The One Hundred and Second Meeting of The Nutrition Society was held at the Borough Polytechnic, Borough Road, London, S.E.1, on Saturday, 26 May, 1956, at 10.30 a.m., when the following papers were read:

Further studies on antibiotic and copper supplements for fattening pigs.

By R. S. Barber, R. Braude, K. G. Mitchell and J. A. F. Rook, *National Institute for Research in Dairying, University of Reading*, and J. G. Rowell, *Agricultural Research Council Statistics Group, University of Cambridge*

Barber, Braude & Mitchell (1955) showed that the addition of 0·1% copper sulphate to a fattening pig diet was as effective as aureomycin (chlorotetracycline) in increasing growth rate.

Results are reported of three further experiments with fattening pigs.

Supplements of either copper sulphate (0·1%), oxytetracycline (terramycin) (10 g/ton) or aureomycin (20 g/ton) were equally effective in significantly increasing growth rate. All the supplements improved the efficiency of food utilization but only with aureomycin did the effect attain the 5% level of significance. The rate of food consumption (lb./day) was significantly increased by all three supplements. The previous finding (Barber *et al.* 1955) that copper sulphate had no effect on food consumption, the increase in growth rate of the pigs resulting entirely from a significant improvement in their food-conversion rate, was not, therefore, substantiated.

A supplement of 10 lb./ton of 'Vigofac' (a fermentation product produced by Pfizer Ltd, which contains 4 g terramycin/10 lb.) was included in one of the experiments, and gave results that did not differ significantly from those obtained by adding terramycin alone.

There were no obvious differences in commercial carcass quality between the control and supplemented groups, nearly 80% of the carcasses being grade A.

The copper content of liver, kidneys, spleen, muscle and fat from control pigs and those given the copper supplement was determined. The liver copper of the treated pigs varied widely, but the mean value was eight times that of the controls. Smaller increases in the other organs and tissues from the supplemented pigs were observed.

As in previous work (Braude, Coates, Davies, Harrison & Mitchell, 1955), a tendency for aureomycin to reduce gut and liver weight was found. Copper supplementation also tended to reduce the weight of these organs but to a smaller extent. Contrary to our previous findings, aureomycin significantly reduced spleen weight.

An almost complete refusal of food was observed when pigs were given a meal to which either 1·0% or 0·5% copper sulphate had been added. Normal growth and food intake were resumed immediately the copper supplement was reduced to 0·1%.

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REFERENCES

Barber, R. S., Braude, R. & Mitchell, K. G. (1955). *Brit. J. Nutr.* **9**, 378.
Braude, R., Coates, M. E., Davies, M. K., Harrison, G. F. & Mitchell, K. G. (1955). *Brit. J. Nutr.* **9**, 363.

Some observations on the magnesium metabolism of dairy cattle. By C. C. BALCH, M. J. HEAD, C. LINE, J. A. F. ROOK and S. J. ROWLAND, *National Institute for Research in Dairying, University of Reading*

As part of an investigation of hypomagnesaemia in cattle, balance trials have been done with milking cows receiving initially winter feed and then, also in the stall, young spring herbage either freshly cut or after storage at −20°. The findings help to explain the cause and rapid onset of hypomagnesaemia which occurs frequently in cattle on changing from winter feeding to grazing.

Results for several animals are given in the table. The ingested magnesium not excreted in the faeces, which provides the Mg required for secretion in the milk and retention in the body, is referred to as the 'available' Mg; the Mg excreted in

Magnesium metabolism of milking cows

Cow	Feed	Length of trial (days)	Mg intake (g/day)	Mg in faeces (as percentage of that ingested)	'Available' Mg (g/day)	Mg in urine (g/day)	Mg requirement on winter feed (g/day)
A	Hay+dairy cubes	8	30·4	67·4	9·9	7·6	2·3
	Rye grass+clover (top-dressed with N)	8	20·3	72·1	5·7	0·8	—
B	Hay+silage+concentrates	8	27·7	78·3	6·0	3·0	3·0
	Cocksfoot* (top-dressed with N)	6	13·8	81·9	2·5	0·0†	—
C	Hay+silage+concentrates	8	27·6	78·1	6·1	2·2	3·9
	Cocksfoot* (no N)	6	15·3	73·1	4·2	0·9	—
D	Hay+silage+concentrates	8	23·3	77·8	5·2	1·1	4·1
	Cocksfoot* (no N)	6	12·9	84·6	2·0	0·0†	—
E	Hay+silage+concentrates	8	25·4	84·1	4·1	0·1	4·0
	Cocksfoot* (top-dressed with N)+concentrates	3	16·4	90·5	1·6	0·0‡	—

