How Essential Are Essential Fatty Acids?

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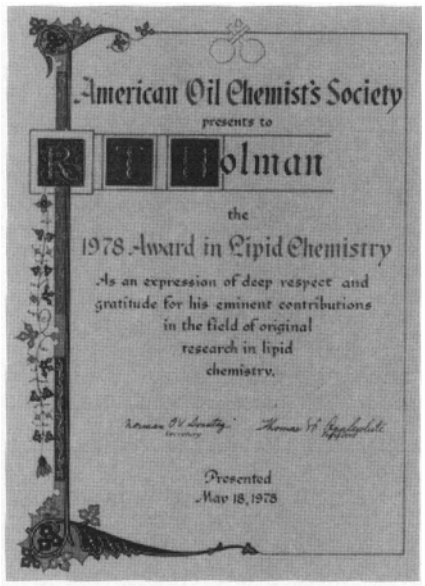
Plenary Lecture
St. Louis, MO, May 16, 1978
Award in Lipid Chemistry 1978



Members and guests of the American Oil Chemists' Society, Ladies and Gentlemen:

This morning at the meeting of the Publications Committee I was asked what I was to preach about today. Until then I had not thought about this lecture as a sermon, but it well might be. In a paraphrase of a song written in the idiom of Texas where I once sojourned: "Brethren and sistern, a preachment you're about to receive on rat tails, scaly skin, and the lipids of bleed." But seriously, the story to be told is dependent upon factors which are usually treated in sermons. The text for the day could well be "Train up a child in the way he should go, and when he is old, he will not depart from it." I wish to indicate in my credits how this text might apply.

Any accomplishment leading to an honor such as I receive here cannot be made alone. There must be considerable input from the outside, and I wish first to record my gratitude to Divine Providence for guiding my progress on this pilgrimage. It was absolutely necessary, for example, that Providence select for me the proper Parents. I am grateful to them for teaching and demonstrating a philosophy of life which could be emulated. They have sacrificed for me, they encouraged my education, and they got me started on the road.

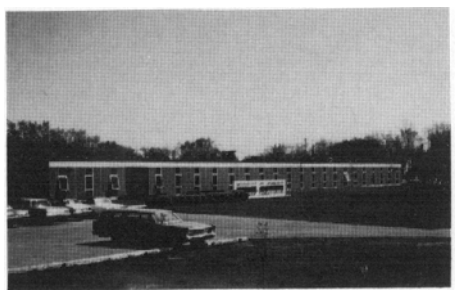
One also must have the proper Partner. My partner Karla has maintained a supportive home atmosphere for our family for more than 35 years and has constantly encouraged me in my creative work. With her, a life of adventure is possible. Whatever honor we receive, we share equally.

One must also choose his Professor wisely. Professor George Burr gave me the inspiration to work in the field of lipids back in 1941. He encouraged me to investigate more than one project at a time, he always gave me sound advice, and he opened my eyes to opportunity.

The Award in Lipid Chemistry is being given this year for our studies relating to the structural and quantitative requirements of essential fatty acids. This work could not have been accomplished without a host of Participants, all of my colleagues throughout the past 30 years. Those who have contributed most heavily to the measurement of requirement and the study of quantitative relationships are listed below.

Carl Widmer	*1948-1950*	*Joseph J. Rahm*	*1960-1964*
Herbert Hayes	*1952-1972*	*Hilda F. Wiese*	*1963-1964*
Eldon G. Hill	*1953-present*	*Balwant Ahluwalia*	*1963-1965*
James J Peifer	*1954-1956*	*Cecelia Pudelkewicz*	*1964-1967*
Erik Aaes-Jørgensen	*1956-1958*	*Kirsten Christiansen*	*1966-1968*
William O. Caster	*1960-1970*	*Yves Marcel*	*1966-1969*
Hans Mohrhauer	*1960-1962*	*John R. Paulsrud*	*1968-1970*
	1965-1967	*Susan Johnson*	*1973-present*

Another required factor contributing to the ultimate accomplishment is a Purpose for the work. My program has been to study essential fatty acids, to attempt to learn what makes them essential, and to estimate how much is needed by animals and by man.



To accomplish a program of research, one also needs a Place, an environment which permits ideas to develop and solutions to be found. I am grateful to The Hormel Institute and its founders who provided an academic atmosphere in which such erudite academic researches as I summarize for you today could be done. The account will show that fundamental studies lead to very practical results. This work could only have been done in a place of freedom.

I consider myself extremely fortunate that all the necessary conditions for a successful outcome were so nearly optimal. For these many conditions, which were not subject to my control, I cannot take credit, and the honor I am shown here must be shared by the many who prepared the way for me and by those who assisted me along the way.

In 1929 Burr and Burr (1) described a deficiency syndrome induced by feeding rats a fat-free diet. The signs of the deficiency included scaliness of skin (Fig. 1), necrosis of the tail, diminished growth, degeneration of kidneys, and failure to reproduce. The syndrome was traced to the deficiency of a single fatty acid in the diet, and the Burrs found that this acid was linoleic acid (2). In Figure 2, several of the symptoms are arranged diagrammatically. The normal growth rate shown by the dotted line is to be contrasted with the diminished growth rate of animals fed a diet deficient in essential fatty acids. After a few months on a fat-free diet, scaliness of skin such as shown in Figure 1 begins to appear, it increases in magnitude for some months, and sometimes diminishes to some degree in old animals. The necrosis of the tail appears later than do the skin signs. Normally yellow-brown skin pigment diminishes early in the deficiency, and the rat skin of a deficient animal is bluish white. The caloric consumption increases early in the deficiency to approximately double the normal intake. Water consumption also increases dramatically, but urine volume decreases. The permeability of the skin to water has been shown to be much increased, and the animal consumes more water and food to restore the water loss and to compensate for the loss of heat due to evaporation. Many other morphologic and physiologic abnormalities are also induced by the deficiency of EFA and have been reviewed (3).

