Heritability in Dental Caries, Certain Oral Microflora and Salivary Components[1]

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INTRODUCTION

STUDIES OF THE ETIOLOGY of dental caries have suggested that both environmental and genetic factors may be concerned in the development of dental decay. Several major genes have been identified which condition defective formation of enamel or dentin, and rampant caries usually appears in such teeth (Witkop, 1958). However, these genes are rare and probably account for only a small proportion of all individuals with carious teeth. The role of genetic factors in the commonplace variety of dental caries remains uncertain. The few twin studies of ordinary dental caries have provided equivocal conclusions (Böök and Grahnen, 1953). The present twin study was begun to learn whether the use of measures more refined than those previously employed would reveal significant heritability in the common type of tooth decay.

The voluminous literature regarding etiological agents in dental caries includes many reports of exogenous and endogenous factors which may influence the frequency of dental decay. If, as seemed likely, genetic factors do participate in regulating susceptibility to decay, then study of the heritability of individual factors possibly related to tooth decay might permit partial separation of this complex. Hence, numbers of certain oral microflora and the rate flow, pH, and amylase activity of saliva were determined in a series of twins in whom the frequency of dental caries was also being measured. This paper reports on the heritability estimates calculated for dental caries and for the salivary characteristics measured.

MATERIALS AND METHODS

Twins. The 38 like-sexed twin pairs (19 monozygotic, 19 dizygotic) for the present study were with four exceptions students attending either Michigan State University (16 pairs) or junior or senior high schools in Lansing, Michigan (18 pairs). Ages ranged from 14 to 38 years with a median of 19 years. Only two pairs were over 24 years of age. All except 5 pairs were living and eating together either at University dormitories or at home. The monozygotic series included 9 male and 10 female pairs and the dizygotic twins, 5 male and 14 female pairs.

Zygosity was determined from serologic tests using 15 antisera for groups ABO

(2), MN (2), Rh (5), Lutheran, Kell, Kidd, Duffy, P, and S. Any pair exhibiting differences in any of these tests was classified as dizygotic. In addition, any pair which showed a marked dissimilarity in eye or hair color was considered dizygotic. Anthropometric measurements (35) made by Dr. Philip J. Clark on the same twins for another purpose corroborated zygosities determined by non-serologic methods.

Dental Caries. The frequency of dental caries was determined by clinical examination employing explorer and mirror coupled with an interpretation of full mouth X-rays. To minimize exposure to radiation of both operator and subjects, X-rays were taken using one second exposures with Kodak Ultra-speed film. A lead diaphragm and aluminum filters were employed to further reduce exposure time and areas exposed. By employing these procedures, exposure time was reduced by 78 percent, the facial dose was reduced by 96 percent, and gonadal dose was reduced by 92 percent (Richards, 1958). Coded summaries of the clinical and X-ray findings were recorded on forms kindly provided by the Dental Division of the Michigan State Health Department.

Analyses of dental caries were based on the ratio of the number of decayed and filled tooth surfaces to the number of surfaces available to decay times 100. We have used the term "caries experience ratio" (CER) proposed by Horowitz, Osborne, and DeGeorge (1958) to describe this relationship. All teeth were considered to have 5 surfaces. Unerupted teeth, congenitally missing teeth, and teeth removed because of trauma or for orthodontic advantage were excluded from both numerator and denominator. Teeth extracted because of advanced caries and teeth bearing restorative full crowns were tabulated as having had 3 decayed surfaces prior to removal or restoration (Bodecker, 1939). In all tabulations, a single surface decay was considered equivalent to a single surface filling with the exception that an active caries on the mesial or distal surface of any posterior tooth was scored as a two-surface lesion because, in filling such decays, it is generally necessary to approach from the occlusal surface. This adjustment was not necessary if the tooth had an active caries or a filling on its occlusal surface because no provision was made for multiple carious foci on a single surface. All clinical examinations and interpretations of x-rays were made by the same operator (J. E. L.). The dental examinations of a given pair of twins did not always occur on the same day, but were usually given within the same week.

