LECITHIN FEEDING IN THE SYNDROME OF PSORIASIS

II. Chemical Studies of the Relationship of Lecithin Feeding to Fat Metabolism[1]

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In a previous communication one of us (L. G.) (1) has reported the feeding of soybean lecithin mixtures to be of value in some cases of psoriasis. Soybean lecithin was used because of the lipotropic character of the lecithin, and the fact that soybean lecithin was so easily available. This mixture is stable and is reported to contain lecithin 20%, cephalin 20%, oil 30%, phytosterols 2%, inositol and allied compounds 15%, and carbohydrates 10%. Inositol is also a lipotropic substance. Since that report some 16 additional patients have been treated for varying periods of time with improvement in approximately 60% of them. In most instances the patients receiving the lecithin were also on a fat poor diet. These patients received no vitamin mixtures in addition to the lecithin, although supplementation of lecithin therapy with vitamin B complex has been suggested for those patients who do not respond to simple lecithin feeding. There were no reactions to this soybean lecithin. Gross and Kesten (2), and Sullivan and Nicholls (3) have studied the effect of lecithin in relation to vitamin research.

In an effort to secure a better controlled study of patients with this difficult disease and to examine, after a fashion, their metabolism of fats, five patients with psoriasis of varying severity were observed in the hospital during lecithin feeding. It is important to note that these patients were also on a low fat diet, approximately 40 gms per day. At present the study has been completed on only four patients.

The following blood lipid chemistry studies were made on these patients:

Fasting—cholesterol, cholesterol esters, and phosphatides; lipokrit index

Fat tolerance tests—using 500 cc. 20% cream with samples taken at 2, 4 8 and 12 hours and all the lipid studies repeated with each sample.

Repetition of the fat chemistry studies after 1, 2 and 4 weeks of lecithin feeding.

Liver function tests were also made. These tests were the bromsulfalein and the cephalin-cholesterol flocculation tests (4, 5).

For the determination of the lipids, Bloor extracts of serum were made and

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aliquots of these were used. Total cholesterol was determined as follows: saponification in ether-alcohol followed by selective extraction first with petroleum-ether then chloroform. The final determinations were made colorimetrically using the Liebermann-Burkhardt reaction with acetic anhydride and sulphuric acid. The color was determined in an Evelyn electrophotolometer using the 620 filter.

Free cholesterol was determined by extracting with petroleum ether the residue left after evaporation of the original Bloor extract. This was subsequently concentrated and the phospholipids precipitated in acetone solution by magnesium chloride. The precipitate was used for phospholipid P determinations and the supernatant fluid was collected quantitatively with rinsing. After concentration of this acetone solution free cholesterol was precipitated by means of digitonin. This precipitate was freed from interferring substances by extraction with petroleum-ether after which the digitonin-cholesterol complex was dissociated by boiling benzene. The benzene extract was concentrated and freed of digitonin by successive treatments with petroleum-ether. This extract was then worked up in the same fashion as that for the cholesterol determination.

The phospholipid precipitate was digested with sulphuric acid and hydrogen peroxide in the usual fashion. The P was determined colorimetrically by a modification of the Fiske-Subbarow method using the Evelyn photolometer with the 660 filter.

All samples were run in duplicate and, if satisfactory tests were not obtained, additional determinations were made. All determinations were run with several standards and the K value determined for each set of determinations.

The lipokrit method (6) is essentially a simple volumetric micromethod for the determination of lipids modified after the Hemolipokrit method of Ruckert. Freshly prepared special reagent of methylene blue sulphuric acid—Scharlach-R amyl alcohol was mixed with serum in the special lipokrit pipette and shaken and then centrifuged and readings were taken in 30 minutes and after 24 hours of hydrolysis. The 30 minute reading is supposed to represent neutral fats, fatty acids, cholesterol, and cholesterol esters. The 24 hours reading is supposed to indicate cerebrosides and phosphatides.

It is realized, of course, how difficult it is to interpret studies of fat metabolism as related to psoriasis. In 11 cholesterol determinations on 9 cases of psoriasis Strickler and Adams (7) reported 4 cases above 170 mgs. and 7 normal. In an extensive study of 35 cases of psoriasis Rosen and Krasnow (8) reported an average cholesterol of 165 $\pm$ 13, an average lecithin of 248 $\pm$ 16. With regard to cholesterol these investigators had 29% of their cases below 171 $\pm$ 16 mgs., 63% within 171 $\pm$ 16 mgs., and 9% above 171 $\pm$ 16 mgs. With regard to lecithin, 9% were below 242 $\pm$ 22, 66% within 242 $\pm$ 22, and 25% above 242 $\pm$ 22. Montgomery (9) has indicated recently the lack of uniformity of cholesterol determinations in patients with psoriasis. Fat tolerance tests were done also in 10 patients with psoriasis by LeWinn and Zugerman and normal results were obtained, as indicated by the changes in the total cholesterol content of whole

CHART OF LIPOKRIT READINGS

*0 = fasting serum; 2 = 2 hours after fat meal; 4 = 4 hours after fat meal; 8 = 8 hours after fat meal; 12 = 12 hours after fat meal.

