required to lower XA excretion to minimal levels in the pregnant rat. The absence of any effect of vit B_6 dose, in the range employed, on the obstetrical performance of the various groups is not surprising since the leukocyte and plasma levels of pyridoxal phosphate in the cord blood of infants indicate that the fetus is supplied with vit B_6 at the expense of the maternal stores(10).

In order to define the vit B_6 requirement of the pregnant rat by means of erythrocyte GPT activity, it would be essential to determine the dose required to produce a plateau in the enzyme response(3). This would involve the administration of closely spaced doses of vit B_6 to establish, within a limited range, the point at which GPT ceased to respond. This approach is necessary since the absolute enzyme activities of the pregnant and nonpregnant rats may be different when their respective vit B_6 requirements have been met. Obviously, the present data are inadequate to define the vit B_6 requirement of the pregnant rat.

Summary. The response of several parameters of vit B_6 nutriture (erythrocyte GPT and GOT, hepatic GPT and GOT, and xanthurenic acid excretion following a tryptophan load) to dosages of vit B_6 ranging from 15 to 400 μg pyridoxine · HCl daily has been determined in pregnant and nonpregnant rats. All parameters except erythrocyte GPT appeared to be markedly affected by the state of pregnancy *per se*. Erythrocyte GPT was the most sensitive to the wide range of vit B_6 doses employed. Erythrocyte GPT appears to be well suited for use as a parameter for the study of vit B_6 requirements in pregnancy.

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Received September 9, 1965. P.S.E.B.M., 1965, v120.

Effect of Moderate Ingestion of Alcohol upon Serum Triglyceride Responses of Normo- and Hyperlipemic Subjects.* (30628)

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Several years ago we observed(1) that subjects having a behavior pattern (Type A) associated with increased incidence of clinical coronary heart disease(2,3) as a group clear exogenously-derived fat from their blood more slowly than a group of subjects having the converse, or Type B, behavior pattern. Having made this observation, we are pursuing studies concerning the mechanism responsible for this derangement. This cause could not reside in differences in dietary intake since the diets of the group were closely similar, both quantitatively and qualitatively, except

* Supported by Grants HE 03429 and HE 05121, Nat. Inst. Health, and the Life Insurance Medical Research Fund.

TABLE I. Effect of Alcohol Ingestion upon Pre- and Postprandial Serum Triglyceride Levels of Hyperlipemic and Normolipemic Subjects.

				Control period*							
					Serum triglyceride (mg/100 ml)				Serum triglyceride		
Subject	Age	Ht	Wt	Avg chol. (mg/100 ml)	Before meal	4 Hr after	9 Hr after	Avg chol. (mg/100 ml)	Before meal	4 Hr after	9 Hr after
				A. Hyperlipemic subjects							
Br†	43	72	195	242	244	429	243	239	139	294	129
Sw	35	73	250	220	121	207	172	223	146	282	191
Co†	37	69	174	232	90	215	70	247	77	180	62
Col	47	75	230	308	95	244	156	287	177	166	101
Ca	40	69	195	268	134	259	271	256	106	305	185
Ma†	44	72	200	250	91	229	135	256	101	241	121
Avg	41	72	207	253	129	264	174	251	128	245	132
S.E. of mean				±11.0	±22.9	±31	±27.0	±8.1	±12.8	±22.5	±18.2
				B. Normolipemic subjects							
Sol	51	69	168	182	74	168	108	197	56	137	44
Al	47	69	165	206	72	133	124	192	60	110	88
Ku	46	72	195	290	62	112	72	278	70	137	66
Li	44	72	175	232	41	82	56	243	58	148	54
Od	45	72	185	208	57	118	87	222	34	68	38
Du	53	73	214	252	46	75	—	248	82	125	63
Mi	43	70	180	262	55	117	—	280	63	141	84
Mu	37	71	197	201	56	95	59	202	64	111	45
Avg	45	71	185	229	58	113	83	233	61	122	60
S.E. of mean				±11	±3.7	±9.9	±10.2	±10.8	±4.5	±8.5	±6.1
Avg of both groups				239	88	177	129	241	90	175	79
S.E. of mean				±9.3	±8.6	±24.5	±19.4	±7.9	±10.4	±19.6	±13.2

* Avg of 2 separate tests.