Microflora. Paraffin-stimulated saliva was collected from both members of each twin pair during the same morning. Subjects were instructed to deposit 15 to 20 ml. of saliva into sterile vials on first rising before brushing teeth or consuming any food or liquid. Saliva specimens were refrigerated immediately after collection. They were plated as soon as possible, usually on the afternoon of the day they were collected. The interval between collection and plating of specimens from a given twin pair was always the same for both members of the pair. Ten fold dilutions were made up in 0.05 % yeast extract water. One-tenth ml. quantities of the appropriate dilution (that dilution yielding a readily countable plate) were pour plated using the following media: tryptone glucose extract (TGE) containing 4 percent skim milk for the growth and subsequent count of miscellaneous bacteria; Rogosa SL agar, modified slightly by substituting for half of the glucose equal amounts of sucrose and arabinose for

growth of lactobacilli; crystal violet azide blood agar[3] (CVA) for culture of strepto-cocci. All plates were incubated aerobically at 37°C. The TGE and CVA plates were incubated for 3 days and the Rogosa SL agar plates were incubated for 4 days.

Rate of flow, pH, and amylase activity of saliva. The work of many prior investi-gators (see review by Cox, 1952) and preliminary work of the present study indicated that the rate of flow, pH, and amylase activity of saliva are influenced by a large number of environmental and physiological factors. Intrapair diurnal variation and the influence of mealtime were controlled to some extent by invariably collecting specimens simultaneously from both members of each twin pair. However, there was considerable interpair variability in time of day of saliva collections because of variations in the time when both members of each pair were free of other commit-ments. With the exception of 3 monozygotic pairs, both members of each pair had eaten at approximately the same time prior to collection. As mentioned above, all but five pairs were living and eating together either at home or at University dormi-tories.

In addition to diurnal physiological variations, whole saliva is heterogeneous because of anatomical, histological, and physiological differences between the three paired glands which produce it. This heterogeneity could best be controlled by collecting the secretions of each pair separately. However, the methods presently available for separating the secretions of the submaxillary from those of the sub-lingual glands involve either cannulation of Wharton's ducts or preparation of indi-vidual casts of the mouth. Since neither of these procedures seemed practicable, a mixture of sublingual and submaxillary secretions were collected from the floor of the mouth. At the same time, parotid secretions were collected through the use of plastic discs designed by Curby (1953) for this purpose.

Following an explanation of the technique, subjects were instructed to clear their mouths of saliva. Curby discs (held in place by vacuum) were then centered over Stenson's ducts. Subjects were reminded not to swallow during the period of col-lection. Within a minute or two saliva appeared in collecting tubules if the subjects were copious salivators. From time to time a tubule was passed over the floor of the mouth to collect accumulated sublingual-submaxillary (S & S) saliva. Salivas were collected in graduated centrifuge tubes to measure volume. The duration of collection was determined by the time needed to collect sufficient parotid saliva (about 0.5 cc.) for test procedures and varied from 7 to 52 minutes. Fastening of Curby discs and the passing of a tubule over the floor of the mouth provided stimuli for salivation.

The collection of saliva was not without problems. In a few subjects Stenson's ducts were located too high in the buccal pouch to permit easy attachment of the Curby devices. It was of incidental interest that a marked similarity in the position of Stenson's ducts and in the anatomy of the papillae surrounding them was noted in monozygotic twins but considerable intrapair variability was observed in the same trait among dizygotic twins. The following standard was adopted for checking on the attachment of the Curby devices: if saliva had not appeared in collecting tubules

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[3] This medium comprises: brain heart infusion agar (Difco), 52 gm; agar, 5 gm; crystal violet, 1 ml. of a 0.2 per cent aqueous solution; sodium azide, 100 mg; and distilled water, q. s. 1 liter. Defi-brinated sheep's blood (4 ml. per 150 ml. media) is added to cool, melted agar prior to use.

within 5 minutes, it was presumed that the devices were improperly placed and they were removed and replaced. More often than not, the devices had been properly placed, and failure to observe saliva was due to the subject's slow rate of flow. Some individuals swallowed S & S saliva in spite of reminders.

Rate of flow was estimated from recorded volume and duration of collection. The pH of specimens was measured with a potentiometer immediately following collection. Amylase activity was measured in duplicate using a modification of the method of Myers, Free, and Rosinski (1944) for determination of serum amylase. Amylase determinations were usually completed within 12 hours following collection and specimens were refrigerated at 4°C until tested.