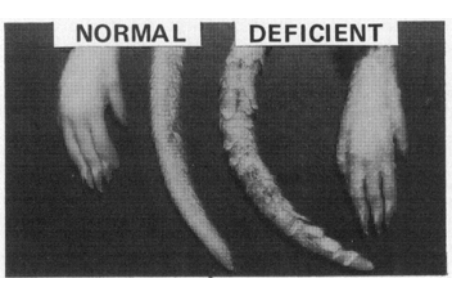


FIG. 1.

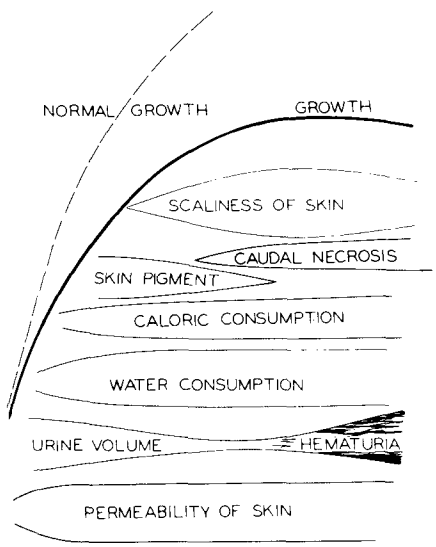


FIG. 2. Essential fatty acid deficiency symptoms in the rat (one year).

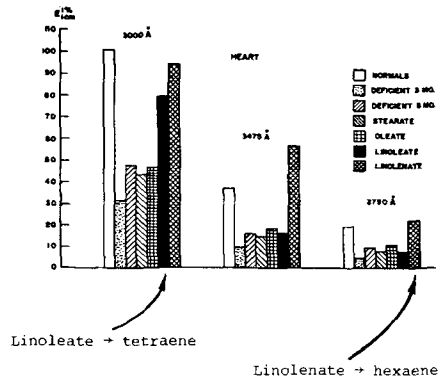


FIG. 3.

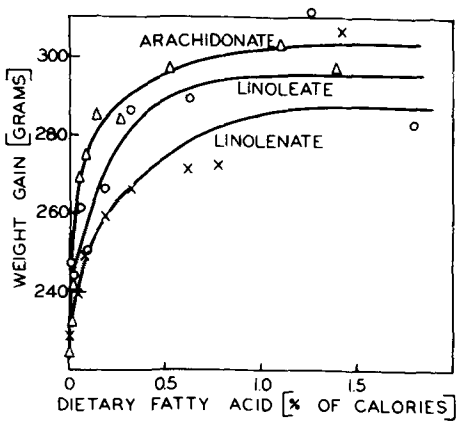


FIG. 4. EFA dose level and weight gain.

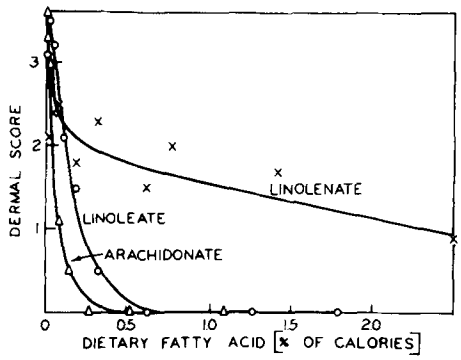


FIG. 5. EFA dose level and dermatitis.

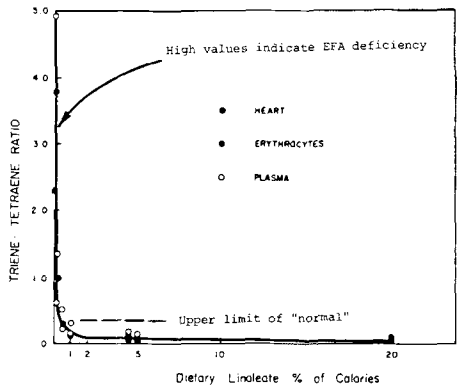


FIG. 6. Test for EFA status.

The last piece of work on essential fatty acids which George Burr and his students completed at the University of Minnesota was the comparison of the fatty acid composition of lipids of vital tissues from rats fed fat-deficient diet, corn oil, or fish oil (4). In rats fed fat-deficient diet, the lipids of tissues contained an increased content of trienoic acids measurable by alkaline isomerization, and there was a decrease in the tetraenoic, pentaenoic, and hexaenoic acids. In the rats fed corn oil, tissue lipids had a normal amount of tetraenoic acid, and in the rats fed fish oil, the tissue lipids were very rich in pentaenoic and hexaenoic acids. When I began my independent work on the essential fatty acids at Texas A&M, I decided that the way to more accurately describe this interesting phenomenon would be to feed single purified fatty acids, and to measure their effects upon composition of fatty acids of tissue lipids. This task was undertaken by Carl Widmer, my first graduate student (5). The crucial results of that experiment are shown in Figure 3. When we fed linoleic acid, we found that it stimulated the amount of tetraenoic acid in tissue lipids, but that it did not increase the pentaenoic nor the hexaenoic acids. The significance of this observation is that linoleic acid is the precursor of arachidonic acid. Totally saturated stearic acid and monounsaturated oleic acid had no significant effect upon the contents of tetraenoic, pentaenoic, and hexaenoic acids in tissue lipids. However, when we fed linolenic acid, we found increases in the hexaenoic acid which were not induced by any other dietary fatty acid. Our contribution to the then rather meager knowledge of metabolism of polyunsaturated acids was that linoleic acid is the precursor of arachidonic acid and linolenic acid is the precursor of the 6 double-bonded acid.

