blank. Even in the case of normal urine eluates a positive blank is always obtained—*e.g.*, there is a measurable bluish fluorescence in samples untreated with alkali. This blank is due in part to Rayleigh scattering of the light rays by the solvent itself, and in part to traces of unknown fluorescent compounds. In pellagra, however, the value of the blank is markedly increased, reaching in well-developed cases four or five times the normal values (see Table 1). The substance in the normal subject's urine. The presence of a relatively large amount of fluorescent material in the untreated urinary eluate of the pellagrin suggests that we may be dealing with the material responsible for the photosensitivity of these patients.

We wish to emphasize the fact that the dog with black tongue behaves exactly like the patient with pellagra with respect to the excretion of these fluorescent substances. This is contrary to our earlier impres-

TABLE 1

FLUORESCENCE* OF URINARY ELUATES

Range in normal subjects	Before treatment			After 50 mgms nicotinic acid by mouth		
	Blank†	After alkali	Increase (= substance F_2)	Blank†	After alkali	Increase (= F_2)
	10–15	30–50	20–35	15–20	50–70	35–50
Pellagra: Subject W (very mild)	12	12	0	22	40	18
Subject H (moderately severe)	38	38	0	26	43	17
Subject S (moderately severe)	104	69	0	60	71	11
Subject E (very severe)	64	36	0	40	34	0

* Fluorescence is expressed in Najjar-Wood units, one unit being the fluorescence caused by 1 microgram quinine sulfate dissolved in dilute sulfuric acid.[1] The figures given represent urinary excretion during a 4-hour period preceding and immediately following the administration of a dose of 50 milligrams nicotinic acid by mouth.

† The figure given represents the actual reading of the blank minus a constant correction which has been made for the Rayleigh scattering effect caused by the solvent itself. The fluorescence as given is caused by traces of unknown substances plus that of F_1 when present.

responsible for the increased fluorescence of the blank has not been identified as yet. Studies of its fluorescent spectrum are in progress. For the present we shall designate it as F_1 and we shall designate the fluorescent substance obtained from normal urine eluates after alkali addition as F_2.

The earliest change in the urine in pellagra appears to be the disappearance of F_2. This occurs before any appreciable increase in F_1 is noticeable (patient "W"). As the disease progresses the increase in F_1 becomes more and more striking. Conversely, it would appear that the first step in the healing of a severe case is some reduction in the excess of F_1, and that subsequent to this F_2 makes its appearance. The effect of treatment with a single dose of nicotinic acid is shown in Table 1. It may be seen that in the most severe case (patient "E") treatment produced only a reduction in F_1 without the appearance of any F_2. In the less severe cases the dose of nicotinic acid employed caused the appearance of F_2.

Our present interpretation of the significance of these two fluorescent compounds in the urine is as follows: an enzyme of which nicotinic acid is a component serves normally to convert the substance F_1 (which is fluorescent regardless of the reaction of the medium) into F_2, a substance which fluoresces in alkaline but not in acid solution. In states of nicotinic acid deficiency this conversion does not take place, and as a result F_1 accumulates. It is worthy of note that in pellagra the total fluorescence (found here altogether in the blank) is often far greater than the maximum fluorescence obtainable by any procedure sion. At the time of our previous publication we had had the opportunity to study only the urine of a single black tongue dog, before and after treatment. Because of the failure of the dog to excrete F_2 after what we supposed to be adequate treatment we concluded that the dog's metabolism might be different from that of man. It is now clear, however, that the failure of that animal after treatment to exhibit F_2 in his urine was attributable to inadequate treatment; he behaved exactly like the most severe of the pellagra patients, showing only a reduction in an elevated blank as the result of therapy. The adequately treated dog and the normal dog show F_2 in the urine just as in the case of the normal human being, and the concentration of F_2 can likewise be increased in the normal dog by the administration of nicotinic acid.

We have then two criteria by which to characterize states of nicotinic acid deficiency: (1) the disappearance of a normal substance which produces fluorescence on alkalinization, and (2) the appearance of an abnormal substance, which is fluorescent without any such treatment. Both of these substances can be measured quantitatively.

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EFFECT OF ALCOHOL ON VITAMIN A CONTENT OF BLOOD IN HUMAN SUBJECTS

IN this laboratory it has been shown in dogs that ingestion of alcohol will increase the amount of vita-

min A contained in their blood.[1] Pett,[2] using his instrument for testing vitamin A deficiency visually in human subjects, has observed unaccountably short recovery times in dark adaption the day following taking of alcohol. This he considers due to an increase in blood vitamin A.