THE DATA

Caries. All mean intrapair variances were computed from the formula: $V = \Sigma d^2/2n$, in which V is the variance, d is the intrapair difference, and n is the number of pairs. The calculated ratio between the variance in dizygotic twins and the variance in monozygotic twins (F test) was considered to support the hypothesis that genetic factors significantly influence the trait being studied if the probability (P) of getting a ratio as large or larger by chance was less than .05. A comparison of mean intrapair variances based on the CER for all teeth is presented in Table 1. Corresponding data for the separate quadrants of the mouth shown by many investigators to have different caries incidence rates are also given. Anterior teeth include the centrals, laterals, and cuspids, and posterior teeth include the first and second bicuspids, and the first, second, and third molars. These data indicate the presence of genetic factors regulating susceptibility to dental caries, confirming similar findings by Horowitz, Osborne, and DeGeorge (1958). Comparisons of the

TABLE 1. MEAN INTRAPAIR VARIANCE ANALYSES OF CARIES EXPERIENCE RATIOS (CER)* FOR ALL TEETH AND FOR FOUR QUADRANTS OF THE MOUTH (DZ; DIZYGOTIC, MZ; MONOZYGOTIC)

Group		Present Study				Horowitz, Osborne, DeGeorge (1958)			
	Zygosity	Number Pairs	Variance	F	P	Number Pairs	Variance	F	P
All Teeth	DZ	19	130	6.84	<.001	13	79	2.72	<.025
	MZ	19	19			22	29		
Ant. Max	DZ	19	152	4.22	<.005	17	59	1.59	>.10
	MZ	19	36			27	37		
Ant. Mand.	DZ	19	39.5	4.82	<.001	19	45.2	5.58	<.001
	MZ	19	8.2			30	8.1		
Post. Max.	DZ	19	290	6.04	<.001	17	248	2.64	>.01
	MZ	19	48			26	94		
Post. Mand.	DZ	19	221	5.02	<.001	15	156	2.48	.025
	MZ	19	44			25	63		
Active Caries	DZ	19	26.0	5.31	<.001				
	MZ	19	4.9						

$$*CER = \frac{\text{Number of decayed or filled surfaces}}{\text{Number of surfaces available for decay}} \times 100$$

present study with earlier twin studies of dental caries are meaningless because earlier studies used the whole tooth as the unit of measure whereas Horowitz *et al.* and the present authors have used the surface as the unit.

Corresponding data (variances rounded) from the study of Horowitz *et al.* are also included in Table 1. It is of interest to note that the closest correspondence between the two bodies of data is found in the variances calculated for the anterior mandibular quadrant. Though this similarity may be no more than coincidence, it may also reflect the well known fact that teeth in that quadrant are more resistant to decay than those in any other quadrant of the mouth. Their resistance is thought to be due to their anatomy (self-cleansing, absence of pits and fissures) and to their location within the mouth which leads to their being constantly bathed in the buffered, neutral to slightly alkaline S & S saliva.

A second point of interest is the fact that with one exception, the variance ratios calculated were higher in the present study than the corresponding ratios in the study of Horowitz *et al.* Ethnic and environmental differences between the two twin samples in addition to test and sampling errors may account in part for the differences noted. The agreement between findings on anterior mandibular teeth suggests that these sources of differences may not be major factors. A more likely explanation may be developed from the fact that the present twin series is a younger group. Twins in the study of Horowitz *et al.* ranged from 18 to 55 years of age with a median at 24 years. Only 2 pairs in the present series were over 24 years of age. The arithmetic bases for the larger variance ratios of the present study are that the variances for monozygotic twins in the younger series are either about the same as or considerably smaller than those of monozygotic twins in the older series. In addition, the variances for dizygotic twins in the present study are either about the same as or considerably larger than the corresponding data from Horowitz *et al.* In general, it might be anticipated that intrapair differences in caries experience of twin pairs would tend to increase with the increasing environmental diversity accompanying aging, at least up to the point where one member of a pair has at least one caries per surface. At the same time, if it is assumed that the intrapair correlation in ages at eruption of the teeth is higher in monozygotic than in dizygotic twins, then differences in length of exposure to risk of decay make a greater contribution to variability within dizygotic than within monozygotic pairs. However, intrapair differences in ages at eruption would tend to become less significant as twin pairs age, and would become small relative to the total length of exposure in twins over 30 years of age. Hatton (1955) reported that the intrapair variance in ages at eruption of deciduous teeth was higher in dizygotic than in monozygotic twins. Hence, the larger variance ratios of the present study may have resulted from the inclusion of intrapair variability in ages at eruption of teeth, a source which may have made an insignificant contribution to the total variability in the older series.