* This sward produced a high incidence of hypomagnesaemia in grazing cattle. † Blood serum Mg dropped from above 2·0 to 1·0 mg/100 ml. ‡ Blood serum Mg dropped from above 2·0 to 0·7 mg/100 ml.

the urine is the excess of 'available' Mg over requirement. On the winter rations used, the high Mg intake ensured that the requirement was met, even when the availability of the Mg in the ration was low. On the young herbage, the lower Mg intake, coupled with a generally lower availability (see also Head & Rook, 1955) frequently caused the 'available' Mg to fall well below the requirement found with the winter feed. When this occurred, the urine Mg fell, within 2–3 days, to very low levels and hypomagnesaemia quickly followed, yet the animals continued in positive Mg balance at the previous levels of from 0·1 to 2·0 g/day. Thus, it appears that the cows were unable to draw on body reserves, other than those circulating in the serum

and other extracellular fluids, to meet the shortage of Mg. The intake of herbage by cows fed in the stall was similar to that of others grazing the swards, and it is thought that the above findings apply also to field conditions.

REFERENCE

Head, M. J. & Rook, J. A. F. (1955). *Nature, Lond.*, **176**, 262.

The nutritional value of quail's eggs. By I. A. Simpson and Soh Chin Chai, *Institute for Medical Research, Kuala Lumpur, Federation of Malaya*

Fifty-four quail eggs were used for this work. The following measurements were made: total weight of eggs with shell 497·0 g (mean 9·2 g), weight of edible portion 416·6 g (mean 7·7 g), edible portion 83·7%.

The results of analysis are shown below compared with the values for hen eggs recorded by Platt (1945) in *Tables of Representative Values of Foods Commonly Used in Tropical Countries.*

	Edible portion of egg (amount/100 g)	
Nutrient	Quail	Hen
Water	72·0 g	74 g
Crude protein (N × 6·25)	11·6 g	13 g
Fat (lipids)	14·0 g	11 g
Ash	1·0 g	—
Calcium (as Ca)	57·2 mg	55 mg
Iron (as Fe)	3·3 mg	2·8 mg
Vitamin A (as alcohol)	233 μg (≡ 703 i.u.)	1000 i.u.
Carotenoids	0·88 mg	0·24 mg*
Thiamine	0·12 mg	0·14 mg
Riboflavin	0·57 mg	0·34 mg

* (Mann, 1943), on yolk only.

In general the results found for quail eggs do not differ greatly from those recorded for hen eggs with the exception that slightly higher values for lipids and riboflavin have been found and a much higher value for carotenoids.

REFERENCES

Mann, T. B. (1943). *Analyst*, **68**, 233.
Platt, B. S. (1945). *Spec. Rep. Ser. med. Res. Coun., Lond.*, no. 253.

An unusual form of vitamin A activity in herring eggs. By L. R. Fisher, G. F. Harrison, S. K. Kon, P. A. Plack, S. Y. Thompson and Pamela E. E. Todd, *Unit for Biochemical Research bearing on Fisheries Problems* at the National Institute for Research in Dairying, University of Reading*

In the course of a study of vitamin A in herring, Carr-Price tests were done by our usual method (Thompson, Ganguly & Kon, 1949) on the non-saponifiable residue

* Grant-aided by the Development Fund.

of the fat obtained from ripe herring eggs by extraction with a mixture of ethanol and light petroleum. Only small amounts of vitamin A were found, of the order of 0·6 μg/g, together with about 0·3 μg/g of carotenes.

As Junker (1952–3) had reported by biological test much higher values for herring eggs, 2–14 μg/g depending on maturity of gonads, we carried out biological assays which fully confirmed the German findings. The vaginal-smear method with rats (Pugsley, Wills & Crandall, 1944) gave a value of >4 μg/g and a liver-storage test with chicks a value of 5·3 μg/g with true fiducial limits at $P = 0{\cdot}95$ of 4·3 — 6·3 μg/g.

