THE VITAMIN A RESERVE OF THE POPULATION IN DENMARK AND NORWAY DURING THE WAR

By TORBEN K. WITH and KRISTIAN ØDEGAARD, *From the Rigshospital, Copenhagen, Medical Department A* (Professor Carl Sonne, M.D.) *and the Hygienic Institute of the University of Oslo* (Professor Axel Ström, M.D.)

(With 1 Figure in the Text)

As the clinical observations on malnutrition made during the recent war practically always point towards deficiency in several vitamins as well as deficiency in calories and protein, it is of interest to look for reliable biochemical criteria for the evaluation of the role played by the deficiency of the individual vitamins in the health state of the populations of occupied Europe. For vitamin A it is possible with great accuracy to determine the reserve of the organism, as 95% of the amount is known to be stored in the liver, and the vitamin A concentration of this organ may be determined by a relatively simple and accurate analytical method.

It would thus have been of great interest to compare the vitamin A reserves of the different European countries occupied by the Germans, and in order to render this possible, one of the authors of the present paper encouraged scientific workers in several of those countries to perform determinations of vitamin A on the livers of persons who had succumbed to accidents and other forms of sudden death. This initiative was unfortunately not taken before early in 1943, and although Prof. A. Scheunert of Leipzig and Prof. Y. Peragallo of Pavia were interested in the investigation, the plan could only be carried out in Denmark and Norway owing to the development of the war.

The result of these investigations in Denmark and Norway is given below. As the rations in Denmark during the whole war were much better than in Norway, one would expect that the material would show whether vitamin A deficiency played any prominent role in the malnutrition caused by the war. Further, our material is of interest because vitamin A determinations were made on a quite similar material from Denmark before the war (1939) (cf. With, 1940, 1941*a*).

TECHNIQUE OF ANALYSIS

The analytical technique is described in detail by With (1940, 1944, 1946). 10–20 small pieces from different regions of the liver, together weighing about 20 g., most of them from the right liver lobe, were saponified. Then extraction with peroxide-free ether, washing with distilled water, drying by means of anhydrous sodium sulphate and evaporation followed. The residue was dissolved in redistilled chloroform, and the Carr-Price reaction was carried out and measured in the Pulfrich step photometer with the spectral filter S 61.

It is to be emphasized that measurements carried out by Jensen & With (1939) on a greater number of livers showed that the extinction of the Carr-Price reaction with the spectral filter S 61 of the Pulfrich photometer and the extinction at 328 mμ (determined by spectrography) varied parallel to each other—i.e. they showed a constant proportion—for which reason Carr-Price measurements on liver extracts are to be regarded as equal to spectrographic determinations (cf. With, 1946).

The factor for conversion of Carr-Price measurements to expressions in i.u. has been much discussed (With, 1940, 1941*b*). We have here used a factor calculated on the basis of the measurements of Jensen & With mentioned above and the internationally accepted conversion factor for spectrographical determinations ($E^{1\%}_{1\,cm.} = 1$ corresponds to 1600 i.u./g.); this international factor has, by the way, also been subject to much criticism (With, 1940), but, nevertheless, we have thought it most convenient to adhere to it. As Jensen & With found the value 2·6–2·7 for the proportion Carr-Price extinction (S 61) divided by 328 mμ extinction, we may conclude that $E^{1\%}_{1\,cm.} = 1$ (Carr-Price reaction, S 61) corresponds to 1600 divided by 2·6–2·7, which gives *c.* 600 i.u./g. This factor—$E^{1\%}_{1\,cm.} = 1$, corresponds to *c.* 600 i.u./g.—for the Carr-Price reaction, which, of course, is to be regarded with the same reservations as the international factor for conversion of spectrographic readings, is used for the conversion of our readings to i.u. It is to be remembered that the Carr-Price extinctions are to be calculated for the mixture of test solution and reagent.