Data on active caries were included in all CER tabulations presented in Table 1, but, for reasons given below, were also considered apart from filled caries. The number of active caries i.e., carious lesions which have not been filled, depends on the number of surfaces available to decay, the caries attack rate, and the frequency and quality of dental care received. In addition, the number of active caries observed depends

on methods used to detect such caries. It had been anticipated that twins in the present study, particularly those in the University group, would have had better than average dental care, and, hence, that the frequency of active caries might not show significant heritability. Knutson and Klein (1938) suggested that tooth mortality, i.e., the number of teeth removed because of advanced caries, is a reasonable criterion of the level of dental health care. The fact that only 22 of the 76 individuals studied had lost any teeth due to advanced caries and that the average tooth mortality was 0.5 tooth per individual indicated that the twins in the present study had received superior dental care. In spite of a high level of care, there were only 3 persons found to be free of active caries and the average number of carious surfaces in the total sample was 8.4. The heritability suggested by the data on active caries (Table 1) may reflect the effects of both biological factors responsible for caries and the psychic and socio-economic factors which encourage (or discourage) frequent visits to the dentist. That we have measured behavioral as well as more purely physiological variation is suggested by the total correlation coefficient (.16), calculated from all individuals, between the number of active and the number of filled carious surfaces which did not differ significantly from zero.

Many investigators (see Neel and Schull, 1955) have pointed out some of the difficulties in interpreting the heritability estimates often accompanying twin studies. On the other hand, Clark (1956) pointed out that such estimates are of interest from the evolutionary point of view as indices of the susceptibility of characters to genetic change. The h^2 values in Table 2 were calculated from our data and from those of Horowitz *et al.* using the formula:

$$h^2 = \frac{V_{DZ} - V_{MZ}}{V_{DZ}},$$

in which h^2 is the proportion of variability in dizygotic twins attributable to genetic variation and V_{DZ} and V_{MZ} are the respective intrapair variances of dizygotic and monozygotic twin pairs. Since nearly all persons are edentulous at birth and many end life in the same condition, one would expect some age between eruption of the first tooth and loss of the last to yield a maximum h^2, and these data suggest that the maximum occurs no later than the mean age in the present series (18.7 years). Though differences in the h^2 values in the two studies are completely dependent on the variance differences already discussed above, they are included because they may be illustrating an age effect. The h^2 values for all traits considered are included without comment in Tables 3 and 4.

Microflora. A summary of computations based on logarithmic transformations of bacterial counts is presented in Table 3. Ever since Miller's series of classic papers (see Cox, 1952) on the pathogenesis of dental caries in which he assigned oral microflora a significant role as acid formers, investigators have sought to explain the relationship between species and numbers of microorganisms in the mouth and the frequency of caries. Both streptococci and lactobacilli have been suggested as important etiological agents in dental caries both on the basis of their acidogenic power and on the basis of the correlations observed between number of organisms and number of decayed, missing or filled teeth (e.g., Sullivan and Storvick, 1950 and

TABLE 2. HERITABILITY ESTIMATES (h²) OF CARIES SUSCEPTIBILITY FROM PRESENT
STUDY AND FROM HOROWITZ ET AL. (1958)

Group	$h^2 \times 100$	
	Present Study	Horowitz et al.
All Teeth	85	63
Ant. Max.	76	36
Ant. Mand.	79	82
Post. Max.	84	62
Post. Mand.	80	60

TABLE 3. MEAN INTRAPAIR VARIANCE ANALYSES OF CERTAIN ORAL MICROFLORA; BASED
ON LOGARITHMIC TRANSFORMATIONS OF ORIGINAL COUNTS

Culture	Zygosity	Number Pairs	Variance	F	P	$h^2 \times 100$
Miscellaneous	DZ	17	0.057	3.35	<.01	70
Bacteria	MZ	17	0.017			
Streptococci	DZ	17	0.082	3.90	<.01	74
	MZ	17	0.021			
Lactobacilli	DZ	17	2.7	1.35	>.25	25
	MZ	17	2.0			

Jay, 1936). Whether a relationship exists between dental caries and number and kinds of oral microflora in twins in the present study will be discussed in a future paper. However, the data presented in Table 3 are of interest apart from their possible relationship to caries.