Modifications of the extraction procedure by preliminary treatment with aqueous KOH (Davies, 1933), alcoholic KOH, aqueous HCl, grinding with anhydrous sodium sulphate (Glover, Goodwin & Morton, 1947) or incubation with papain, trypsin, enzyme preparations from the stomach or the intestine of rats, or Takadiastase as a source of phosphatases, did not increase the Carr-Price value. The fat fraction obtained by direct solvent extraction or after treatment with aqueous KOH had no unaccountable biological activity. The biological activity remained in the residue after simple solvent extraction. It was also present in the supernatant liquid of spun homogenates of eggs in water or saline and was associated in it with the proteins.

The vitamin A recovered from chick and rat livers after liver-storage tests with herring eggs was physico-chemically indistinguishable from that stored from vitamin A acetate.

Preliminary tests did not indicate the presence of a hidden form of vitamin A or of an uncharacterized precursor in the eyes and bodies of *Meganyctiphanes norvegica*, in the eggs of *Pandalus bonnieri* and in the eggs of the hermit crab *Eupagurus prideauxi*.

REFERENCES

Davies, A. W. (1933). *Biochem. J.* **27**, 1770.
Glover, J., Goodwin, T. W. & Morton, R. A. (1947). *Biochem. J.* **41**, 97.
Junker, M. (1952–3). *Arch. Fischwiss.* **4**, 19.
Pugsley, L. I., Wills, G. & Crandall, W. A. (1944). *J. Nutr.* **28**, 365.
Thompson, S. Y., Ganguly, J. & Kon, S. K. (1949). *Brit. J. Nutr.* **3**, 50.

Observations on dietary requirements and hair growth. By W. MONTAGNA (introduced by R. J. L. ALLEN), *Brown University, Providence, Rhode Island, U.S.A.*

Under normal conditions, the amount of time which a follicle requires to produce a hair of a length characteristic of the species is always the same. Thus, in the mouse and the rat, the individual follicles require 19 and 24 days, respectively, to complete a cycle of growth. Furthermore, if a hair is plucked from a quiescent follicle, the follicle becomes active at once and within 21 days in the mouse and 26 days in the rat it has completed its growth cycle and is again resting. Armed with this information it is possible to study the effects of a variety of experimental conditions on hair growth. (1) If the diet of mice is reduced to 1·5 g daily, and the resting hairs are

plucked on the day the animals are placed on this régime, or 3 days before, the follicles remain quiescent for as long as the food remains restricted. They will begin to grow on the same day that the animals are given an adequate amount of food. If the hairs are plucked 5 days before the food is restricted the follicles grow slowly and require a longer time to complete their period of growth. When they are plucked 10 days before the restriction of food commences, the follicles complete their cycle of growth in a normal time. (2) Acute vitamin A deficiency inhibits all spontaneous growth of hair in the mouse, but not the growth initiated by plucking. (3) Large doses of vitamin A applied topically to the skin have no effect on the rate of growth of hairs, but the skin which contains resting hair follicles becomes grossly irritated. (4) In biotin deficiency the hair seems to grow at a normal rate but the keratin is faulty and the hair shaft becomes dissociated. (5) Riboflavin deficiency is said to cause a scaliness of the epidermis and a loss of hair, but these could be secondary responses of the skin to systemic disturbances.

Intracellular localization of vitamin C in the adrenal cortex. By G. H. Bourne, *Department of Histology, London Hospital Medical College*

Vitamin C was first demonstrated histochemically in the cells of the adrenal cortex by Bourne (1933*a*,*b*) and further studies were carried out by Leblond (1934) and Giroud and Leblond in a series of papers (see Giroud, 1939). The chemical specificity of the reaction (using acid silver nitrate) has survived for over 20 years without serious criticism, although caution is necessary in interpreting the results. However, there has been, and still is, considerable controversy as to the accuracy of the intracellular localization. Bourne (1951) and Giroud and Leblond claimed that in many organs the reaction in addition to being in the form of scattered granules in the cells was localized in the Golgi region. A number of authors have criticized the localization of the vitamin in the Golgi apparatus. It has been attributed to the use of photographic hypo., to lack of speed in placing the tissue in acid silver nitrate or to be simply an impregnation artifact. Eränko (1954) applied the technique to frozen dried sections and found that in both these and in ordinary frozen sections the granules of vitamin C were distributed throughout the cell.