Somewhat later in the work done by Mohrhauer in our laboratory at The Hormel Institute, we studied the quantified effects of graded dose levels of dietary linoleic, linolenic, and arachidonic acids upon fatty acid composition of lipids of liver (6), heart (7), and other tissues. We found that linoleic acid stimulated growth much better than linolenic acid, and that arachidonate, the 4 double-bonded acid, stimulated growth most (Fig. 4). When we studied the effect upon the dermal symptoms, the scaliness of skin and tail, a similar phenomenon was found. Arachidonic acid eliminated dermal symptoms at much lower dietary intake than did linoleate (Fig. 5). Arachidonate was two to six times more effective than linoleate, but linolenic acid never really relieved skin symptoms totally, even at high levels of intake. This confirmed in a quantified manner what had been suspected for quite a long time, that linolenic acid is not fully effective as an essential fatty acid.

The study of effects of graded dose levels of linoleic acid led to the setting of quantitative requirements for essential fatty acid based upon biochemical parameters. In a nutritional study involving several kinds of dietary fat and several levels of fat intake, we had found that the ratio of the triene to tetraene decreased dramatically as the dietary content of linoleic acid increased. Figure 6 shows that the triene:tetraene ratio remained low (normal) at intakes of linoleic acid above about 1% of calories, but rose dramatically higher at lesser intakes (8). A ratio of about 0.4 or less was considered normal, and any values higher than that, abnormal. When Mohrhauer and I studied the effects of varying dose levels of single pure essential fatty acids using the newly available analysis by gas chromatography, we found that the 20 carbon acid with 3 double bonds (20:3ω9), which was the substance measured as triene by alkaline isomerization, decreased as we added more linoleic acid to the diet (Fig. 7). Simultaneously, the arachidonic acid (20:4ω6) formed from linoleic acid rose strongly. The new ratio, 20:3ω9/20:4ω6, could be used in the same way as the old triene:tetraene ratio for setting the requirement for essential fatty acids, and it has the advantage of greater precision.

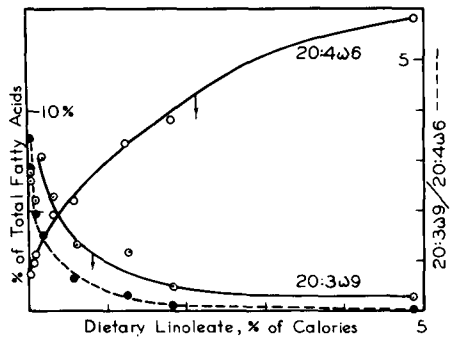


FIG. 7. Male rats–total liver lipids.

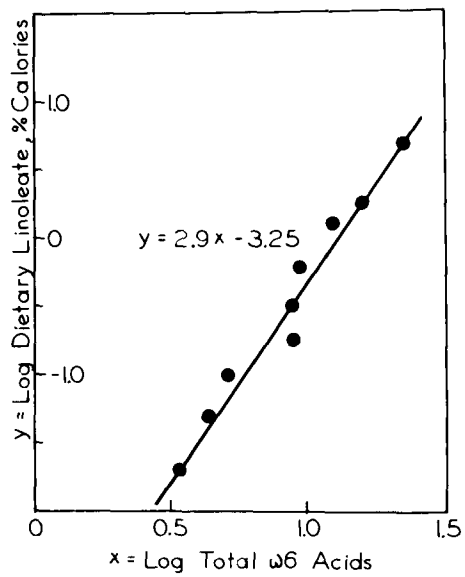


FIG. 8. Male rats–liver total lipids

Seventy percent of the maximum achievable change in a biochemical measure may be considered normal and adequate (9). In the study shown in Figure 7, this degree of change is provided by a little more than 2% of calories of linoleate for the synthesis of 20:4ω6, and by a little less than 1% of calories of linoleate for the suppression of 20:3ω9. These values are indicated by the arrows. This suggested that approximately 1% of calories of the diet in the form of linoleate met the essential fatty acid requirement. This type of data also provides mathematical expressions for calculating the intake of linoleate an individual may have had in recent time (10). If the logarithm of the total ω6 acids in tissue lipids (all of the acids formed from the essential fatty acid, linoleic acid) is plotted against the logarithm of linoleate fed in the diet, a straight line is obtained (see Fig. 8). This is perhaps the best estimate of the intake of linoleic acid of an individual. Another calculation relating dietary intake of linoleate to the sum of dienoic + tetraenoic - trienoic acids in tissue lipids has been derived for infants (11) and male adults (12), permitting an estimate of human intake of essential fatty acid from serum analysis.