The factor found by Jensen & With has been confirmed by measurements on human liver by the Swedish authors Engel *et al.* (1942) who found the value 2·77 corresponding to a conversion factor of 580 i.u./g. for $E^{1\%}_{1\,cm.} = 1$. Further, the factor was confirmed by spectrographic measurement on one of our livers from Oslo.

https://doi.org/10.1017/S002217240001370X Published online by Cambridge University Press

To co-ordinate our analyses ten of the livers from Oslo (nos. 2–11) were sent to Copenhagen in salted condition; 10 g. of liver were weighed off in Oslo and taken into analysis after the salt had been removed with water. The analyses performed in Copenhagen on the salted specimens showed an average of 4·2 % lower values than the analyses in Oslo before the livers were salted. The loss of *c.* 5 % can naturally be ascribed to the salting and the duration of the voyage from Oslo to Copenhagen (about 3 weeks), as control analyses made in Copenhagen on livers before and after they had been salted for 3 weeks also showed a loss of *c.* 5 %. It may therefore be concluded that the analytical technique in Copenhagen and Oslo yielded identical results.

MATERIAL

In Copenhagen forty livers were analysed in the period March–November 1943 and in Oslo fifty livers in the period October 1943–August 1944. The distribution according to sex and age is seen from the tables.

All the livers from Copenhagen were from the University Institute of Forensic Medicine.* The record numbers of the Institute are given in Table 1, together with the cause of death as verified by autopsy. The livers from Oslo were partly from the pathological institution of the municipal hospital of Oslo (Ullevål Sykehus)†, in the table marked U and partly from the Forensic Institute of the University of Oslo†—marked Rm.—the record numbers of the institutions, besides the causes of death as verified by autopsy are given in Table 2.

All the livers used were from persons who died a sudden death, most of them from accidents or poisoning, but some of them from 'natural' causes (cf. Tables 1 and 2). These 'natural' causes of death were diseases which did not make the persons unfit to work, and the information obtained concerning the case histories as well as the autopsy findings showed that the diseases in question were not liable to have influenced the eating habits or the absorption power of the persons in question and consequently not their liver reserve of vitamin A.

In Tables 1 and 2 the cases are divided into three groups, accidental death, poisoning, and sudden death from disease, and the average values and medians for each of these groups are given. In both the Copenhagen and Oslo materials the groups, sudden death from disease, show higher values than the two other groups, a finding supporting our view that our cases in this group are comparable to cases of accidental death and poisoning with regard to the vitamin A reserve. In the diagram we have therefore compared the entire Copenhagen and Oslo materials without subdivision into groups.

* We offer our sincerest thanks to Prof. Knud Sand, M.D., for the opportunity to use the material.

† We offer our sincerest thanks to Prosector E. Hval, M.D., and Prof. G. Waaler, M.D., for the opportunity to use their material.

DISCUSSION

The materials from Copenhagen and Oslo should be quite comparable as both are from the capitals of their respective countries, but they cannot be regarded as representative for their countries as a whole, as presumably the rural population and the population in the coastal towns and villages must have had better opportunity to obtain animal and vegetable sources of vitamin A and fat fish respectively than the population of cities.

The opportunity of the population of Oslo and its immediate surroundings to obtain vitamin A has unquestionably been much smaller than was the case in Copenhagen in the periods in question and, by the way, during the whole war. In Denmark the only vitamin A-containing article of food which was rationed was butter; milk, cheese, eggs, fat fish as well as carrots and other green vegetables were obtainable—some of them at a moderate cost—all through the war. So it was possible to obtain 2000–3000 i.u. of vitamin A daily at a moderate cost in Copenhagen. In Norway, on the contrary, the rationing was much more severe and the rations much smaller; whole milk was only allowed to children, to women during pregnancy and lactation and to sick persons; if eggs were obtainable they were given to pregnant women and the sick; while the butter ration in Denmark was 43 g./day during the period in question it was only 30 g. in Norway, and here the 'butter' was often given as margarine, which most often was not vitaminized. Further, the possibilities of obtaining vitamin A outside the rationing were strictly limited in Oslo—and this was the case for both animal and vegetable sources of vitamin A. Most of the vitamin A consumed by the population of Oslo during the war was, however, derived from animal sources, according to investigations carried out in the Hygienic University Institute of Oslo in 1942. For further details, the reader is referred to a survey of the Norwegian food situation during the war on the basis of investigations in Bergen in 1943 given by Notevarp (1945).

