TABLE II. Liver Lipids (mg/100 g Wet Tissue) in Rats after Feeding with Various Diets during a 4 Month Period. Mean, S.D. and range.

Diet	No. of rats	Liver wt (g)	Total cholesterol	Phospholipids	C/P
Normal control	20	11.4 ± 1.8 8.3-14.1	252 ± 42 202-348	2849 ± 252 2440-3350	.09 ± .02 .07-.16
Neomycin	15	10.1 ± 1.5 7.6-12.1	295 ± 45 225-321	2912 ± 218 2400-3398	.09 ± .03 .08-.14
Neomycin + cholesterol	18	10.2 ± 1.3 7.6-12.8	1660 ± 350 1026-2333	3017 ± 388 2300-3725	.53 ± .14 .36-.89
Cholesterol	20	15.0 ± 2.7 13.5-16.1	1676 ± 266 1260-2043	2880 ± 413 2020-3675	.61 ± .13 .38-.95

did not influence lipid levels in serum and liver under the described experimental conditions.

It was believed for a long time that rats were resistant to induction of hyperlipemia and hypercholesterolemia. Recently, however, this opinion has been modified(8) and these animals are now frequently used for study of various dietary factors inducing atherosclerosis.

In our experiment no significant hypercholesterolemia was observed, despite the prolonged ingestion of cholesterol rich diet by rats.

The only significant change in this study was the rise of total cholesterol in livers of animals given cholesterol rich diets, with and without neomycin.

Summary. 1) Because ingestion of neomycin results in hypocholesterolemia in human subjects, the effect of this antibiotic on lipids of serum and liver has been studied in rats. 2)Basal control diet or cholesterol rich diet, alone or added with 0.2% neomycin, has been fed to rats for 4 months. This prolonged administration of neomycin to rats was not associated with any significant changes in serum or liver levels of cholesterol and phospholipids.

The author is indebted to Upjohn Co. of Canada for generous supply of Mycifradin Sulphate.

1. Samuel, P., Steiner, A., PROC. SOC. EXP. BIOL. AND MED., 1959, v100, 193.

2. Waksman, S. A., Lechevalier, H. A., *Science,* 1949, v109, 305.

3. Schweinburg, F. B., Jacob, S., Rutenburg, A. M., PROC. SOC. EXP. BIOL. AND MED., 1952, v79, 335.

4. Dickson, R. S., *Vet. Med.,* 1955, v50, 143.

5. Kowalewski, K., Couves, C. M., Lang, A., *Acta Endocrinol.,* 1959, v30, 268.

6. Abell, L. L., Levy, B. B., Brodie, B. B., Kendell, F. E., *J. Biol. Chem.,* 1952, v195, 155.

7. Zilversmit, D. B., Davis, A. K., *J. Lab. and Clin. Med.,* 1950, v35, 155.

8. Wilgram, S. F., PROC. SOC. EXP. BIOL. AND MED., 1958, v99, 499.

Received August 28, 1959. P.S.E.B.M., 1959, v102.

Comparison of Glutamic-Oxalacetic and Glutamic-Pyruvic Transaminase Concentrations in Human Saliva and Serum.* (25281)

SAMUEL DREIZEN, ROBERT E. STONE, JO G. DREIZEN AND TOM D. SPIES
Dept. of Nutrition and Metabolism, Northwestern University Medical School

Since development of practical methods for determination of glutamic-oxalacetic and glutamic-pyruvic transaminases, measurements of serum and cerebrospinal fluid concentrations of these enzymes have been used in differential diagnosis of an ever increasing list of disease states. These enzymes are present in varying amounts in all body tissues and function in the transference of amino groups from one amino acid to another. Increased

* Northwestern University studies in nutrition at Jefferson-Hillman Hospital, Birmingham, Ala. These studies supported by grants from Roche Anniversary Foundation and Pfizer Fn.

TABLE I. Comparison of GOT and GPT Content in Saliva and Serum of 81 Patients.