The present author has studied the penetration of the acid silver nitrate through 'squashes' of adrenal cortex under the microscope and finds no evidence of diffusion of the vitamin C in the intact cells and that the vitamin appears to be associated, as Eränko found, with large granules distributed through the cytoplasm of the cell. In the adrenal-cortex homogenates however, silver-nitrate staining ability is retained in the supernatant not in the granular fraction so that damage to the cell causes discharge of the vitamin from the granules to which it is bound (the latter appears to contain both phospholipid and protein). Further studies using acid selenium dioxide which is specifically reduced to selenium metal by vitamin C, confirm these results. This reagent does not react with chlorides to produce compounds which degrade under the influence of light resulting in visible products which confuse the interpretation of the reaction.

It seems that most of the vitamin C in the adrenal cortical cells is attached to granules which are scattered fairly evenly throughout the cell, but the possibility that in some physiological states of the cell the vitamin is associated with the Golgi material cannot be excluded.

REFERENCES

Bourne, G. (1933*a*). *Nature, Lond.*, **131**, 874.
Bourne, G. (1933*b*). *Nature, Lond.*, **132**, 859.
Bourne, G. H. (1951). In *Cytology and Cell Physiology*. [G. H. Bourne, editor.] Oxford: University Press.
Eränko, O. (1954). *J. Histochem. Cytochem.* **2**, 167.
Giroud, A. (1939). *L'Acide Ascorbique dans la Cellule et les Tissues. Protoplasma-Monogr., Berl.*
Leblond, C. P. (1934). *La Vitamine C dans l'Organisme.* Paris: Laval.

Experimental nutritional hepatic fibrosis. By I. MACDONALD and G. A. THOMAS, *Departments of Physiology and Anatomy, Guy's Hospital Medical School, London, S.E.*1

It has been found (Macdonald & Thomas, 1956*a*) that feeding rabbits on a diet of casein hydrolysate and sucrose, together with dried yeast and vitamins A and D leads to an increase in the amount of fibrous tissue in the liver. The nature of the fibrous reaction was histologically very similar to that seen in West African children (Walters & Waterlow, 1954) with kwashiorkor.

The distribution of the fibrous tissue was such that a periportal fibrosis developed. At first this is confined to the portal areas, but subsequently these areas are linked by strands of connective tissue which join the areas together, thus completely surrounding the lobules with a fibrous sheath. Fibrosis around the central vein of the lobules also occurs, though this is less frequent than periportal fibrosis. In extremely fibrous livers, fibres are seen joining the central vein of one lobule to that of neighbouring lobules, resulting in a pseudolobulation. In the early lesion fat is found in the liver cells around the central vein. This disappears later, a little fat being left behind in what appear to be phagocytes in the fibrosed portal areas.

It has been shown (Macdonald & Thomas, 1956*b*) that the extent of the fibrous reaction of the liver is related to the degree of infection of the liver with the parasite *Eimeria stiedae*. This finding supports the hypothesis of a dual aetiology and suggests that the chronic liver infection and a dietary factor each exert a subthreshold stimulus to fibrosis in the liver.

In order that the nature of the dietary factor may be determined various supplements known to be associated with hepatic function have been added to the diet but none has been found to prevent the development of the fibrosis.

It was therefore decided to try various types of casein and to alter the sugar. Another, less completely hydrolysed casein was used, and calcium caseinate was also substituted for the original protein and lactose replaced the sucrose. None of these variants affected the outcome.

REFERENCES

Macdonald, I. & Thomas, G. A. (1956*a*). *Clin. Sci.* (In the Press.)
Macdonald, I. & Thomas, G. A. (1956*b*). *J. Physiol.* **131**, 25P.
Walters, J. H. & Waterlow, J. C. (1954). *Spec. Rep. Ser. med. Res. Coun., Lond.*, no. 285.