In Fig. 1 a schematic comparison of the Copenhagen and Oslo materials is carried out, and a material of exactly the same origin collected and analysed in Copenhagen in 1939 (With, 1940, 1941*a*) is included. The 1939 material includes seventeen cases of accidents and intoxication and five cases of sudden death from 'natural' causes; information about the single cases is to be found in the experimental protocols by With (1940).

Table 1. *Vitamin A content of the liver in forty persons from Copenhagen and surroundings in the period March–November* 1943

No.	Sex	Age	Cause of death (autopsy diagnosis)		Vitamin A (i.u./g.)
			(1) Accidental death		
D 42/43	M.	38	Head injury		146
D 67/43	M.	37	Head injury		122
D 79/43	M.	55	Fracture of the skull		100
D 80/43	M.	29	Shot wound		139
E 19/43	M.	14	Head injury		254
D 93/43	F.	32	Fracture of the skull		97
D 146/43	M.	38	Diving accident		173
D 156/43	F.	2	Traumatic subarachnoidal haemorrhage		266
D 157/43	M.	29	Shot wound		262
D 167/43	M.	26	Shot wound		80
D 169/43	M.	20	Shot wound		206
D 170/43	M.	33	Shot wound		152
				Average	166·4
				'Median'*	152·5
			(2) Poisoning		
D 44/43	F.	23	Barbiturate poisoning		111
D 58/43	M.	45	Barbiturate poisoning		65
E 16/43	M.	47	Cantharidine poisoning		334
D 94/43	F.	46	Carbon monoxide poisoning		166
D 106/43	M.	48	Barbiturate poisoning		106
D 127/43	M.	43	Carbon monoxide poisoning		139
E 33/43	F.	57	Carbon monoxide poisoning		876
D 166/43	M.	50	Barbiturate poisoning		28
D 186/43	F.	24	Carbon monoxide poisoning		150
				Average	219·4
				'Median'*	133·2
			(3) Sudden death from disease		
D 43/43	M.	43	Coronary thrombosis		207
D 45/43	M.	48	Subarachnoidal haemorrhage		141
D 48/43	M.	56	Coronary thrombosis		226
D 49/43	M.	52	Coronary thrombosis		139
D 51/43	M.	55	Coronary thrombosis		175
D 53/43	M.	50	Coronary thrombosis		265
D 65/43	M.	65	Myocardial fibrosis		65
E 17/43	F.	29	Acute pancreatitis; pregnancy		265
E 18/43	F.	71	Myocardial fibrosis		138
D 87/43	F.	63	Coronary thrombosis		112
D 91/43	F.	49	Cerebral haemorrhage (nephrosclerosis)		331
D 99/43	M.	54	Luetic aortitis		37
D 101/43	F.	49	Cancer of the uterus		88
D 147/43	F.	60	Pulmonary embolism		696
D 151/43	M.	57	Incarcerated hernia		279
D 155/43	M.	53	Myocardial fibrosis		6
E 34/43	M.	59	Pulmonary embolism		135
D 160/43	M.	64	Coronary thrombosis		475
D 165/43	M.	24	Acute enteritis		232
				Average	211·3
				'Median'*	179·7

* The groups were divided into three equal subgroups; the mean of the middle subgroup is described as the 'median' (Moore, 1937).

https://doi.org/10.1017/S002217240001370X Published online by Cambridge University Press

Table 2. *Vitamin A content of the liver in fifty persons from Oslo and surroundings in the period October 1943–August 1944*