The effects of dietary nonessential fatty acid upon the metabolism of essential fatty acids have also been studied. Figure 9 indicates that as dietary saturated fat is increased, the caloric efficiency and weight gain are decreased, and that the triene:tetraene ratio is elevated (13). This is especially true at low linoleic acid levels. This suggests that high proportions of saturated fat in the diet increase the requirement for essential fatty acid. This kind of study was also done with monoenoic acids yielding the same conclusion (14). Figure 10 shows that with dietary linoleate held constant, an increase of dietary linolenate suppresses the amount of metabolic products normally formed from linoleic acid in the heart, brain, and liver (15). Dietary linoleic acid was also observed to suppress the metabolism of dietary linolenic acid (16). This indicates that there is a strong competitive interaction between the acids of the family of linoleic acid and the acids of the family of linolenic acid. Study of this phenomenon in subcellular systems with the chain elongation reaction confirmed that linolenic acid suppresses the metabolism of dietary linoleic acid (17). Another research group demonstrated a similar effect for desaturation (18). These studies at the enzyme level led to quantitative studies delineating the preferred pathway for metabolism of linoleate to arachidonate (19).

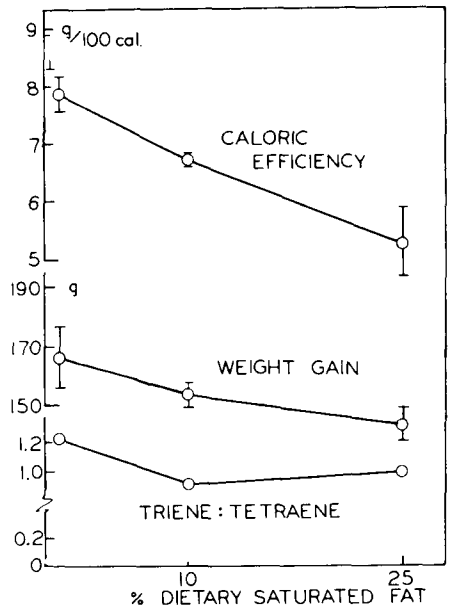


FIG. 9.

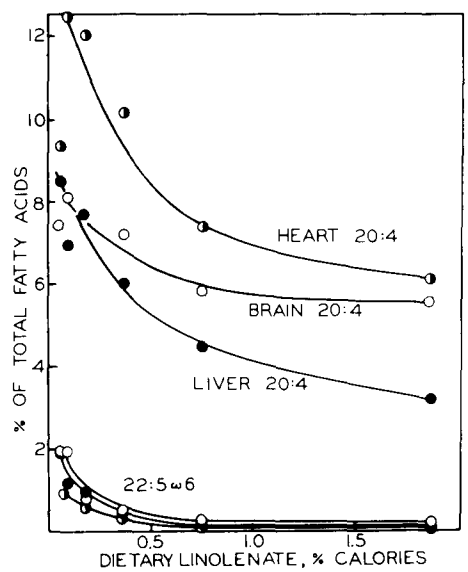


FIG. 10. Competition with linoleate.

Next I would like to consider what meaning essential fatty acids may have in human nutrition. In this area, Dr. Arild Hansen, a pediatrician trained under Professor Burr, made the initial observations on essential fatty acid deficiency in children at the University of Minnesota Medical School. Figure 11 is an example of an eczema patient that

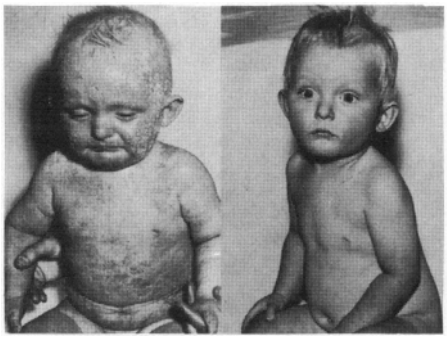


FIG. 11.

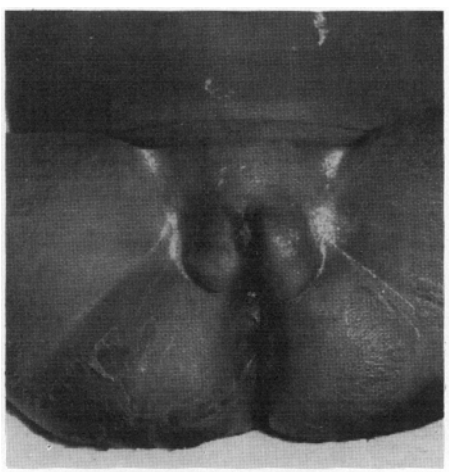


FIG. 12.

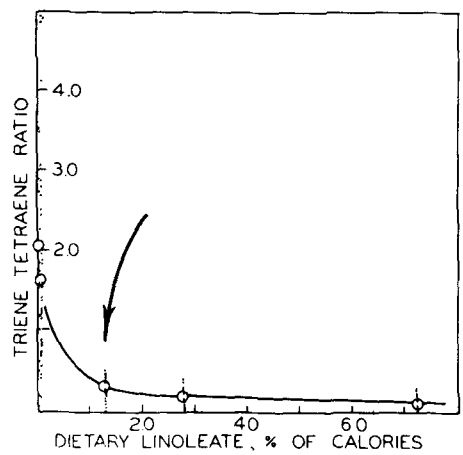


FIG. 13. Minimum EFA requirement (infants 2-4 months).

came to his clinic with intractable eczema. The child was given 28% of calories as lard and, after a few weeks, the dermatitis was gone. While he was at the University of Texas Medical School, Dr. Hansen made a study of five different infant formula preparations and found them to vary quite drastically in essential fatty acid content. At that time, it was common practice to feed infants a formula prepared from skim milk sweetened with sucrose, and Hansen found that on such a dietary regimen, eczema occurred frequently (20). Figure 12 shows a case of eczema produced by feeding the skim milk-sucrose diet which contains very little essential fatty acid. Giving the child linoleate prepared in our laboratory cured the dermatitis. Data from Hansen's study involving over 400 children were

