Keith, J. I., Kuhlman, A. H., Weaver, E. & Gallup, W. D. (1934). *Rep. Okla. agric. Exp. Stn*, 1932–34, no. 164 (quoted by Phelps *et al.* 1965).
Kemmerer, A. R. (1960). Unpublished work (quoted by Phelps *et al.* 1965).
Kemmerer, A. R., Heywang, B. W. & Vavich, M. G. (1961). *Poult. Sci.* **40**, 1045.
Kircher, H. W. (1964). *J. Am. Oil Chem. Soc.* **41**, 4.
Kratzer, F. H., Davis, P. N. & Marshall, B. J. (1955). *Poult. Sci.* **34**, 462.
Kühn, B. & Bengen, F. (1906). *Z. Unters. Nahr.-u Genussmittel.* **12**, 145.
Lorenz, F. W. (1939). *Poult. Sci.* **18**, 295.
Lorenz, F. W. & Almquist, H. J. (1934). *Ind. Engng Chem.* **26**, 1311.
Marshall, B. J. & Scott, W. J. (1948). Unpublished work (quoted by Phelps *et al.* 1965).
Masson, J. C., Vavich, M. G., Heywang, B. W. & Kemmerer, A. R. (1957). *Science, N.Y.*, **126**, 751.
McDonald, M. W. & Shenstone, F. S. (1958). Unpublished work (quoted by Phelps *et al.* 1965).
Moore, H. K., Richter, G. A. & Van Arsdel, W. B. (1917). *Ind. Engng Chem.* **9**, 451.
Morgan, C. L. & Ringrose, R. C. (1939). *Rep. S. Carol. agric. Exp. Stn*, **52**, 93 (quoted by Phelps *et al* 1965).
Naber, E. C. & Morgan, C. L. (1957). *Poult. Sci.* **36**, 429.
Narain, R., Lyman, C. M. & Couch, J. R. (1957). *Poult. Sci.* **36**, 1351.
Nordby, H. E. (1963). Sterculic acid derivatives and cyclopropene compounds in relation to the Halphen test and physiological function. Thesis, University of Arizona.
Nordby, H. E., Heywang, B. W., Kircher, H. W. & Kemmerer, A. R. (1962). *J. Am. Oil Chem. Soc.* **39**, 183.
Phelps, R. A., Shenstone, F. S., Kemmerer, A. R. & Evans, R. J. (1965). *Poult. Sci.* **44**, 358.
Pieraerts, J., Ipatieff, N. & Simar, E. (1928*a*). *Matières grasses*, **20**, 8056.
Pieraerts, J., Ipatieff, N. & Simar, E. (1928*b*). *Matières grasses*, **20**, 8085.
Schneider, D. L. (1962). Some physiological and biochemical effects of *Sterculia foetida* oil on animal systems. Thesis, University of Arizona.
Schneider, D. L., Vavich, M. G., Kurnick, A. A. & Kemmerer, A. R. (1961). *Poult. Sci.* **40**, 1644.
Shenstone, F. S. & Vickery, J. R. (1956). *Nature, Lond.*, **177**, 94.
Shenstone, F. S. & Vickery, J. R. (1959). *Poult. Sci.* **38**, 1055.
Shenstone, F. S. & Vickery, J. R. (1961). *Nature, Lond.*, **190**, 168.
Thomasson, H. J. (1955). *J. Nutr.* **56**, 455.
Wilson, T. L., Smith, C. R. Jr. & Mikolajczak, K. L. (1961). *J. Am. Oil Chem. Soc.* **38**, 696.

Toxic Contaminants

Chairman: PROFESSOR F. G. YOUNG, DSc, PhD, FRS, *Biochemical Laboratory, University of Cambridge*

Pesticide residues in fat-containing foods and in human fat

By H. EGAN, *Laboratory of the Government Chemist, Ministry of Technology, London, SE1*

The term 'pesticide' is broadly applied to a wide range of insecticides, fungicides, herbicides, molluscicides, rodenticides and similar substances. Over 100 different agricultural pesticides, of which there are perhaps 800 different formulations, are at present cleared for use in Great Britain. These can be broadly classified according to their general chemical nature into the principal types shown in Table 1. The toxicity to mammals may vary considerably from one pesticide to another within the same chemical type, as indicated in the table. Edson, Sanderson & Noakes (1965) have recently published a useful detailed list of acute toxicities for a large number of pesticides. Safety to both users and consumers in agricultural, veterinary or food storage practice, however, cannot be judged by acute toxicity alone.