No.	Sex	Age	Cause of death (autopsy diagnosis)		Vitamin A (i.u./g.)
			(1) Accidental death		
Rm 113/43	M.	22	Traumatic injury (fall)		257
U 754/43	M.	67	Traffic accident		205
U 759/43	M.	38	Traffic accident		199
U 760/43	F.	52	Traumatic injury (fall)		301
Rm 116/43	M.	40	Shot wound		503
U 781/43	M.	55	Traumatic injury (fall)		83
U 830/43	M.	50	Traffic accident		90
U 833/43	M.	35	Traffic accident		106
U 919/43	M.	*c.* 50	Explosion accident		232
U 920/43	M.	*c.* 30	Explosion accident		119
U 921/43	M.	*c.* 30	Explosion accident		351
Rm 17/44	F.	53	Traumatic injury (fall)		954
U 67/44	M.	44	Traffic accident		84
U 111/44	M.	30	Traffic accident		201
U 126/44	M.	28	Traumatic injury (fall)		201
U 170/44	M.	53	Traffic accident		376
U 226/44	M.	51	Traumatic injury (fall)		35
U 227/44	F.	28	Traumatic injury (fall)		825
Rm 75/44	M.	45	Traumatic injury (fall)		397
U 320/44	M.	32	Shot wound		1343
U 339/44	M.	42	Traffic accident		86
U 450/44	M.	39	Traffic accident		190
Rm 118/44	M.	21	Traffic accident		193
U 480/44	F.	4	Traffic accident		1260
U 518/44	M.	56	Traffic accident		251
Rm 144/44	M.	27	Shot wound		222
Rm 145/44	M.	24	Shot wound		484
				Average	317·5
				'Median'*	232·9
			(2) Poisoning		
U 770/43	M.	33	Methyl alcohol poisoning		492
U 776/43	M.	29	Methyl alcohol poisoning		699
U 778/43	M.	49	Methyl alcohol poisoning		253
U 779/43	F.	43	Methyl alcohol poisoning		154
U 783/43	M.	69	Opium poisoning		370
U 785/43	M.	25	Methyl alcohol poisoning		123
U 25/44	M.	50	Methyl alcohol poisoning		156
U 27/44	F.	34	Methyl alcohol poisoning		43
U 30/44	F.	32	Methyl alcohol poisoning		177
U 31/44	M.	37	Methyl alcohol poisoning		67
U 68/44	F.	56	Barbiturate poisoning		67
U 102/44	M.	43	Cyanide poisoning		137
Rm 35/44	M.	66	Cyanide poisoning		166
Rm 44/44	M.	56	Carbon monoxide poisoning		242
Rm 45/44	M.	65	Carbon monoxide poisoning		885
U 441/44	M.	35	Barbiturate poisoning		421
				Average	278·2
				'Median'*	191·3
			(3) Sudden death from disease		
Rm 18/44	M.	52	Myocardial fibrosis		336
Rm 19/44	M.	22	Myocardial fibrosis		386
Rm 23/44	M.	46	Coronary thrombosis		1274
Rm 26/44	M.	57	Coronary thrombosis		285
Rm 71/44	M.	45	Myocardial fibrosis		526
Rm 76/44	M.	21	Myocardial fibrosis		504
Rm 78/44	M.	60	Coronary thrombosis		255
				Average	510·9
				'Median'*	108·7

* See footnote to Table 1.

https://doi.org/10.1017/S002217240001370X Published online by Cambridge University Press