Group	No.	Mean Saliva	Mean Serum	σ* Saliva	σ* Serum	r†	σ_r‡	r/σ_r	Incubated saliva Mean	Incubated saliva σ
				Glutamic-oxalacetic transaminase—units/ml						
Males	33	28.7	16.3	24.4	8.6	−.066	.173	.38	2.0	4.4
Females	48	26.9	20.2	15.9	9.2	.341	.129	2.64	4.4	8.1
Total	81	27.5	18.6	20.2	9.2	.116	.110	1.06	3.4	7.0
Edentulous	39	24.9	19.0	20.9	10.1	.241	.151	1.60	3.6	7.7
Dentulous	42	30.2	18.2	19.9	8.3	−.061	.154	.40	3.3	6.2
				Glutamic pyruvic transaminase—units/ml						
Males	33	11.9	15.9	9.0	9.9	.292	.159	1.84	158.2	104.1
Females	48	12.9	16.8	10.4	10.6	.080	.140	.57	173.5	105.9
Total	81	12.5	16.4	9.9	10.2	.163	.108	1.51	167.3	107.0
Edentulous	39	9.2	17.2	7.1	11.7	.144	.157	.92	153.6	111.4
Dentulous	42	15.5	15.7	11.1	8.6	.282	.142	1.99	180.0	100.3

* σ = Stand. dev. † r = Coef. of correlation. ‡ σ_r = Stand. error of coef. of correlation.

levels in blood reflect damage to those organs or systems which are particularly rich in these enzymes. Human saliva contains detectable amounts of many enzymes found in blood(1). Existence of transaminases in saliva has recently been reported(2). The present studies were undertaken to determine levels of GOT and GPT in human saliva and to compare concentrations contained therein with those of concurrently collected samples of serum.

Materials and methods. Paraffin stimulated whole saliva and venous blood were obtained from 81 consecutive outpatients of the Nutrition Clinic, Hillman Hospital. The 81 subjects ranged from 12 to 79 years and consisted of 27 white males, 6 negro males, 39 white females and 9 negro females. Thirty-nine were edentulous and 42 had some or all of their natural teeth. Fifteen ml of saliva and 5 ml of blood were collected concurrently from each patient. Blood was centrifuged immediately following collection. Serum was drawn off and stored in refrigerator with saliva samples until ready for testing. The GOT and GPT content in units/ml was determined in 0.2 ml aliquots of each specimen of saliva and serum by the colorimetric method of Mohun and Cook(3). Readings of per cent transmittance were made in a Coleman Universal spectrophotometer set at wave length of 550 mμ. Since many bacteria contain GOT and GPT (4), 10 ml of each saliva sample were added to sterile brown screw cap 25 ml bottles containing 100 mg dextrose. The bottles were sealed, incubated and agitated in water bath maintained at 37°C for 4 hours. Ten ml of pooled serum treated in identical fashion served as control. After 4 hours, 0.2 ml of each incubated aliquot was analyzed for GOT and GPT by above method. The data were then subjected to statistical analysis.

Results. Each saliva sample contained measurable quantities of GOT in 5 to 109 units/ml. GPT was found in 72 of the 81 (88.9%) saliva samples in amounts ranging from 2 to 43 units/ml. The saliva GOT content exceeded that of GPT in 74 of the 81 (91.4%) cases. Incubation of saliva with glucose at 37°C for 4 hours resulted in each instance in a pronounced increase in GPT concentration and decrease in GOT content. In contrast, incubation of pooled serum with glucose did not materially alter the GPT level but was accompanied by a significant depression in GOT value. The serum GOT and GPT concentrations in each patient were within the generally accepted limits of normal, none exceeding 39 units/ml.