Table 1. *Chemical classification of pesticides and toxicity to mammals*

Chemical type	Example	Typical action	General toxicity* <100 mg/kg	100–500 mg/kg	>500 mg/kg
Organo-phosphorus	Malathion	Insecticide	+	+	+
Organo-chlorine	DDT	Insecticide	+	+	+
Chlorophenoxy acid	2,4-D	Herbicide	−	−	+
Heterocyclic	Aminotriazole	Herbicide	−	+	+
Carbamate	Propham	Herbicide	−	−	+
Dithiocarbamate	Thiram	Fungicide	−	+	+
Organo-metallic	Phenylmercury salicylate	Fungicide	+	−	−

*Based on mouse or rat acute oral LD_{50} values only.

The synthetic organo-chlorine insecticides typified by DDT and dieldrin have received special attention in recent years, mainly on account of their persistence; that is to say, on account of the stability of residues of the applied pesticide or immediate degradation products. Persistence of itself is not necessarily an undesirable feature; indeed, it can be a most valuable characteristic. It cannot be predicted, so to speak, from chemical constitution; nor is it easy to characterize or measure by any simple laboratory test based on physical properties.

The persistent pesticides and some of their immediate conversion products are chemically and physically stable. The products may be either more or less toxic than the parent pesticide: heptachlor is converted into a stable and more toxic epoxide whereas p,p′-DDT is dehydrochlorinated to the substantially non-toxic p,p′-DDE (1,1-dichloro-2,2-di-(4-chlorophenyl) ethylene). Another feature common to the persistent organo-chlorine pesticides is their solubility in fatty material. Residues of these substances are stored in the adipose tissue of animals exposed to them. Storage is not indefinitely cumulative, however; Hayes, Durham & Cueto (1956) have estimated that for man a uniform dietary DDT intake leads to an equilibrium residue level in the fat in about 1 year. Organo-chlorine insecticides feature in the so-called food-chain effect, in which stable residues are passed from smaller animal species to larger predators, being concentrated in fatty tissue in the process. A general account of food chains has recently been given by Rudd (1964). Marine organisms remove and concentrate minute traces of DDT from water and so may lead ultimately to much higher residue levels in fish, fish-eating birds and their eggs, as observed by Holden (1962) and by Moore & Walker (1964); similarly terrestrial birds may ingest DDT with earthworms or small mammals and themselves become a source to larger predatory species.

Modern analytical chromatographic methods of residue analysis give results which are not always directly comparable with those obtained by older colorimetric methods: the latter are less specific and may give high indications. Caution may also be necessary in the interpretation of results obtained by modern analytical methods. Apart from the normal precautions to ensure that chromatographic data are free from interference due to non-pesticidal substances, care is necessary to ensure that any residues present are not themselves altered by decomposition or isomerization

in the course of the analysis. Furthermore, it is most important that results eventually obtained with the very sensitive modern analytical methods are not themselves misused. When using gas–liquid chromatography it is normal to employ at least two different columns, one polar and the other non-polar in character, together with an independent analytical method such as paper or thin-layer chromatography or infrared spectrophotometry as described by Abbott, Crosby & Thomson (1965). A commercially available system in use in the United States employs gas–liquid chromatography followed by microcoulometry.

The occurrence of pesticide residues in foods has been reviewed by Durham (1963), who also gives a general description of toxicity studies and of the quantitative dose–effect relationships for the organo-chlorine and the organo-phosphorus compounds. A normal dose–response curve is found for the various pesticides which have been studied, implying that small amounts can be harmless even though larger doses may produce poisoning. Organo-chlorine insecticide residues found in American milk by Henderson (1965) were mainly of the DDT type. Marth (1965) has reviewed the occurrence of organo-chlorine residues in a wide range of biological material, including animal and vegetable foods, water and human tissue. The residues of certain organo-phosphorus insecticides found in Canadian vegetables are given by Coffin (1964). Total-diet studies conducted by Mills (1963) in the United States have to date given a reassuring picture. Homogenized samples corresponding to the dietary intake of 19-year-old youths were examined both qualitatively for organo-phosphorus residues and quantitatively for organo-chlorine residues. No individual pesticide level exceeded 0·1 ppm.