WEEK	PATIENT	30 MINUTE READING	TOTAL LIPIDS	WEEK	PATIENT	30 MINUTE READING	TOTAL LIPIDS
Before lecithin feeding	N. M. 0*	.100	.544	Before lecithin feeding	M. H. 0	.200	1.134
	N. M. 2	.040	.904		M. H. 2	.290	1.094
	N. M. 4	.390	1.602		M. H. 4	.670	1.426
	N. M. 8	.200	1.072		M. H. 8	.350	1.106
					M. H. 12	.150	.594
First week with lecithin feeding	N. M. 0	.160	1.072				
	N. M. 2	.560	.992	First week with lecithin feeding	M. H. 0	.170	.674
	N. M. 4	.700	1.012		M. H. 2	.460	.724
	N. M. 8	.660	1.008		M. H. 4		
	N. M. 12	.280	.400		M. H. 8	.360	.384
					M. H. 12	.190	.622
Fourth week with lecithin feeding	N. M. 0	.160	.256				
	N. M. 2	.440	.536	Fourth week with lecithin feeding	M. H. 0	.200	.824
	N. M. 4	.590	.866		M. H. 2	.520	1.048
	N. M. 8	.680	1.220		M. H. 4	.560	1.232
	N. M. 12	.530	.998		M. H. 8	.500	1.292
					M. H. 12	.120	.768
Before lecithin feeding	A. K. 0	.090	.246	Before lecithin feeding	C. Y. 0	.610	1.246
	A. K. 2	.070	.426		C. Y. 2	.500	1.364
	A. K. 4	.110	.674		C. Y. 4	.830	1.610
					C. Y. 8	.740	1.940
First week with lecithin feeding	A. K. 0	.380	.452		C. Y. 12	.610	1.126
	A. K. 2	.400	.556				
	A. K. 4	.320	1.136	First week with lecithin feeding	C. Y. 0	.420	1.188
	A. K. 8	.240	1.200		C. Y. 2	.640	1.744
	A. K. 12	.160	1.036		C. Y. 4	.760	1.840
					C. Y. 8	.600	1.656
Fourth week with lecithin feeding	A. K. 0	.220	.604		C. Y. 12	.280	1.528
	A. K. 2	.530	.638				
	A. K. 4	.560	.572	Fourth week with lecithin feeding	C. Y. 0	.840	1.104
	A. K. 8	.460	.484		C. Y. 2	.760	1.144
	A. K. 12	.460	.460		C. Y. 4	1.020	1.308
					C. Y. 8	1.160	1.304
					C. Y. 12	.640	.976
Before lecithin feeding	A. B. 0	.060	.276	First week with lecithin feeding	A. B. 0	.280	.352
	A. B. 2	.120	.264		A. B. 2	.120	.360
	A. B. 4	.360	.360		A. B. 4	.300	.468
	A. B. 8	.260	.452		A. B. 8	.320	.344
					A. B. 12	.240	.288

blood. There have been criticisms of the fat tolerance tests used at the present time, either the test with the 20% cream or the olive oil-cholesterol mixture, but there are no other practical clinical measures available at present for the study of fat metabolism.

Because of the confusion of the different reports regarding cholesterol levels an effort was made to see whether disturbances existed in the interrelationships of the different lipid components. So, the ratios which Schaaf (10) proposed for xanthoma were employed in these selected cases of psoriasis.

$$\frac{\text{mg. \% combined cholesterol}}{\text{mg. \% free cholesterol}} = A = 2.0 \text{ average}$$

$$\frac{\text{mg. \% phosphatide} - P \times \text{molecular weight of cholesterol}}{\text{mg. \% free cholesterol} \times \text{atomic weight of P}} = B = 2.0 \text{ average}$$

$$\frac{\text{mg. \% combined cholesterol} \times \text{atomic weight of P}}{\text{mg. \% phosphatide} - P \times \text{molecular weight of cholesterol}} = A/B = C = 1.0 \text{ av.}$$