We have made a few preliminary observations on the vitamin A content of the blood in human beings before and after taking alcohol. The Carr-Price colorimetric method, modified by Clausen, was used for these determinations.

The conditions under which the alcohol was taken was the usual "social" evening drinking common in this country. The alcohol was in the form of mixed drinks taken by each subject as desired during the evening.

TABLE I

Subject	Age	Sex	Weight (lbs.)	Amount of alcohol (95%)	Evelyn photoelectric units of vitamin A per 100 Ml of serum*		
					Basal	Four hours after alcohol	Twelve hours after alcohol
A	35	M	170	135 cc	33.8	42.1	42.9
A				145 cc	47.6	50.0	46.6
B	30	M	160	162 cc	93.4	217.7	76.5
C	35	M	160	150 cc	30.5	45.3	41.7
D	31	M	170	126 cc	50.8	54.3	48.3
E	36	F	120	54 cc	40.5	42.2	40.7
F	30	M	240	216 cc	57.0	59.8	48.2
G	29	F	150	261 cc	48.4	60.1	39.5
						Two hours	
H	35	M	160	20 cc	37.8	38.7	
I	33	M	150	20 cc	60.3	62.2	

* One Evelyn Photoelectric unit of vitamin A is approximately equal to three international units of vitamin A.

Blood samples were drawn before the ingestion of alcohol, at the end of the evening (approximately four hours after the first drink) and the following morning (approximately 10 to 12 hours after the ingestion of alcohol).

In every case there was some elevation in the amount of vitamin A in the blood after taking alcohol. Although the increase in most cases was relatively slight, in subject B, whose initial vitamin A content of the blood was extremely high, the rise was also remarkably large. This subject had not been taking any vitamin A concentrates previous to the experiment. The high concentration in the blood suggests that his tissues also contained large amounts of the vitamin.

COMMENT

These findings show that the ingestion of alcohol raises the vitamin A content of the blood of man. We feel that this rise in the blood is probably due to a shift of vitamin A from other tissues of the body, notably the liver, to the blood. Further work will be necessary to determine what factors determine the degree of rise in the blood. Probably the amount of alcohol ingested and the degree of saturation of the body with vitamin A are important factors. Possibly tolerance to alcohol may also be a factor. This effect of alcohol on mobilizing vitamin A from the tissues might form the basis of a test for vitamin A storage in the body.

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[1] S. W. Clausen, *et al.*, SCIENCE, 91: 318.
[2] L. B. Pett, SCIENCE, 92: 63.

ULTRAVIOLET TRANSMISSION BY THE VITELLINE MEMBRANE OF THE HEN'S EGG

THAT the developmental behavior of chick embryos may be altered by exposure to ultraviolet radiation, even when irradiated through the shell or the shell membrane, has been reported by several workers.[1, 2, 3]

It has been shown by Sheard and Higgins[1] and Landauer[2] that the shell may transmit some radiation as short as 2,800–3,000 Å, although perhaps less than one per cent. Absorption by the albumen was not measured by them, although it constitutes an important factor from the absorption view-point.

Hinrichs[3] removed a part of the egg shell and shell membrane and exposed the blastoderm directly to the radiation, which meant that the vitelline membrane (and possibly a very thin layer of albumen) still acted as a filter to the underlying embryo. Although the transmission by the vitelline membrane was not measured, Hinrichs was convinced that it did not effectively shield the embryo from the ultraviolet radiation.

However, in any attempt to correlate quantitatively the effects produced as a function of wave-length with the absorption spectrum of the substance responsible for the behavior in question, it is essential to have numerical data on the ultraviolet transmission by this membrane. It was in connection with an investigation of this kind, to be reported elsewhere by Mr. James O. Davis, that the measurements now reported were undertaken.

An unincubated egg (New Hampshire breed) from the University of Missouri Poultry Farm was opened, and the yolk placed on a watch glass. The vitelline membrane was cut half-way around at the equator

[1] C. Sheard and G. M. Higgins, *Jour. Exp. Zool.*, 57: 205, 1930.
[2] W. Landauer, *Storrs Agric. Exper. Sta. Bull.*, No. 179. 1932.
[3] M. Hinrichs. *Jour. Exp. Zool.*, 47: 309, 1927.