In Great Britain, details of any residues likely to occur in crops or animal tissue resulting from the use of new pesticides are, under present voluntary arrangements, submitted by manufacturers to the Ministry of Agriculture, Fisheries and Food. In appropriate cases, the Minister consults the Advisory Committee on Pesticides and Other Toxic Substances before the use of the pesticide is approved. Produce may be examined for pesticide residues by public analysts appointed by local food and drugs authorities; Hamence (1964, 1965) has reported that of 1317 samples, mostly fruit and vegetables, examined for organic insecticides in 1963, only forty-two showed a positive response in a biological sorting test; and that of the thirty-six of these which it was possible to subject to more specific examination, only eight contained residues in excess of 1 ppm. Because of the interest in persistent organo-chlorine residues, studies of selected home-produced and imported foods for residues of the principal compounds have since 1962 been made by the Plant Pathology Laboratory of the Ministry of Agriculture, Fisheries and Food and by the Laboratory of the Government Chemist (1964, 1965). Residue levels found in bulk milk, butter, mutton fat and beef fat are summarized in Table 2. The highest levels found are in fat of home-produced mutton. These almost certainly arise from the use of sheep-dipping preparations containing dieldrin, the use of which in Great Britain will not be authorized after 1 January 1966. Residues of up to 10 ppm of dieldrin have been observed in fat from sheep slaughtered within a few weeks of dipping. The levels appear to depend, among other things, on the weight of the fleece at

Table 2. *Approximate average pesticide residue levels in fat-containing foods (Laboratory of the Government Chemist,* 1965)

Food	Dieldrin (HEOD)	Total* DDT	BHC isomers
Bulk milk	0·002	0·005	<0·005
Butter: home-produced	<0·05	0·05	0·05
imported	<0·05	0·05–0·40	0·0–0·05
Mutton kidney fat: home-produced	1·0	<0·05	0·05
imported	<0·05	0·1	0·0–0·25
Beef kidney fat: home-produced	<0·1	<0·1	0·05
imported	Normally low	<0·05	0·05

*DDT+DDE+TDE.

dipping, and fall as the interval between dipping and slaughter increases. Egan (1965) has estimated that under UK conditions dieldrin residues in the kidney fat of newly shorn sheep fall to 0·25 ppm at 5–6 months after dipping. For γ-BHC a level of 0·5 ppm is reached after 3–4 months for shorn sheep but only after about 6 months for unshorn sheep. Residues in beef kidney fat are much lower, though an occasional imported sample has been found to contain 1 ppm of organo-chlorine pesticide. The origin of such residues in beef fat is not clear. They could arise from residues in feeding-stuffs; or they could be due to contamination, accidental or otherwise, in the treatment of storage or other premises. Residues arising from the use of insecticidal vaporizers have been studied by Dyte (1963) and were stated by Hamence (1965) to be the source of some relatively high residues found on fruit.

Residues of p,p′-DDT and p,p′-DDE in butter vary from 5 parts per hundred million, which may be taken as the limit of detection with the very sensitive gas–liquid chromatographic method described by de Faubert Maunder, Egan & Roburn (1964), to about 0·5 ppm in some samples. The lowest levels, if they are of interest, present the analyst with a special difficulty since independent analytical methods for the confirmation of the identity of the insecticides detected may not be available. The lower limit of detection for milk is approximtely 1 part per thousand million.

DDT-type residues in human fat are frequently expressed in terms of 'total DDT equivalent', in which the DDE present is re-calculated as original DDT and the resulting level added to that of the DDT present as such: it is not possible to deduce the dietary DDT:DDE ratio from the residue ratio, however. Both DDT and DDE are slowly eliminated as DDA (di-(4-chlorophenyl) acetic acid) and other soluble degradation products.

Several workers have studied residue levels in human fat; a recent summary is given by Egan, Goulding, Roburn & Tatton (1965). Relatively high levels of DDT are found in the general north American population—about 15 ppm total equivalent DDT—whereas in Britain the total DDT level is about 5 ppm. There is a suggestion that on the European continent a progressive rise to the American level is found as one progresses eastwards. Some 65% of the total DDT residue is present as DDE, though at very high experimental DDT dosages, the proportion may be much

lower. According to W. J. Hayes (1965, private communication) the DDT level in the general population of India is some two or three times as much as that found in America. However, in an American study of men subjected to prolonged and intensive industrial exposure to DDT, Ortelee (1958) found no evidence of general chronic DDT poisoning. No complaints or symptoms of illness related to DDT exposure were observed by Hayes *et al.* (1956) when studying human volunteers receiving up to 35 mg/day over a period of about 18 months although residue levels in subcutaneous fat approached those associated with prolonged industrial exposure, e.g. 300 ppm or more.