Food, growth and renal function in the early days of life. By Elsie M. Widdowson and R. A. McCance, *Medical Research Council Department of Experimental Medicine, University of Cambridge*

It has been known for some years that the urea clearances are lower in the 1st fortnight of a child's life than they are later, whether surface area or body-weight is used as the basis of comparison. This was at first interpreted as indicating that the infant's kidney was rather inefficient by adult standards, but was this correct? The healthy kidney can and does do all that is necessary, in infancy as at all ages, to maintain the internal environment in the same steady state. We had always appreciated that growth must relieve the kidneys of newborn animals of some of the work which they were called upon to do in adult life, for if an animal is growing, some of the material in its food must be incorporated into the structure of its tissues, and will not present itself in any form for excretion by the kidney. The extreme importance of growth, however, in the regulation of the stability of the internal environment was only recently brought home to us as a result of some experiments which were made on newborn piglets. Two piglets of about the same weight were selected from each of four litters and placed in metabolism cages so that their urine could be collected quantitatively. They were fed every 2 h for about 40 h by stomach tube; one of the piglets was given sow's milk, and the other an equal volume of water. The protein in the food of the 'fed' piglets provided about 3300 mg nitrogen/kg/day. These piglets gained weight; 90% of the N in their food was incorporated into tissue protein, and only 10% was broken down to urea and required to be excreted. The piglets which received water lost weight, and they katabolized about 300 mg of their tissue N/kg/day. There was little to choose between the blood urea of the piglets which were 'fed' or 'not fed' and their N excretions were not very different. If the whole of the N in the food of the 'fed' piglets or its equivalent of tissue protein had been converted to urea, as it would have been in an adult, the concentration of urea in the blood would have risen from 30 to 800 mg/100 ml. in 24 h.

The capacity of the animal to grow and the composition of its food are clearly of vital importance in maintaining the composition of its body fluids at a level which is compatible with life.

Citral poisoning. By E. H. Leach, *University Laboratory of Physiology, Oxford,* and J. P. F. Lloyd (introduced by G. H. Bourne), *Oxford Eye Hospital, Walton Street, Oxford*

Citral administered to rabbits subcutaneously or by mouth causes damage to vascular endothelia even with a single dose of 5 μg/kg. In monkeys, similar damage is caused by a daily dose of 1 μg/kg over a period of 3 weeks. Larger doses cause little further symptoms in rabbits but can produce severe diarrhoea in monkeys.

Other unsaturated aldehydes containing the C:CH.CHO group have a similar effect; these include acrolein, crotonaldehyde, furfural, cinnamic aldehyde and crocetin aldehyde. The corresponding alcohols and acids are also active. Citronellal,

an unsaturated aldehyde with the double bond elsewhere in the molecule has no similar toxic action nor do saturated aldehydes.

The citral effect can be reversed by administration of sulphydryl compounds such as cysteine, uramino-cysteine, B.A.L., and *o*-aminothiophenol. In larger doses the latter two substances can cause an effect in a normal rabbit similar to that resulting from citral poisoning; *p*-chloromercuribenzoic acid, a sulphydryl-blocking agent, has a similar effect. Aldehyde-trapping agents such as hydroxylamine and sodium bisulphite appear to reverse the effect of citral.

Vitamin A can protect against and reverse the citral effect. An improvement in fluorescence microscopy has made it possible to show the presence of vitamin A in endothelial cells. It seems probable that in endothelial cells, vitamin A plays a role similar to that which, it has been shown by Wald, it does in the rods of the retina where retinene combines with sulphydryl groups. But in the endothelial cells it would play some part in the oxidation—reduction systems. It is noteworthy that Bennett (1951) has shown that capillary endothelial cells contain detectable sulphydryl compounds.

A compound such as citral might act as a retinene competitor because both compounds terminate in a C:CH.CHO group. Citral and retinene would compete for the sulphydryl groups and citral would thus interfere with the metabolism of the cells.

If man is sensitive to citral the expected dose to cause damage would be about 50 μg a day. This is contained in 1 mg of orange oil or 2 g of orange peel. It has been detected in orange marmalade and may be present in citrous fruit 'squashes' flavoured with the corresponding oils. Orange juice made by compression of the whole fruit might contain it. The effects of intoxication would be damage to blood vessels and might be a contributory cause in some cardiovascular diseases.

REFERENCE

Bennett, H. S. (1951). *Anat. Rec.* **110**, 231.