The mean GOT and GPT concentrations in serum and saliva of the 81 patients and the statistical evaluation are shown in Table I. Mean GOT levels were: serum, 18.6; saliva, 27.5; and saliva incubated with glucose, 3.4 units/ml. The corresponding mean GPT concentrations were: 16.4, 12.5, and 167.3 units/ml respectively. There was no significant correlation between the serum and saliva levels of these enzymes, the correlation coefficients (r) being 0.116 for GOT and 0.163 for GPT (Fig. 1). Neither of these values exceeded

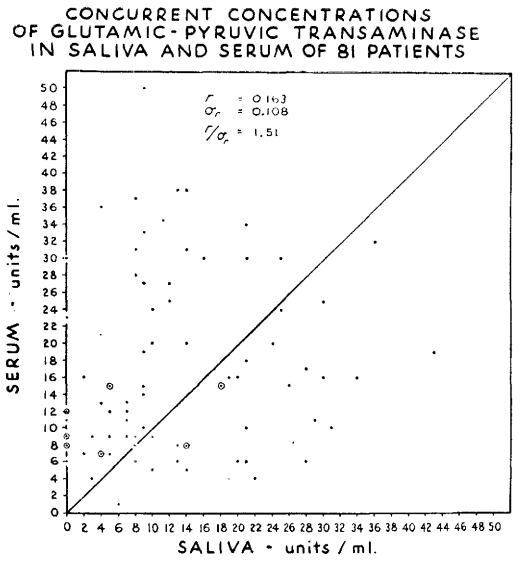

FIG. 1.

the standard error of coefficient of correlation (σ_r) by the 3 or more times required for statistical significance.

There was no appreciable sex difference in mean serum and saliva levels of GOT and GPT (Table I). The edentulous patients, as a group, had lower levels of both GOT and GPT in saliva than did the group with some or all of their natural teeth. A similar finding obtained for GPT but not for GOT when saliva was incubated with glucose. The difference between mean saliva GPT values in dentulous and edentulous patients was statistically significant, the actual difference between means being 3.07 times greater than the standard error of difference between means (σ_D). By the same formulation, the differences in mean levels of GPT in incubated saliva-glucose mixtures and of GOT in saliva and in incubated saliva-glucose mixtures were not significant.

Discussion. These studies demonstrate that GOT and GPT are common constituents of human whole saliva. In the absence of pathologic conditions associated with elevated blood transaminase concentrations, the mean GOT content of saliva exceeds that of serum and mean serum GPT content surpasses that of saliva. Lack of a significant correlation between serum and saliva values indicates that the concentration in saliva is not a direct reflection of that in blood.

The present findings suggest that the oral flora and oral tissues both contribute to transaminase content of saliva. Incubation of whole saliva with glucose, which stimulates growth of oral glycolytic microorganisms was followed in each instance by a striking elevation in the saliva GPT level and the almost complete disappearance of GOT from samples so treated. The rise occurred in aliquots from each sample of saliva including those which did not contain measurable amounts of GPT before incubation. The significantly higher saliva GPT value in the group of patients with natural teeth in contrast to the edentulous group provides evidence that the natural attrition of the supporting structures of the teeth and superimposed destructive processes affect the transaminase content of saliva. The degree to which the transaminase concentration of saliva is affected by oral and periodontal disease remains to be determined.

Summary and conclusions. Detectable quantities of GOT and GPT were found in 100% and in 89% respectively of samples of stimulated saliva from 81 patients. The saliva GOT content exceeded saliva GPT level in 91% of cases. Incubation of an aliquot of each saliva sample with glucose resulted in a pronounced increase in GPT and diminution in GOT. Comparison of saliva transaminase levels with those of concurrently collected samples of serum showed no significant correlation, thus signifying that the prime and immediate source of saliva transaminase is oral rather than systemic.

1. Chauncey, H. H., Lionetti, F., Winer, R. A., Lisanti, V. F., *J. D. Res.*, 1954, v33, 321.

2. Pellegrini, R., *Boll. Soc. ital. biol. sper.*, 1957, v33, 70.

3. Mohun, A. F., Cook, I. J. Y., *J. Clin. Path.*, 1957, v10, 394.

4. Campbell, L. L., *J. Bact.*, 1956, v71, 81.

Received August 25, 1959. P.S.E.B.M., 1959, v102.