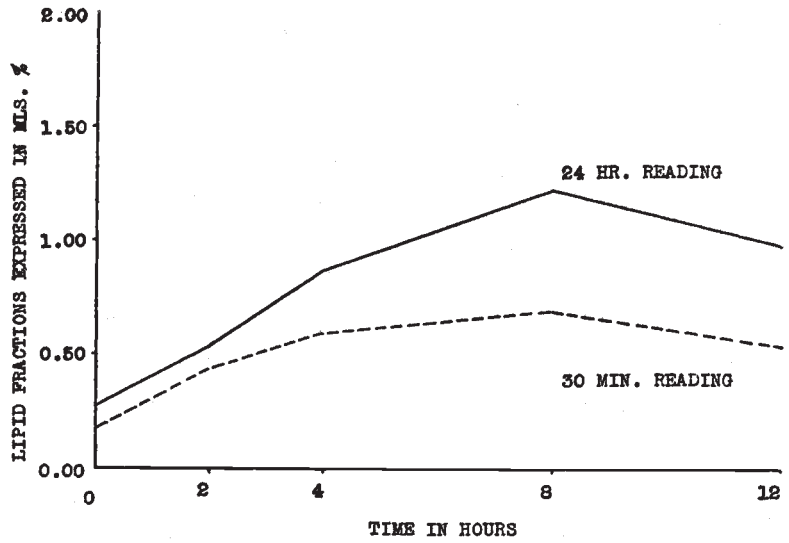

FIG. 1. LIPOKRIT READINGS DURING A FAT TOLERANCE TEST ON A PSORIASIS CASE
Patient N.M. Age 39

Schaaf believed that "as long as there are no qualitative changes in the ratio, quantitative changes of the individual fats and lipoids may then occur without causing any damage." Such ratios were determined on the cases of psoriasis in conjunction with the so-called fat tolerance test. In their work on lipocaic and psoriasis, Clark, Dragstedt, Walsh and Becker (11) measured total fasting blood serum lipids.

There are wide variations for the figures of normal lipoid substances in the blood. In many instances there is chiefly a question of technic. Weidman and Sunderman (12) indicated this in 1925. Their cholesterol figure for man was 160–80. Montgomery and Osterberg's (13) figures for normal lipid levels for man were total cholesterol 180, cholesterol esters 125, lecithin 225, fatty acids 345, total lipids 525.

Sullivan and Fershtand (14) used the lipokrit volumetric method in studying

ANALYSES OF THE LIPID FRACTIONS OF THE SERA OF FOUR PSORIASIS PATIENTS BEFORE AND DURING TWO FAT TOLERANCE TESTS

(First tolerance before lecithin feeding; "3"—after 3rd week of lecithin feeding)

PATIENT AND TIME	CHOLESTEROL		PHOSPHOLIPID PHOSPHORUS	RATIO OF EMULSIFYING AGENTS		
	Total	Free		A*	B†	C‡
	mgm. %	*mgm. %*	*mgm. %*			
N. M. 0	247	70	7.0	3.52	1.25	2.83
N. M. 2	280	73		3.84		
N. M. 4	292	85		3.44		
N. M. 8	305	73		4.18		
N. M. 12	274	59		4.68		
N. 3 M. 0	155	70	7.0	2.22	1.25	1.78
N. 3 M. 2	200	74	6.6	2.70	1.11	2.43
N. 3 M. 4	220	65	8.7	3.39	1.67	2.04
N. 3 M. 8	213	110	9.5	1.94	1.08	1.81
N. 3 M. 12	206	90	8.8	2.29	1.22	1.88
M. H. 0	260	68	9.1	3.82	1.66	2.31
M. H. 2	270	86	10.6	3.14	1.54	2.04
M. H. 4	280	90	11.6	3.12	1.61	1.94
M. H. 8	292	80	11.8	3.65	1.84	1.99
M. H. 12	220	60	9.5	3.67	1.97	1.86
M. 3 H. 0	250	63	9.3	3.98	1.84	2.14
M. 3 H. 2	241	62	9.3	3.90	1.86	2.10
M. 3 H. 4	230	75	8.8	3.06	1.46	2.10
M. 3 H. 8	260	75	11.5	3.46	1.91	1.86
M. 3 H. 12	243	80	11.0	3.04	1.71	1.78
A. K. 0	198	72	6.0	2.76	1.04	2.66
A. K. 2	197	75	8.4	2.62	1.39	1.88
A. K. 4	175	83	8.5	2.11	1.28	1.64
A. K. 8	179	90	7.5	1.98	1.04	1.91
A. K. 12	209	66	6.0	3.16	1.13	2.79
A. 3 K. 0	176	70	8.0	2.52	1.42	1.77
A. 3 K. 2	183	66	8.2	2.78	1.54	1.87
A. 3 K. 4	175	58	8.5	3.02	1.83	1.65
A. 3 K. 8	190	71	8.8	2.68	1.54	1.73
A. 3 K. 12	195	75	9.1	2.60	1.50	1.73
C. Y. 0	433	95	14.2	4.54	1.86	2.44
C. Y. 2	294	115	11.6	2.55	1.26	2.03
C. Y. 4	300	93	14.5	3.22	1.94	1.66
C. Y. 8	370	106	13.5	3.49	1.59	2.20
C. Y. 12	315	99	12.1	3.18	1.52	2.09
C. 3 Y. 0	450	73	15.0	6.15	2.56	2.40
C. 3 Y. 2	390	78	14.1	5.01	2.24	2.22
C. 3 Y. 4	355	80	15.6	4.44	2.43	1.83
C. 3 Y. 8	387		14.4			
C. 3 Y. 12	368	59	12.5	6.24	2.64	2.36