Plastics and food technology. By R. L. WORRALL, 31 *Braeside Avenue, Sevenoaks, Kent*

The increasing use of plastics as food containers calls for careful consideration of their chemistry. Although a solid finished plastic may be chemically inert in the ordinary sense of the word, it can nevertheless produce marked biological effects. In factories where plastic products are made, allergic reactions of workers to the solid finished products are not unknown, and are evidently due to escape of reactive chemical entities from the solid surfaces of the finished plastics. A case of this kind was described recently by Dr L. B. Bourne (unpublished), at a symposium of the Society of Chemical Industry on 12 April 1956 on epoxide resins.

Oppenheimer, Oppenheimer, Stout & Danishefsky (1953) found that a variety of sterile plastics, some of them devoid of plasticizers and other impurities, produced malignant tumours after implantation under the skin of rats and mice, the proportion of these

animals which developed malignant tumours ranging from 12·5 to 45·4%. Control solids, including glass and cotton fibres, gave negative results. On removal from the animals, most of the plastic films appeared as transparent and almost as glossy as before implantation.

These findings suggest that while solid plastics in bulk form may be chemically inert, there may be some occasional outward migration of highly reactive atoms or free radicals, or possibly monomers, from the solid surface of a plastic polymer. This possibility is particularly relevant to fat as a foodstuff or tissue component, since various atoms and free radicals can initiate, under favourable conditions, the self-perpetuating chain reactions of fat autoxidation.

In a preliminary investigation, a layer of fat is spread on the surface of a solid plastic, and a control layer of the same fat on a glass surface. After a certain period of exposure to the air, the fat is tested for rancidity by means of flavour. Results are incomplete, and will require comparison with colorimetric estimations.

In a hitherto unpublished paper communicated to the Leukemia Society, the author has put forward the view that carcinogenesis is primarily the initiation of neutral fat autoxidation, by irradiation or chemical agents. The present investigation of plastics in relationship to fat autoxidation is relevant to this point of view.

REFERENCE

Oppenheimer, B. S., Oppenheimer, E. T., Stout, A. P. & Danishefsky, I. (1953). *Science*, **118**, 305.

Trends in dietary supplies in the United Kingdom of fats, essential fatty acids and pyridoxin. By DOROTHY F. HOLLINGSWORTH, MARGARET C. VAUGHAN and GRACE M. WARNOCK, *Scientific Adviser's Division (Food), Ministry of Agriculture, Fisheries and Food, Great Westminster House, Horseferry Road, London, S.W.*1

An attempt has been made to compare the amounts of these nutrients in British food supplies before 1914 and in 1950 and 1952, despite the disadvantage that estimates of food supplies for 1909–13 (Royal Society Committee, 1917) are not strictly comparable with those for 1950 and 1952 (Ministry of Food, 1953) mainly because the former cover Eire.

Estimates of fat content have been based on the food tables of the Medical Research Council: Accessory Food Factors Committee (1945), and of the essential fatty acids (E.F.A.) (linoleic, linolenic and arachidonic) from values given by Hilditch (1956) and Deuel (1954), which are maximal. It has been assumed that the E.F.A. content of hydrogenated fats (including all whale oil) is negligible and that none is lost in unhydrogenated oils: no account has been taken of any possible effect on E.F.A. of continued re-heating, since data on this subject are lacking. Pyridoxin has been calculated from analyses summarized by Bicknell & Prescott (1953). All figures for E.F.A. and pyridoxin must be accepted with reserve, since the available data are limited, those for fish being particularly scanty.

Total fat. The total fat supply rose from 100 to 130 g/head/day between 1909–13 and the 1930's. During the second world war it varied between 113 and 124 g but fell to 106 g in 1947, gradually rising again to 131 g in 1950 (Ministry of Food, 1953). In 1909–13 about 8% of the total fat was derived from vegetable sources compared with about 30% in the 1950's. Some 9% of the total fat was hydrogenated in the 1950's.

Essential fatty acids. Dairy foods, eggs, meat and meat products (including lard and dripping) supplied roughly the same amount (7 g/head/day) of E.F.A. in the 1950's as in 1909–13. Neglecting any destruction of E.F.A. by bleaching agents and flour improvers, cereals provided in both periods about 2 g, while the contribution of fish fell from 1·3 to 0·5 g. In 1909–13 margarine, which was made mainly from animal fats without hydrogenation, contributed only 0·3 g E.F.A. In the 1950's margarine, compound cooking fats and edible oils together furnished nearly six times as much total fat as in 1909–13 and, though made mainly from partly hydrogenated vegetable oils and whale oil, were still so much richer in E.F.A. that together they contributed over 4 g E.F.A.