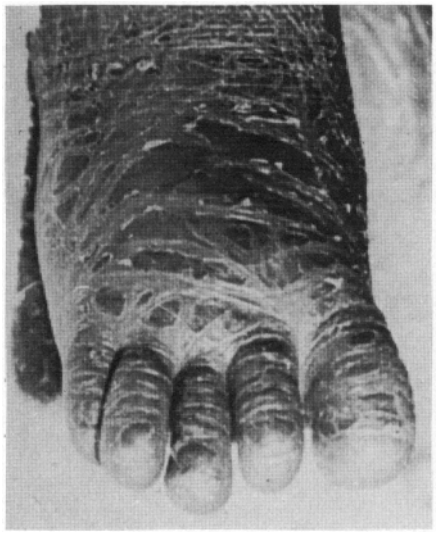


FIG. 14.

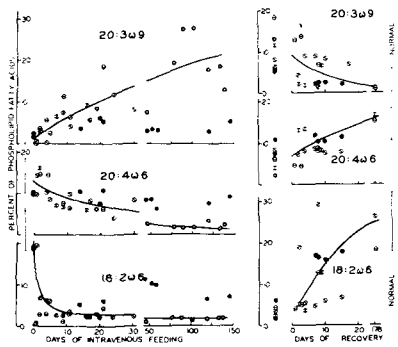


FIG. 15.

recalculated following relationships deduced from our studies with rats. When we plotted the triene:tetraene ratio against dietary input of essential fatty acids in infants, we got the same curve with the break at the same place as we had found for rats (11), suggesting that the minimum requirement for essential fatty acid in infants is in the order of 1% of calories (see Fig. 13). In the very recent WHO-FAO consultation on fat in human diet, the human requirement was placed at 3% of calories to insure infant growth and provide for lactation (21).

In 1969, in a consultation with Dr. Whitten of Detroit, we encountered a case of an overt essential fatty acid deficiency in an infant (22). The child had experienced a volvulus on the first day of life, which caused death of the bowel by strangulating its blood supply. Exploratory surgery on the second day of life revealed the condition, the necrotic bowel was removed, and the duodenum was anastomosed to the colon, leaving almost no absorptive intestine. The infant was maintained on a fat-free intravenous preparation, and after three months it had developed the generalized scaly dermatitis shown in Figure 14. Serum lipid analyses made serially throughout the onset of this deficiency disease revealed that the 20:3ω9/20:4ω6 ratio rose dramatically. This child was the impetus for a study of several children in the same clinic who had experienced a malfunction of the intestine called "failure to thrive" because its cause is not fully understood. Intravenous nutrition was provided temporarily to support life until intestinal function could develop. These children had changes in serum fatty acid patterns which indicated that they developed essential fatty acid deficiency while they were on intravenous feeding, but when they could be given food by mouth, every one recovered. These studies indi-

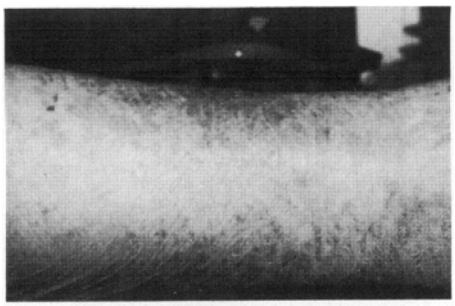


FIG. 16.

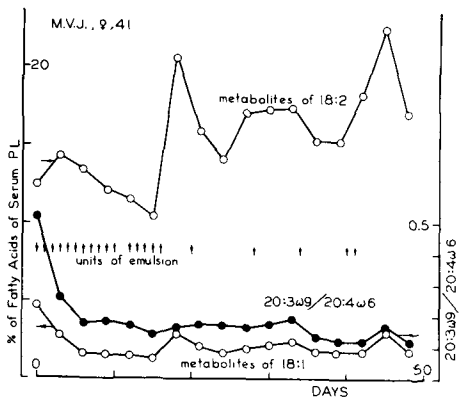


FIG. 17.

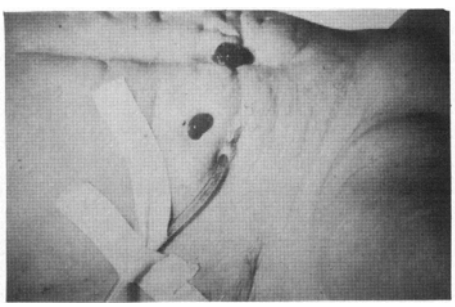


FIG. 18.

cated that fat-free intravenous feeding induces essential fatty acid deficiency in humans, and that the deficiency is reversible by normal food (Fig. 15).

This phenomenon is not limited to children. Figure 16 shows a case of a 78-year-old woman who experienced a mesenteric infarction, underwent the total surgical removal of the small bowel, and within 6 weeks developed a flaky skin (23). Consequently, an analysis of serum phospholipid fatty acids was made, and the 20:3ω9/20:4ω6 ratio was 0.6. At that time we did not consider this value as alarming in comparison to 0.4, the accepted upper limit of normalcy.

