss	19.5	26.6	30.0	23.5	22.4	10.0	27.1	--	12.5	13.6	16.3	10.3	11.8	8.5	--
Lozada	20.7	26.3	30.0	22.6	23.4	13.6	25.1	--	14.9	12.1	16.4	10.8	11.6	7.1	--
Miller	17.0	25.6	28.1	24.1	20.6	15.3	29.6	--	19.8	15.0	13.2	11.5	10.6	8.3	--
Nokes	17.7	25.9	28.4	22.1	23.5	15.2	28.9	--	18.0	9.9	11.7	12.2	11.1	8.3	--
Ortisi	17.5	26.3	29.4	22.3	18.5	12.6	28.1	--	21.1	12.3	14.5	7.8	12.5	6.6	--
Payne	18.1	25.1	29.8	24.0	20.4	13.7	27.4	--	14.0	15.1	14.5	9.2	9.9	6.8	--
Roesler	19.1	25.9	29.8	23.5	22.5	11.4	27.8	--	15.0	14.1	15.2	7.5	12.2	7.7	--
Amsink	19.3	26.1	30.0	24.1	21.5	10.2	26.2	9.2	14.2	16.4	15.1	9.8	8.8	7.3	20.2
Bubel	16.0	26.6	30.0	19.6	17.6	12.6	27.3	17.6	22.0	16.6	12.6	6.3	4.3	7.6	23.3
Burcar	16.2	26.3	30.0	23.8	21.4	12.8	27.7	7.5	16.4	14.1	13.6	11.9	8.8	7.5	23.3
Denton	18.1	25.9	30.0	24.0	23.9	9.7	26.3	8.6	12.0	17.3	16.1	9.7	7.6	9.1	22.6
Huston	19.4	25.7	30.0	23.3	21.3	9.7	27.9	6.1	12.8	18.1	16.5	10.9	7.9	8.4	23.4
Jefferson	18.7	25.2	30.0	23.5	25.4	12.3	25.2	5.5	8.9	14.9	16.0	13.1	10.9	8.2	23.2
Mabie	19.1	26.2	29.9	23.6	20.8	10.3	27.0	13.9	10.4	17.0	15.2	10.1	8.0	7.1	18.9
Newsome	18.8	25.5	30.0	23.3	22.2	12.2	27.7	7.1	9.2	16.2	13.9	11.0	11.6	8.9	20.9
Peterson	17.8	27.0	30.0	23.0	20.8	11.6	27.0	4.5	17.0	16.9	15.1	11.1	8.7	6.9	19.6
Powell	19.1	26.0	30.0	23.9	19.9	10.6	27.6	11.8	11.3	18.3	13.0	9.7	11.2	6.1	21.4
Stam	19.2	27.0	30.0	23.9	24.1	12.2	24.9	6.7	11.8	13.4	17.8	10.1	7.7	8.2	20.1
Bowling	17.9	26.0	30.0	23.4	20.9	11.3	28.0	6.8	14.4	18.0	17.5	6.5	8.3	8.8	18.9
Bradley	17.5	26.0	29.3	22.3	17.9	10.5	28.5	8.4	20.2	18.1	15.0	5.4	11.0	7.4	17.5
Eldred	15.0	26.0	30.0	21.6	19.6	9.7	26.8	11.3	20.4	17.8	15.9	6.4	9.8	7.7	17.2

Appendix A.2.--Continued.

Whites	GLU.	GLU-N.	GLY.	SER.	TAU.	THREO.	U-HIST.	L-HIST.	ORN.	ALA.	CYST.	TRYP.	PHE.	BAIB	TYR.
Fulayter	20.2	25.6	30.0	22.1	23.3	10.1	28.0	7.4	13.9	14.9	17.5	6.0	10.6	8.2	18.8
Naranjo	15.6	26.2	29.8	23.2	20.3	10.2	27.2	16.5	12.6	16.1	15.5	6.5	9.3	9.8	21.8
Parker	18.9	26.2	30.0	23.6	20.9	12.3	27.8	6.8	11.3	11.8	17.6	8.7	10.6	11.4	24.3
Roeschke	15.8	26.0	30.0	22.6	21.7	8.8	28.0	13.6	19.3	14.8	14.6	5.5	8.3	9.0	17.2
Schultz	18.0	27.6	30.0	21.7	22.3	10.6	23.1	14.8	13.6	14.7	19.4	5.8	8.8	7.6	18.9
Simpson	19.9	26.6	30.0	21.2	19.2	7.3	26.9	9.7	15.4	16.0	17.4	6.4	10.0	10.9	20.1
Vanoordt	17.3	26.2	30.0	22.8	20.6	8.8	27.6	6.4	15.0	19.0	17.3	6.0	10.4	10.6	20.0
Asselin	18.9	27.6	30.0	24.0	21.1	7.6	26.0	9.2	11.7	15.0	17.5	6.0	11.9	12.2	19.6
Banta	17.4	26.6	30.0	24.0	21.6	10.9	27.4	8.8	16.2	15.1	13.9	5.4	8.4	12.3	19.6
Bartlett	19.2	25.6	30.0	23.9	19.5	12.5	27.4	4.8	12.7	16.3	18.7	5.9	10.8	10.0	21.4
Bazzy	17.8	26.6	30.0	23.2	20.6	10.5	27.1	13.4	12.3	16.9	16.7	5.4	8.6	8.9	20.2
Burten- shaw	19.0	26.1	30.0	23.3	21.4	11.5	27.6	7.8	14.3	14.4	17.4	6.4	10.9	7.9	16.9
Klinowski	18.8	26.1	30.0	23.8	20.8	14.1	27.9	11.0	14.4	14.2	15.2	6.0	9.9	6.8	17.4
Lange	17.8	26.1	30.0	23.8	21.8	9.4	27.9	5.8	15.1	16.3	16.5	6.3	12.5	8.7	20.4
Lute	18.4	26.3	30.0	23.8	21.1	12.3	27.6	4.8	19.0	15.9	14.1	5.8	10.4	8.3	17.8
Rose	18.2	26.3	30.0	23.7	20.6	11.1	27.4	8.7	11.6	19.3	16.0	5.6	11.7	8.3	14.6
Slapper- gosh	16.6	26.0	29.9	23.0	23.0	9.5	28.1	4.0	16.6	12.4	16.3	7.0	11.1	14.5	21.4
Sum	767.5	1098.6	1253.1	973.0	886.7	479.4	1147.3	278.5	617.2	640.3	656.7	354.7	425.3	357.0	620.9
Mean of Means	18.3	26.2	29.8	23.2	21.1	11.4	27.3	9.0	14.7	15.2	15.6	8.5	10.1	8.5	20.0

THE RACK

Appendix A.3.--The Chromatographic Rack

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