The obvious question which arises in connection with the heritability shown by both the miscellaneous organisms and streptococci is the degree of independence between the two populations. The correlation between the two groups of organisms was .62 (P < .01 if $\rho = 0$) suggesting that either the streptococci are contributing to the heritability shown by miscellaneous organisms or that both are correlated with a common factor or factors. The miscellaneous organisms were not subcultured for identification of species present so that we have no direct evidence on this point. Studies by Bibby, Volker, and Van Kesteren (1942) and Richardson and Jones (1958) indicated that streptococci were among the most numerous of all major groups of oral microflora.

The lactobacilli counts showed extreme variability (0 to 8.5 million) between individuals. Permar, Kitchin, and Robinson (1946) noted that even on replicate platings of the same specimen of saliva lactobacillus counts showed wide variations. The use of log transformations failed to yield a distribution approximating the normal. Until some method is devised for eliminating non-random count fluctuations, further attempts to determine the heritability of lactobacilli counts seem likely to fail. Of course, all of the variability may be due to environmental factors, but, if true, its non-random character still demands some explanation.

Rate of flow, pH, and amylase activity. The pertinent statistics regarding rate of flow, pH, and amylase activity are presented in Table 4. Rate of flow was based on

cc. of saliva secreted in 15 minutes, proportional adjustments being made for those specimens collected over a longer or shorter period of time. Analyses of rate of flow were based on logarithmic transformations of raw data because of the non-normal character of their distribution. If rate of flow is related to caries frequency as has been suggested by investigations on experimental animals and humans (see review by Cox, 1952), the heritability shown by rate of flow is in part responsible for the heritability shown by dental caries, and a small beginning has been made in isolating the genetic variables which contribute to the decay process.

Data from the secretions of the parotid are not independent of the corresponding data from S & S secretions. The lack of correspondence between findings from the two types of specimens, particularly with regard to pH determinations, is probably due to technical difficulties in the collection of S & S salivas. Parotid secretions were extremely unlikely to be contaminated by S & S saliva, but the converse was not always true. Specimens of S & S saliva known to be contaminated with parotid saliva were eliminated, but minimal contamination may well have gone undetected. Further, S & S secretions contain the oral microflora whereas no organisms were detected in parotid specimens plated out during preliminary work. The presence of small amounts of parotid saliva and of the oral microflora in S & S saliva would tend to lower the pH of these specimens.

A study of titratable alkalinity and acidity of whole saliva by Turner, Bell, Scribner, and Meyer (1953) is the only genetic study known to us which is directly related to any of the three variables considered in Table 4. They found that

TABLE 4. MEAN INTRAPAIR VARIANCE ANALYSES OF RATE OF FLOW, PH, AND AMYLASE ACTIVITY OF PAROTID AND SUBLINGUAL-SUBMAXILLARY (S & S) SALIVA SPECIMENS

	Characteristic	Zygosity	Number Pairs	Variance	F	P	$h^2 \times 100$
Parotid	Rate of Flow	DZ	18	0.217	4.82	< .005	79
		MZ	16	0.045			
	pH	DZ	16	0.284	6.60	< .001	85
		MZ	17	0.043			
	Amylase Activity	DZ	19	184	4.60	< .005	78
		MZ	19	40			
S & S	Rate of Flow	DZ	18	0.089	2.78	< .025	78
		MZ	17	0.032			
	pH	DZ	17	0.10	—	—	—
		MZ	17	0.13			
	Amylase Activity	DZ	18	10.82	12.30	< .001	92
		MZ	17	0.88			
Par.-S & S Amylase Activity Ratio		DZ	18	73	4.87	< .005	79
		MZ	17	15			
Whole Stim. Sal. pH		DZ	12	0.080	3.08	< .10	68
		MZ	12	0.026			

the variance of titratable acidity of salivas from 18 pairs of monozygotic twins was significantly smaller than the variance in 18 pairs of siblings. In the present study, the heritability shown for pH probably contributes to the total heritability in dental caries if the correlation coefficient of .196 found by Sullivan and Storvick (1950) between DMF (decayed, missing, filled) teeth and salivary pH described a causal relationship.

The explanation for the amylase activity of S & S salivas exhibiting higher heritability than corresponding data from parotid saliva is unknown to us. Random effects and the fact that the S & S activity of one dizygotic and two monozygotic pairs was not measured offer possible explanations. Further, the fact that in our subjects the amylase activity of parotid saliva was 5-fold higher on the average than S & S activity may be in part responsible. However, one would expect random errors to affect specimens with less activity more than those showing greater activity. Though the rate of flow, pH, and amylase activity of saliva appear to be appreciably influenced by genetic factors, the heritability shown by one or more of these variables may be spurious because the components are not independent of each other. It may be possible to compute corrected heritability estimates after the interrelationships between these three variables have been examined.