Some time later we had the opportunity to make simlilar studies on a woman who had been maintained on fat-free intravenous nutrition but who was subsequently given intravenous emulsion containing essential fatty acid (24). During her recovery from deficiency, we were able to follow changes in serum lipids. The total metabolites of linoleic acid increased to the normal range (Fig. 17). A cyclic variation was noticed, perhaps related to the menstrual cycle. In this study, the 20:3ω9/20:4ω6 ratio, which started near 0.5, dropped to a very low value near 0.1. The intravenous fat emulsion had corrected the essential fatty acid deficiency.

Caldwell and others studied an infant who underwent repeated corrective surgery to its intestine during its first

FIG. 19.

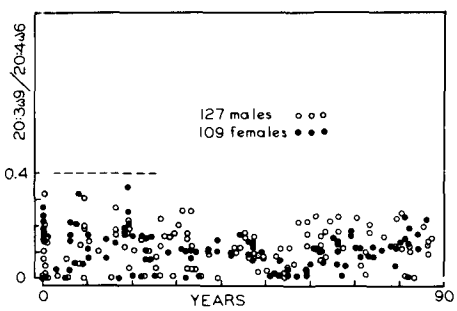


FIG. 20. Triene/tetraene human serum phospholipids (127 moles ○○○; 109 females ●●●).

weeks of life, and who was maintained by fat-free intravenous feeding (25). After its last surgery, the wounds did not heal (Fig. 18) until after the infusions of intravenous fat emulsion containing essential fatty acid were made. Essential fatty acids are required for the synthesis of normal tissue, and their deficiency impedes wound healing because polyunsaturated acids are required for synthesis of normal tissue lipids.

The application of oil containing essential fatty acid to the skin of EFA-deficient adults has been reported to be effective in relieving EFA deficiency (26). Some of my colleagues at the University of Minnesota Hospitals attempted to confirm this in infants without success, and one of their patients treated by applying oil to the skin is shown in Figure 19. This child was given sunflower seed oil cutaneously, but it did not cure the deficiency, as measured by serum lipid analysis or relief of the dermatitis (27). Perhaps the requirement of EFA for rapid growth of an infant is too great to be met by the amount which could be absorbed through the skin.

In recent years, our laboratory has compiled some values for fatty acid composition of human serum lipids to which suspected cases of essential fatty acid deficiency might be compared. Serum samples remaining after diagnostic procedures at our local hospital were analyzed, and patients that were known metabolic cases were excluded. Serum lipids were separated, and fatty acid compositions were measured. The ratios of 20:3ω9/20:4ω6 in serum phospholipids are shown in Figure 20 (28). Note that not one individual had a ratio as high as 0.4. The average ratio was about 0.1 with a standard deviation of 0.1. Thus, the upper limit of normalcy should be about 0.2. In the light of this observation, the cases of the 78-year-old woman who had a

TABLE I

Fatty Acid Pattern of Serum Phospholipids
Extreme EFA Deficiency Following Bowel Resection
Female, S.W., age 93 days

FATTY ACIDS	%	NORM. %	S.V.
12:0	0.0	0.1	0.1
14:0	0.0	0.2	0.2
14:1	0.0	0.1	0.2
16:0	26.8	26.1	4.6
16:1W7	4.1	1.2	0.8
16:2	0.0	0.1	0.2
18:0	11.9	14.0	2.4
18:1W9	23.0	12.8	2.4
18:2W6	1.8	18.9	4.6
18:3W6	0.0	0.4	0.4
18:3W3	0.0	0.2	0.4
20:2W9	0.0	0.2	0.3
20:2W6	0.0	0.3	0.3
20:3W9	27.5	1.7	0.9
20:3W6	0.0	3.7	1.4
20:4W6	1.5	12.7	2.9
20:4W3	0.0	0.3	0.4
20:5W3	1.6	1.4	0.7
22:4W6	1.7	1.9	0.9
22:4W3	0.0	0.7	1.3
22:5W6	0.0	0.5	0.7
22:5W3	0.0	0.6	0.5
22:6W3	0.0	2.0	1.3
OTHER	0.1		
R INDEX	0.532	1	
20:3W9/20:4W6	18.33	0.132	0.070
18:2W6+20:4W6-20:3W9	24.2	29.9	4.2
W6 METABOLITES	3.2	19.6	4.0
TOTAL W6 ACIDS	5.0	38.5	3.9
W3 METABOLITES	1.6	4.9	2.4
TOTAL W3 ACIDS	1.6	5.1	2.4
W9 METABOLITES	27.5	1.9	1.0
TOTAL W9 ACIDS	50.5	14.7	2.7
SATURATED ACID	38.7	40.4	4.2
MONOENE ACIDS	27.1	14.2	2.8
DOUBLE BOND IND	1.200	1.518	0.160
18:2W6/20:4W6	1.200	1.670	0.621
TOTAL PUFA	34.10	45.45	4.397

DIAGNOSIS:
FATTY ACID PATTERN ABNORMAL
EFA STATUS: DEFICIENT (T/T), DEFICIENT (W6), DEFICIENT (D-T+T)
LINOLEATE UTILIZATION NORMAL, W3 ACIDS LOW
PUFA LOW, SATURATED ACIDS NORMAL

ratio of 0.6 and the 41-year-old woman who had a ratio of 0.5 at the beginning of the intravenous fat emulsion were truly EFA deficient.