† Subjects exhibiting Behavior Pattern A.

for alcohol intake. The average alcoholic intake, however, of both the male and female Type A subject was considerably greater than that of their Type B counterparts(2,3).

We therefore considered it necessary to determine whether a relatively modest intake of ethyl alcohol alters the pre- and postprandial serum triglyceride values of either normo- or hyperlipemic subjects. The results of this study, reported below, indicate that such ingestion has no discernible effect.

Methods. Six hyperlipemic and 8 normolipemic subjects were chosen from a group of 68 healthy volunteers employed as firefighters. The firemen had been studied previously after each had ingested a specially prepared test fat meal(4). Three of these hyperlipemic subjects were classified as exhibiting a relatively "coronary prone" behavior pattern (Type A) according to the interview method we recently have described(5). The remaining 3 hyperlipemic and all normolipemic subjects were classified as exhibiting a relatively "coronary immune" behavior pattern (Type B). All subjects, after historical and electrocardiographic studies had been done, appeared to be free from clinical coronary artery disease. Each of the men gave a history of regularly drinking approximately 10-20 g of alcohol per day. Thus none of them had been, or were, chronic alcoholics.

For additional control purposes approximately 2 months after the initial screening tests these 14 subjects, after fasting for 13 hours, again were given the same test fat meal containing 85 g of fat by direct analysis(6). Blood samples were obtained before, and 4 and 9 hours after ingestion of the test meal during which latter time the subjects did not eat any other fat-containing food. The preprandial sample was analyzed both for triglyceride(6) and cholesterol(7). The 2 postprandial samples were analyzed for triglyceride only.

Three months after these control values had been obtained, these subjects were recalled and the entire test was repeated, except that in addition they drank 60 g of alcohol (given as 90 proof bourbon whiskey). They drank half of this amount just before

their regular dinner and the remainder at breakfast 13 hours later, at which time they also ingested their test fat meal.

Results. The average postprandial serum triglyceride values of the group of 6 hyperlipemic subjects given alcohol 13 hours and again immediately before the ingestion of the test meal were essentially the same (Table I) as the control values observed in this group. Even their average preprandial serum triglyceride content of 128 mg/100 ml (observed 13 hours after ingestion of 30 g of alcohol and their regular dinner) was no higher than that observed during the control period. We also did not observe any difference in the responses to alcohol of the three Type A hyperlipemic subjects as compared to the Type B hyperlipemic subjects. The average preprandial serum cholesterol of the hyperlipemic subjects also remained essentially the same as that observed in the control period.

Similar to the hyperlipemic subjects, the 8 normolipemic subjects also failed to exhibit any change in their pre- and postprandial serum triglyceride values after alcohol ingestion. Their average preprandial serum cholesterol also remained unchanged.

Discussion. There are a number of studies (8-11) dealing with the lipemia-producing capacities of alcohol both in man and experimental animals. However, in all clinical studies, relatively large, intoxicating quantities of alcohol have been administered and usually to chronic alcoholics. None moreover have disclosed precisely what happens to the rate of clearing of triglyceride from the blood when normal, non-alcoholic, human subjects drink several ounces of whiskey prior to a fat-containing meal.

The results of the present study suggest that the ingestion of such relatively moderate amounts of alcohol has no effect upon the rate at which triglyceride enters or leaves the blood. This was judged by the actual concentration of fat in blood at any given instant after the fat-containing meal was consumed. The study also suggests that the relative derangement in fat clearing usually observed in most subjects with Pattern A is not due to their documented(2,3) greater consumption of alcohol. Finally this study, like an earlier one(4), indicates that the pre- and postprandial serum triglyceride responses of a given group to a standard meal are remarkably reproducible when the same group is retested even after a period of several months.

Summary. Ingestion of whiskey in an amount equivalent to, or perhaps slightly greater than that consumed for social purposes does not alter the pre- or postprandial serum triglyceride responses of either hyperlipemic or normolipemic subjects. The average hyperlipemia of a group of coronary prone individuals (Behavior Pattern A) cannot be attributed to their moderately increased consumption of alcohol.

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Received September 9, 1965. P.S.E.B.M., 1965, v120.