Ludwig, Weis & Korte (1964) have shown that rats given daily 4·3 μg radio-labelled aldrin excrete aldrin at the same rate as it is ingested after about 8 weeks, the storage level being about 0·15 ppm in the whole rat. The presence of mixed, unidentified hydrophilic metabolism products was detected in the urine and faeces; Cueto & Hayes (1962) and Heath & Vandekar (1964) have also described a number of as yet unidentified metabolites. Again using radio-labelled materials, Backstrom, Hansson & Ullberg (1965) have studied the distribution of DDT, dieldrin and chlordane administered to rats. They obtained clear evidence of the passage of DDT and dieldrin across the placental membrane in pregnant mice and the accumulation in fatty tissue was always confirmed, high concentrations of DDT and dieldrin also being observed in the liver, intestines and mammary glands.

Like DDT and BHC, dieldrin appears to be of no direct nutritional significance. However, a converse effect, that of dietary protein level in the rat on the toxicity of dieldrin, has been described by Lee, Harris & Trowbridge (1964): mortality at 150 ppm added dieldrin was greater for diets low (10%) in protein than for high-protein (25%) diets. Phillips (1963) showed experimentally that in rats on a diet containing 10 ppm DDT the utilization of orally administered carotene and vitamin A decreased. In a recent study with steers, Phillips & Hidiroglou (1965) fed a diet containing approximately 50 ppm DDT: although the liver vitamin A content was found to fall under these circumstances the authors concluded that the extent of the decrease was not of practical significance. The non-persistent MCPA (4-chloro-2-methylphenoxyacetic acid), when similarly used, had no effect on vitamin A storage.

J. Robinson & A. Richardson (1965, private communication) have shown that the concentration of the principal active ingredient of technical dieldrin, 1,2,3,4,10, 10-hexachloro-6,7-epoxy-1,4,4a,5,6,7,8,8a-octahydro-exo-1,4-endo-5,8-dimethano-naphthalene (HEOD), in the body fat of rats fed experimentally for 1 year, decreases exponentially with time when HEOD is subsequently withdrawn from the diet. The implication that a constant dietary intake will result in an equilibrium residue level in the bloodstream and body tissues has been confirmed experimentally.

Dieldrin residues in human fat in Britain average about 0·2 ppm, Robinson, Richardson, Hunter, Crabtree & Rees (1965) considering this to represent the equilibrium level for the national dietary intake. Egan *et al.* (1965) found similar levels and have also examined human milk, for which the average dieldrin and DDT levels appear to conform with the corresponding levels in the body fat. These levels do

not give cause for complacency; neither do they merit alarm. The Advisory Committee on Poisonous Substances used in Agriculture and Food Storage (now the Advisory Committee on Pesticides and Other Toxic Chemicals) (1964) recommended that the usage of aldrin and dieldrin in the United Kingdom should be curtailed, but that no restrictions should be placed on DDT and certain other persistent organo-chlorine pesticides, at least for the time being. In its report, the Advisory Committee concluded that organo-chlorine pesticides as a class could not be regarded as severe liver poisons. Nor, from available evidence, did they present a carcinogenic hazard to man. From rat experiments, even high residues of dieldrin or DDT in body fat give no indication of change in the metabolic activity of fatty tissue, though signs of poisoning can occur in these circumstances when food intake is reduced and fat has to be rapidly metabolized. Although perhaps aesthetically undesirable, there is no evidence that DDT residues found in human fat cause any injury. From the evidence which had been available on residues in food it was concluded that the highest dieldrin levels were attained only in isolated cases, under conditions which were seldom likely to arise; nevertheless, it was agreed that such dieldrin levels were undesirable and justified partial restriction of use. Durham (1963) concluded from the large amount of experimental evidence available that there is no significant hazard from DDT exposure in the United States although the possibility that subtle dangers remain to be discovered cannot be ruled out. The uses of DDT and certain other persistent organo-chlorine compounds in Britain will be reviewed in 1967.