In recent years, we have developed computer programs to help in the diagnosis of essential fatty acid deficiency. Using the population base mentioned above, the programs compare the fatty acid compositions of serum lipids from individuals to those of a normal population, and provide a tentative diagnosis. In Table I the extremely deficient child (Fig. 14) is taken as an example, and the fatty acid pattern of serum phospholipids at 93 days of age is compared against normal values calculated for that age. Several parameters are calculated from these data, including the 20:3ω9/20:4ω6 ratio, the total ω6 acids, and the sum of 18:2ω6 + 20:4ω6 - 20:3ω9, and these too were compared to the normal values for these parameters. This rapid diagnostic help will be used to assay essential fatty acid status of populations and individuals.

Using this approach, a group of children who had cystic fibrosis was compared to normal children. Comparison of the values for the 20:3ω9/20:4ω6 ratio, the total polyunsaturated acids, the total ω6 acids, the arachidonic acid, the monoenoic acids, and the saturated acids all revealed that for the cystic fibrotic children, these parameters were about one standard deviation away from the normal, in the direction of essential fatty acid deficiency (28). This suggests that cystic fibrosis involves an aberration in essential fatty acid metabolism which is the equivalent of marginal essential fatty acid deficiency.

In cases of chronic malnutrition from Argentina, a similar result was found (29). The differences from normal were again in the direction of essential fatty acid deficiency. The double bond index was less than for normals; the 20:3ω9/20:4ω6 ratio was three times higher than normal; the total polyunsaturated acids were less than normal; the ω6 acids were less than normal; arachidonic acid content was half of normal; monoenoic acids were higher than normal; and saturated acids were higher than normal. Thus, chronic malnutrition involves a marginal

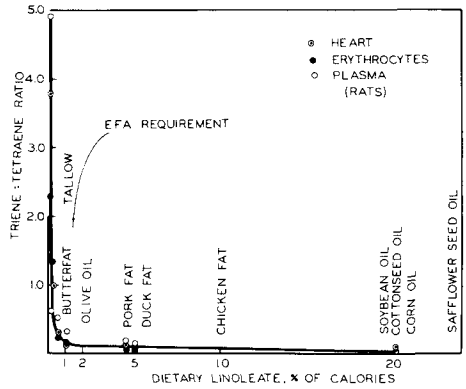


FIG. 21.

deficiency of essential fatty acids.

The question always arises – are our common food fats adequate in essential fatty acids? In Figure 21, I have taken the triene:tetraene ratio curve shown earlier and superimposed over it the names of several of our dietary fats at positions which indicate the level of essential fatty acids provided if they were the sole source of fat and were fed at 40% of dietary calories. Corn oil, cottonseed oil, and soybean oil provide approximately 20% of calories if consumed at that level. Olive oil, butter, and tallow are near the break of the curve indicating that these dietary fats would provide the bare minimum of essential fatty acids if eaten alone. However, normal dietary practice involves mixtures of fats from animal and vegetable sources. Thus, normal humans eating a variety of foods are not likely to become EFA deficient. However, there are many factors which can move an individual toward a deficiency of essential fatty acids or which may increase the requirements. High dietary saturated fat or monounsaturated fat, disease, and malnutrition all move the individual in the direction of EFA deficiency. Genetic defects in metabolism of essential fatty acids might contribute to an essential fatty acid deficiency in a functional sense. Because of such factors, I think that marginal essential fatty acid deficiency exists in the human population. We now know approximately how much essential fatty acid is required by normal individuals, and that common diets can provide this need for the average person. Nevertheless, I expect that the future will reveal that many of man's diseases involve abnormalities in the metabolism of essential fatty acids.

ACKNOWLEDGMENTS

Financial support for this program of studies has been provided by Public Health Service Grant AM 04524; Public Health Service Research Grant HL 08214 from the Program Projects Branch, Extramural Programs, National Heart, Lung and Blood Institute; and by The Hormel Foundation.

REFERENCES

1. Burr, G.O., and M.M. Burr, J. Biol. Chem. 82:345 (1929).
2. Burr, G.O., and M.M. Burr, Ibid. 86:587 (1930).
3. Holman, R.T., Prog. Chem. Fats Other Lipids 9:279 (1971).
4. Rieckehoff, I.G., R.T. Holman, and G.O. Burr, Arch. Biochem. 20:331 (1949).
5. Widmer, C., and R.T. Holman, Ibid. 25:1 (1950).
6. Mohrhauer, H., and R.T. Holman, J. Lipid Res. 4:151 (1963).
7. Mohrhauer, H., and R.T. Holman, Biochem. Probl. Lipids, BBA Library 1:446 (1963).
8. Holman, R.T., J. Nutr. 70:405 (1960).
9. Caster, W.O., P. Ahn, E.G. Hill, H. Mohrhauer, and R.T. Holman, Ibid. 78:147 (1962).
10. Holman, R.T., in "Handbook of Nutrition," Vol. 2, Section E, CRC Press, Cleveland, OH (In press).
11. Holman, R.T., W.O. Caster, and H.F. Wiese, Am. J. Clin. Nutr. 14:70 (1964).
12. Holman, R.T., W.O. Caster, and H.F. Wiese, Ibid. 14:193 (1964).