In 1909–13 the total estimated daily intake of E.F.A. was 10 g/head; by the 1950's this had risen to 14 g. Whatever relative allowances are made in the two periods for the destruction by bleaching agents and flour improvers of the 2 g contributed by cereals, the total figure for the 1950's would still be significantly higher than in 1909–13.

Pyridoxin. Between 1909–13 and the 1950's pyridoxin increased from between 1·0 and 1·5 mg to between 1·6 and 1·9 mg/head/day, the levels varying with the nature and quantity of flour and the consumption of potatoes and other vegetables; these foods together account for about four-fifths of the total.

REFERENCES

Bicknell, F. & Prescott, F. (1953). *The Vitamins in Medicine*, 3rd ed. London: W. Heinemann.
Deuel, H. J. Jr. (1954). In *Progress in the Chemistry of Fats and Other Lipids*. Vol. 2, p. 99. [R. T. Holman, W. O. Lundberg & T. Malkin, editors.] London: Pergamon Press.
Hilditch, T. P. (1956). *The Chemical Constitution of Natural Fats*, 3rd ed., revised. London: Chapman and Hall.
Medical Research Council: Accessory Food Factors Committee (1945). *M.R.C. (War) Memor.* no. 14.
Ministry of Food (1953). *Bull. Minist. Fd, Lond.*, no. 720, p. 6.
Ministry of Agriculture, Fisheries and Food (1956). Unpublished data.
Royal Society Committee (1917). *The Food Supply of the United Kingdom* [Cd 8421]. London: H. M. Stationery Office.

Dietary diseases of civilization. By H. M. SINCLAIR, *Laboratory of Human Nutrition, University of Oxford*

Diseases that are increasing rapidly in this country (and in generàl in the more highly civilized countries) include coronary thrombosis, lung cancer, disorders of the nervous system accompanying lung cancer, duodenal ulcer, leukaemia and idiopathic hypercalcaemia of infants. The last of these is no doubt partly caused

by the high content of vitamin D and calcium in the diets of the infants which are artificially fed, but it cannot be entirely attributed to this cause; a relative dietary deficiency of essential fatty acids (E.F.A.), which I have suggested elsewhere (Sinclair, 1956) may be important in certain diseases of civilization, may play a part in hypercalcaemia of infants as well as in the nervous complications of lung cancer. Evidence for this will be discussed.

Coronary thrombosis may be regarded as arising from atherosclerosis of the coronary vessels and increased coagulability of the blood; both of these may be related to deficiency of E.F.A. It appears that deaths from this may not have increased much in the years before World War 1: in 1911 and 1921 the crude death rates for England and Wales from angina pectoris were the same (41 per million living). Around 1926 a sudden increase occurs in the Comparative Mortality Index for diseases of the coronary arteries and angina pectoris. This rise continues with (in general) increasing acceleration except for slight slowing in 1941, 1948 and 1953. Deaths are commoner in urban than in rural areas. The mortality appears to have no relation to the *per caput* intake of essential fatty acids or pyridoxin as calculated by Hollingsworth, Vaughan & Warnock (1956) in the preceding paper but there appears to be some correlation between the rise in mortality and the increased consumption of vegetable fats.

Unfortunately present chemical methods of estimating E.F.A. give falsely high values: the usual spectrographic estimation of dienes, trienes, etc. is carried out after alkali isomerization at a high temperature. Conjugated polyenes and *trans* isomers are therefore included; these are not only inactive as E.F.A. but in some cases are actually antagonistic. For instance, natural (*cis*, *cis*) linoleic acid, with a malonic distribution of double bonds, is readily oxidized in air at room temperature to the hydroperoxide with a shift of one double bond to give a conjugated arrangement and a shift to the *trans* position. In the manufacture of margarine and in frying of foods similar isomerization and oxidation occurs. Recent analyses will be given.

REFERENCES

Hollingsworth, D. F., Vaughan, N. C. & Warnock, G. N. (1956). *Proc. Nutr. Soc.* **15**, xvii.
Sinclair, H. M. (1956). *Lancet*, **270**, 381.