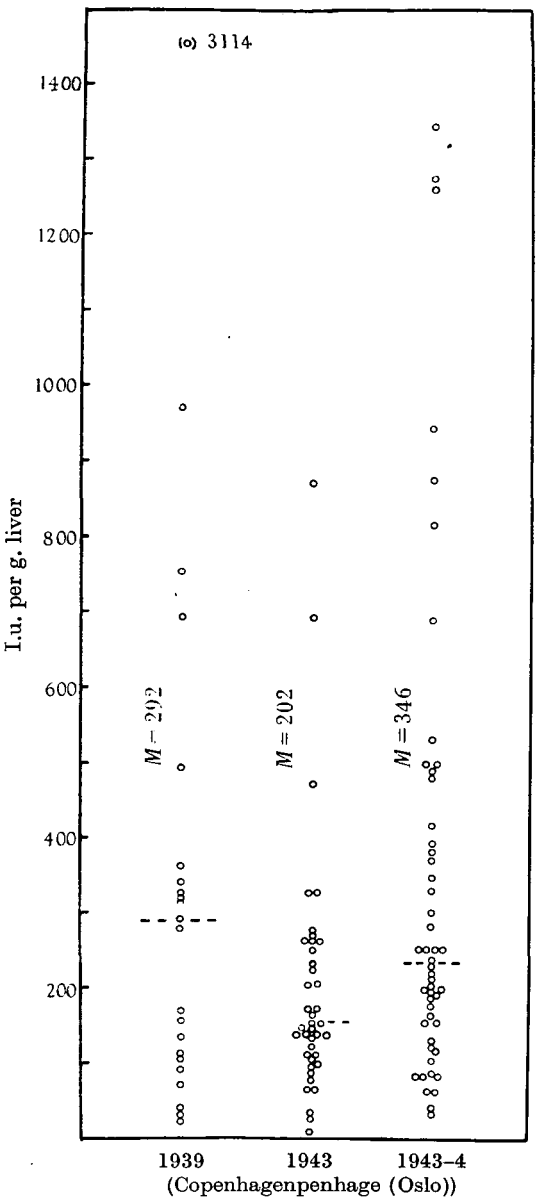

Fig. 1.

As seen from the figure, the level of the liver content of vitamin A was somewhat higher in Oslo than in Copenhagen during the war, and in Copenhagen it was a little higher in 1939 than in 1943. Each dot in the diagram represents one liver, the lines --- represent the medians of the materials and the figures *M* their averages. For the 1939 material the very high value of 3114 i.u./g. is, however, not included in the calculation of the average, as presumably it is due to an analytical error.

Our findings are surprising, as one would have expected a higher level in Copenhagen than in Oslo. As it is improbable that the higher liver reserve in Oslo can be due to a greater intake of vitamin A during the war it is legitimate to assume that the pre-war liver reserve in Oslo—which unfortunately is not known—has been a good deal higher than in Copenhagen. But whatever the explanation may be, our figures show that the population of Oslo as a whole cannot have suffered from serious vitamin A deficiency in 1943–4. That Hovind (1945) found slight haemeralopia to be relatively frequent in Oslo during this period is not necessarily contradictory to our results, as haemeralopia may under certain circumstances be found in individuals with considerable liver reserves (cf. With, 1940; Nylund & With, 1942).

On comparing our results with English (Moore, 1937) and Dutch (Wolff, 1932) investigations of the liver reserve of vitamin A, it is found that the level of the liver reserve in these countries in the pre-war period is of about the same magnitude as we found in Denmark before the war (cf. With, 1940).

SUMMARY

The authors have determined the vitamin A content of the liver in persons who had succumbed to sudden death in Copenhagen in 1939 and 1943 and in Oslo in 1943–4. The values in Oslo after three years of war and severe rationing were higher than in Copenhagen, the population of which had a much milder rationing during the war. The values in Copenhagen for 1943 were somewhat lower than those for 1939. The most plausible explanation of these findings seems to be that the pre-war vitamin A reserve in Norway was high.

REFERENCES

Hovind, B. (1945). *Nord. Hyg. Tidskr.* **7**, 239.
Jensen, H. B. & With, T. K. (1939). *Biochem. J.* **33**, 1771.
Moore, T. (1937). *Biochem. J.* **31**, 155.
Notevarp, O. (1945). *Nord. Med.* **28**, 2157.
Nylund, C. E. & With, T. K. (1942). *Vitamine und Hormone*, **2**, 6, 125.
With, T. K. (1940). *Absorption, Metabolism and Storage of Vitamin A and Carotene.* Monograph. Copenhagen: E. Munksgaard; London: Oxford Univ. Press.
With, T. K. (1941*a*). *Vitamine und Hormone*, **1**, 264.
With, T. K. (1941*b*). *Z. Vitaminforsch.* **11**, 172.
With, T. K. (1944). *Nord. Med.* **24**, 1797.
With, T. K. (1946). *Biochem. J.* In the Press.
Wolff, L. K. (1932). *Lancet*, **2**, 617.

(*MS. received for publication* 25. iv. 46.—Ed.)