Pesticide residues have not been studied systematically in animal feeding-stuffs but the possibility that the so-called chick oedema factor may derive from such has been considered by Friedman (1962). Large-scale poultry losses in which birds suffer from oedema accompanied by gross liver and kidney damage were first reported in the United States by Schmittle, Edwards & Norris (1958). Characteristic signs include droopiness, ruffled feathers and laboured breathing; mortality is high. The causative agent was eventually traced to certain batches of animal fats used in chicken food manufacture and the same factor was shown by Ames, Swanson, Ludwig & Brokaw (1960) to occur in some USP oleic acid. In 1960 the Food and Drug Administration issued a regulation (US Code of Federal Regulations, Title 21, sect. 121.1070) requiring oleic and stearic acids to be prepared from oils and fats 'free from chick oedema factor'. The chemical characterization of the factor has not proved to be an easy matter. Wootton & Alexander (1959) showed it to be a high molecular weight polycyclic hydrocarbon with at least one aromatic ring. Two separate compounds were later indicated by Wooton, Artman & Alexander (1962); in more recent work Wootton & Couchene (1964) have identified these from gas chromatographic and mass spectral evidence as isomeric hexachlorohexahydrophenanthrenes $C_{14}H_{10}Cl_6$, and a simple microcoulometric method for residue determination has been proposed by Higginbotham (1965). Flick, O'Dell & Childs (1964) have produced similar signs in chicks fed on normal rations to which were added 200–400 ppm of chlorinated biphenyl: they conclude that the latter is related to but not identical with the chick oedema factor. Friedman (1962) noted that hydro-

pericardium in chicks can also occur in poisoning from the insecticide chlordane. However, no toxic signs were found with oleic acid heated at 250° for long periods with chlordane, or with other organo-chlorine pesticides; nor did large doses of chlordane given to rats result in any similar response, although high residues have been noted in feeds associated with some outbreaks. Chlordane is a bridged-ring chlorinated pesticide having some structural resemblance to the dieldrin group of insecticides; it is used in Britain only to a limited extent. A condition similar to chick oedema disease has been recorded in the United Kingdom by Wannop & Chubb (1961) in which the trouble appeared to originate from a commercial concentrate of protein, vitamins, minerals and tallow. When samples of this material were examined, the chick oedema factor was not detected, although high TDE residues were noted; TDE is closely related to DDT, differing only in having two instead of three chlorine atoms attached to the α-carbon atom. DDT, DDE and TDE are all C_{14} compounds: the chick oedema factor ($C_{14}H_{10}Cl_6$), DDT ($C_{14}H_9Cl_5$) and DDE ($C_{14}H_8Cl_4$) differ one from the next only by the loss of HCl. Lee *et al.* (1964) claim to have shown that DDT is converted enzymically in vivo in rats into TDE ($C_{14}H_{10}Cl_4$). It is possible that isomerization of p,p′-DDE with subsequent HCl migration could be followed by ring closure leading eventually to a chlorinated phenanthrene derivative: however, no experimental proof of this sequence of changes at present exists.

The author is grateful to Dr J. M. Barnes, Mr R. B. A. Carnaghan, Dr R. Goulding, Dr W. J. Hayes, Dr J. Robinson and to various colleagues in the Laboratory of the Government Chemist for discussion on a number of important points.

REFERENCES

Abbott, D. C., Crosby, N. & Thomson, J. (1965). *Proceedings of the Society for Analytical Chemistry Conference.* (In the Press.)
Advisory Committee on Poisonous Substances Used in Agriculture and Food Storage (1964). *Review of Persistent Organochlorine Pesticides: Report and Supplementary Report.* London: HM Stationery Office.
Ames, S. R., Swanson, W. J., Ludwig, M. I. & Brokaw, G. Y. (1960). *J. Am. Oil Chem. Soc.* **17**, 10.
Backstrom, J., Hansson, E. & Ullberg, S. (1965). *Toxic. appl. Pharm.* **7**, 90.
Coffin, D. E. (1964). *Residue Rev.* **7**, 61.
Cueto, C. & Hayes, W. J. (1962). *J. agric. Fd Chem.* **10**, 366.
de Faubert Maunder, M. J., Egan, H. & Roburn, J. (1964). *Analyst*, **89**, 157.
Durham, W. F. (1963). *Residue Rev.* **4**, 34.
Dyte, C. E. (1963). *Fd Trade Rev.* **33**, no. 3, p. 35.
Edson, E. F., Sanderson, D. M. & Noakes, D. N. (1965). *Wld Pest Rev.* **4**, 36.
Egan, H. (1965). *J. Sci. Fd Agric.* **16**, 489.
Egan, H., Goulding, R., Roburn, J. & Tatton, J. O'G. (1965). *Br. med. J.* ii, 69.
Flick, D. F., O'Dell, R. G. & Childs, V. A. (1964). *Fedn Proc. Fedn Am. Soc. exp. Biol.* **23**, 406.
Friedman, L. (1962). *Feedstuffs*, 17 March.
Hamence, J. H. (1964). *J. Ass. publ. Analysts*, **2**, 99.
Hamence, J. H. (1965). *J. Ass. publ. Analysts*, **3**, 17.
Hayes, W. J., Durham, W. F. & Cueto, C. (1956). *J. Am. med. Ass.* **162**, 890.
Heath, D. F. & Vandekar, M. (1964). *Br. J. ind. Med.* **21**, 269.
Henderson, J. L. (1965). *Residue Rev.* **8**, 74.
Higginbotham, G. R. (1965). *J. Ass. off. agric. Chem.* **48**, 433.
Holden, A. V. (1962). *Ann. appl. Biol.* **50**, 467.
Laboratory of the Government Chemist. (1964). *Annual Report* 1963. London: HM Stationery Office.
Laboratory of the Government Chemist. (1965). *Annual Report* 1964. London: HM Stationery Office.