13. Peifer, J.J., and R.T. Holman, J. Nutr. 68:155 (1959).
14. Mohrhauer, H., J.J. Rahm, J. Seufert, and R.T. Holman, Ibid. 91:521 (1967).
15. Holman, R.T., and H. Mohrhauer, Acta Chem. Scand. 17:S84 (1963).
16. Rahm, J.J., and R.T. Holman, J. Nutr. 84:15 (1964).
17. Mohrhauer, H., K. Christiansen, M.V. Gan, M. Deubig, and R.T. Holman, J. Biol. Chem. 242:4507 (1967).
18. Brenner, R.R., and R.O. Peluffo, Ibid. 241:5213 (1966).
19. Marcel, Y.L., K. Christiansen, and R.T. Holman, Biochim. Biophys. Acta 164:25 (1968).
20. Söderhjelm, L., and A.E. Hansen, Pediatr. Clin. North Am. 9:927 (1962).
21. Dietary Fats and Oils in Human Nutrition. FAO Food and Nutrition Paper 3, report of an expert consultation jointly organized by the Food and Agriculture Organization of the United Nations and the World Health Organization, Rome, Sept. 21-30, 1977. Food and Agriculture Organization of the United Nations, Via delle Terme di Caracalle, 00100 Rome, Italy.
22. Paulsrud, J.R., L. Pensler, C.F. Whitten, S. Stewart, and R.T. Holman, Am. J. Clin. Nutr. 25:897 (1972).
23. Varco, R.L., M.A. Shea, J.R. Paulsrud, S. Stewart, and R.T. Holman (unpublished data, 1970).
24. Holman, R.T., and S. Johnson (unpublished data, 1975).
25. Caldwell, M.D., H.T. Jonsson, and H.B. Othersen, Pediatrics 81:894 (1972).
26. Press, M., P.J. Hartop, and C. Prottey, Lancet 1:594 (1974).
27. Hunt, C.E., R.R. Engel, S. Modler, W. Hamilton, S. Bissen, and R.T. Holman, J. Pediatr. 92:603 (1978).
28. Holman, R.T., S. Johnson, and J.D. Lloyd-Still (unpublished data, 1977).
29. Holman, R.T., S. Johnson, O. Mercuri, M.E. de Tomas, H.J. Itarte, and M.A. Rodrigo (unpublished data, 1977). •

Call for nominations

1979 Honored Student Awards

Nominations are now being solicited for the 1979 AOCS Honored Student Awards. Graduate students at any North American institution of higher learning, in any area of science dealing with fats and lipids, who are doing research toward an advanced degree, and who are interested in the areas of science and technology fostered by this Society, are eligible. Recipients must remain registered graduate students and not have received the advanced degree being sought nor have begun career employment before the meeting at which the award is to be presented.

Nominating professors, who must be members of the Society, may submit one nomination each year. Up to three awards may be granted to candidates for masters degree; up to five awards may be granted to doctoral students. Each nominee must submit an abstract of a paper and be prepared to present it at the AOCS Annual Meeting if chosen for an award.

The award provides funds equal to travel costs, plus $75.00 for attendance at the AOCS 1979 Annual Meeting to be held in San Francisco on April 29-May 3, 1979. Recipients receive a complimentary registration.

Nominating forms may be obtained from AOCS Headquarters, 508 S. Sixth St., Champaign, IL 61820. Completed nominations should be returned before November 1, 1978, to Dr. Joyce L. Beare-Rogers, Chief, Nutrition Research, Bureau of Nutritional Sciences, Health Protection Branch, Department of Health and Welfare, Ottawa, Canada, K1A OL2.

Remember the deadline: November 1, 1978

Award in Lipid Chemistry

In April 1964, the Governing Board of the American Oil Chemists' Society established an Award in Lipid Chemistry under the sponsorship of the Applied Science Laboratories, Inc., State College, PA. Previous awards were presented as follows: Erich Baer, August 1964; Ernest Klenk, October 1965; H.E. Carter, October 1966; Sune Bergstrom, October 1967; Daniel Swern, October 1968; H.J. Dutton, October 1969; E.P. Kennedy, September 1970; E.S. Lutton, October 1971; A.T. James, September 1972; F.D. Gunstone, September 1973; P.K. Stumpf, September 1974; W.O. Lundberg, September 1975; George Popjak, May 1977; and Ralph Holman, May 1978.

The award consists of $2500 accompanied by an appropriate certificate. It is planned that the fifteenth award will be presented at the AOCS Annual Meeting in San Francisco, April 29-May 3, 1979.

Canvassing Committee Appointees

Policies and procedures governing the selection of award winners have been set by the AOCS Governing Board. An Award Nomination Canvassing Committee is appointed; chairman is Earl G. Hammond. The function of this committee is to solicit nominations for the fifteenth award. Selection of the award winner will be made by the Award Committee whose membership will remain anonymous.

Rules

The rules prescribe that nominees will have been responsible for the accomplishment of original research in lipid chemistry and must have presented the results thereof through publication of technical papers of high quality. Preference will be given to individuals who are actively associated with research in lipid chemistry and who have made fundamental discoveries that affect a large segment of the lipid field. For award purposes, the term "lipid chemistry" is considered to embrace all aspects of the chemistry and biochemistry of fatty acids, of naturally occurring and synthetic compounds and derivatives of fatty acids, and of compounds that are related to fatty acids metabolically or occur naturally in close association with fatty acids or derivatives thereof. The award will be made without regard for national origin, race, color, creed, or sex.

Letters of nomination together with supporting documents must be submitted in octuplicate to Earl G. Hammond, Department of Food Technology, Iowa State University, Ames, IA 50010, USA, before the deadline of December 1, 1978. The supporting documents will consist of professional biographical data, including a summary of the nominee's research accomplishments, a list of his publications, the degrees he holds, together with the names of the granting institutions, and the positions held during his professional career. There is no requirement that either the nominator or the nominee be a member of the American Oil Chemists' Society. In addition, letters from at least three other scientists supporting the nomination must be submitted in octuplicate. •

Remember the deadline: December 1, 1978