During preliminary work it had been noted that the ratio of parotid to S & S amylase activity appeared to be somewhat fixed for each individual regardless of the absolute differences which existed between specimens collected at different times of day or on different days. This impression was supported by a comparison computed for monozygotic and dizygotic twins as shown in Table 4. It is possible that the heritability of ratio differences suggested by these data may not be independent of the intrapair differences in either parotid or S & S amylase activity, i.e., that all three sets of differences reflect the same genetic background. However the correlation coefficients between intrapair ratio differences and parotid differences were .33 ± .24 and .02 ± .25 for dizygotic and monozygotic pairs, respectively, and between ratio differences and S & S differences .34 ± .24 and −.24 ± .25 for dizygotic and monozygotic pairs respectively. These coefficients suggest that whatever genetic factors may be involved in determining intrapair parotid–S & S ratio differences, they are not principally those affecting intrapair differences in either parotid or S & S amylase activity. Genetically influenced differences between the parotid and S & S glands in their relative size, their relative proportion of cells producing amylase, or in their response to nervous stimuli are obvious possible explanations for the heritability shown by parotid–S & S ratios.

DISCUSSION

Throughout the present paper, traits have been described as exhibiting "heritability" whenever the intrapair variance in dizygotic twins has significantly exceeded the intrapair variance in monozygotic twins. This definition of heritability is subject to certain reservations and may not always reflect purely genetic differences. It is entirely probable that parents may tend to treat monozygotic twins more nearly alike than dizygotic twins. The estimates of heritability are based on the assumption that the average intrapair differences in environment are the same for monozygotic

and dizygotic twin pairs. Any significant tendency for greater similarity of environment in monozygotic pairs as contrasted with dizygotic pairs would tend to increase the apparent significance of the heritability estimate.

The twin study method is also limited by the fact that we can gain no information concerning the number of gene pairs that may influence a particular trait. Similarly, this method will not permit separation of the effects of a gene that may influence more than a single trait. In this study, for example, salivary rate of flow, pH, and amylase activity have been treated as if they were distinct and independent traits in both parotid and S & S salivas. Analyses of the data indicate that each of these three variables in parotid saliva is correlated with the corresponding variable in S & S saliva, and further that all three variables are correlated with each other. We have no way of knowing whether these correlations represent multiple effects of a single gene, or interactions of multiple genes.

Finally, the genetic factors whose existence is indicated by the heritability estimates may have primary actions far removed from the attributes that have been measured. For example, if there are genes which influence dietary preference for carbohydrates, the high carbohydrate diet might in turn affect amylase activity.

In the light of the limitations and qualifications that must be placed on the twin study method in general, we are reluctant to consider the heritability estimates obtained in this study as more than crude approximations. However, twin studies have certain obvious advantages and can be particularly valuable when they serve as preludes to more rigorous genetic analyses.

SUMMARY

The purpose of the present study was to determine whether genetic factors influence the frequency of dental caries, the rate of flow, pH, and amylase activity of saliva, and the number of microorganisms in the mouth through a study of these attributes in like-sexed twin pairs. It has been shown that the mean intrapair variances of the CER (caries experience ratio) of dizygotic twins exceeded those of monozygotic twins whether one considered the entire mouth or the dentition by quadrants.

Oral microflora were cultured on three media, and significant heritability was calculated for miscellaneous organisms and for streptococci. The degree of independence between these two cultures is presently unknown. The influence of genetic factors on oral lactobacilli could not be determined because of non-random variations in lactobacilli counts.

Similarly, variance ratios computed for rate of flow, pH, and amylase activity of both parotid saliva and a mixture of sublingual and submaxillary saliva specimens suggested that genetic factors influence these variables to a significant extent. The only salivary component measured which did not show significant heritability was the pH of S & S saliva. This exception was probably the result of technical difficulties.

The generality of the findings on salivary components has yet to be confirmed. It appears that several salivary components thought to be related to caries frequency may be regulated to an appreciable extent by genetic factors, and hence, the heritability shown by dental caries may have been partitioned to some extent. The relationship between each of the salivary components studied and dental caries will

appear in a subsequent paper (Goodman, Luke, Rosen, and Hackel, in manuscript). Plans are being made to extend these studies to family groups.

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