Lee, M., Harris, K. & Trowbridge, H. (1964). *J. Nutr.* **84**, 136.
Ludwig, A., Weis, J. & Korte, F. (1964). *Life Sci.* **3**, 123.
Marth, E. H. (1965). *Residue Rev.* **9**, 1.
Mills, P. A. (1963). *J. Ass. off. agric. Chem.* **46**, 672.
Moore, N. W. & Walker, C. H. (1964). *Nature, Lond.*, **201**, 1072.
Ortelee, M. F. (1958). *Archs ind. Hlth*, **18**, 433.
Phillips, W. E. J. (1963). *Can. J. Biochem. Physiol.* **41**, 1793.
Phillips, W. E. J. & Hidiroglou, M. (1965). *J. agric. Fd Chem.* **13**, 254.
Poonawall, N. H. & Korte. (1964). *Life Sci.* **3**, 1497.
Robinson, J., Richardson, A., Hunter, C. G., Crabtree, A. & Rees, H. J. (1965). *Br. J. ind. Med.* **22**, 220.
Rudd, R. L. (1964). *Pesticides and the Living Landscape.* Madison, Wis.: University of Wisconsin Press.
Schmittle, S. C., Edwards, H. M. & Morris, D. (1958). *J. Am. vet. med. Ass.* **132**, 216.
Wannop, C. C. & Chubb, L. G. (1961). *Vet. Rec.* **73**, 586.
Wootton, J. C. & Alexander, J. C. (1959). *J. Ass. off. agric. Chem.* **42**, 141.
Wootton, J. C., Artman, N. & Alexander, J. C. (1962). *J. Ass. off. agric. Chem.* **45**, 739.
Wootton, J. C. & Couchene, W. L. (1964). *J. agric. Fd Chem.* **12**, 94.

The action of chemical improvers on the lipids of flour

By N. W. R. Daniels, *Spillers Limited, Technological Research Station, Cambridge*

Introduction

Although lipids comprise no more than 1–2% of white (70% extraction) flour, the nutritional significance of the flour lipids has received much attention in recent years on account of their relatively high content of essential fatty acids (EFA). Though direct evidence concerning human requirements for these factors is scanty, it has been said that EFA deficiency in the human diet may contribute towards atheroclerosis and coronary thrombosis. Moreover, the readiness with which these polyunsaturated fatty acids may be oxidized led to the suggestion that oxidative treatment of flour might seriously impair its nutritive value in the human diet (Sinclair, 1956). It was this latter possibility that prompted workers in both this country and America to examine the lipids of flour with the object of evaluating quantitatively any changes that may occur in the EFA content as a result of chemical treatment.

Fisher, Ritchie & Coppock (1958*a*,*b*) examined the effect of the improvers, chlorine dioxide, potassium bromate, ascorbic acid, ammonium persulphate, and the pigment bleach, benzoyl peroxide, on the lipids of flour, dough and bread at treatment levels mostly twice and twenty times those currently used with present-day white flours. In no instance was it possible to demonstrate a significant change in the content of EFA as a result of the use of oxidative improvers. These results were confirmed by Gilles, Anker, Wheeler & Andrews (1958) who included infrared spectrophotometry in their examination. The possibility that contamination of chlorine dioxide with chlorine gas may cause significant EFA destruction in bread-making flours was later investigated (Coppock, N.W.R. Daniels & Russell-Eggitt, 1960) using gas–liquid chromatography as a more direct measure of fatty acid composition than the alkaline isomerization method used by previous workers. Whereas contamination of chlorine dioxide with up to 30% chlorine gas was without effect on EFA, the use of increasing levels of treatment with pure chlorine gas (a treatment used in the production of certain speciality flours) brought about some loss of EFA. At the