* Ratio A = $\frac{\text{mgm. \% total cholesterol}}{\text{mgm. \% free cholesterol}}$

† Ratio B = $\frac{\text{mgm. \% phospholipid phosphorus} \times \text{mol. wt. of cholesterol}}{\text{mgm. \% free cholesterol} \times \text{atomic wt. of phosphorus}}$

‡ Ratio C = $\frac{\text{mgm. \% total cholesterol} \times \text{atomic wt. of phosphorus}}{\text{mgm. \% phospholipid phosphorus} \times \text{mol. wt. of cholesterol}} = \frac{A}{B}$

CLINICAL SUMMARIES

NAME	AGE	DURATION OF PSORIASIS	TYPE OF PSORIASIS	LIVER FUNCTION		PREVIOUS TREATMENT	AMOUNT LECITHIN MIXTURE DAILY	DURATION OF FEEDING	CLINICAL RESULT
				Bromosulphthalein	Cephalin flocculation				
		years					*grams*		
A. K.	54	25	Extensive plaque, annular and guttate, all infiltrated on body and on scalp lesions of discreet type	0	0	All types save x-ray	12	4 weeks controlled, 6 weeks total	Much improved
N. M.	39	2	Plaque, annular and guttate, all infiltrated	0	0	All types save x-ray	12	4 weeks controlled, 6 weeks total	Much improved
C. Y.	36	10	Guttate on trunk and diffusely on scalp	0	0	Self-medication only proprietory	12	4 weeks controlled	No significant change
M. H.	52	32	Plaque on trunk, guttate on extremities diffuse scalp involvment. Had previous exfoliative reaction two years ago	0	0	Reactions with strong local medications. 6 weeks of bed rest with exfoliative phase	12	4 weeks controlled, 6 weeks total	No significant change
A. B.	11	1½	Papular placque-like entire body diffuse pityriasis scalp	*	*	Lecithin 5 months ago improved remarkably flared when discontinued	12	4 weeks controlled	Improved

* Not done.

fat absorption in normal and pathological cases. They found the average fasting lipokrit level of 43 normal young men to be 0.576%. McGrath (15) studying the fasting lipokrit of 100 essentially normal hospital patients has found values varying from 0.120 to 0.680% with a mean value of 0.346%. Those patients with fasting lipokrit levels falling within the normal range improved (N. M., A. K., and A. B.). Those patients with abnormally high initial fasting lipokrit levels did not (M. H. and C. Y.). The lipokrit values for the fat tolerance curves cannot be interpreted in this study because no control fat tolerance curves are available as yet.

In a general manner, the cholesterol, cholesterol esters, and phosphatides paralleled the lipokrit for each patient. If this continues to be true, the lipokrit is a much simpler test to carry out.

Also, from these very few preliminary studies there seem to be marked changes from normal in the lipid ratios of Schaaf. This would suggest that this particular phase of fat metabolism, (stability of emulsions?) is disturbed in psoriasis as it may be in xanthomatosis. Further work must be done on this, especially as regards the "A" ratios.

We do not know at present from our figures on these few patients whether a fat tolerance study is of any more help than a study simply of the fasting serum. When a fat tolerance test will be standardized this question will be answered.

From these few patients it is evident that those patients with a relatively low blood lipid level responded better to lecithin feeding than those patients with initial high lipid levels. This has been noted before with lipocaic. It would seem the lecithin feeding in spite of improvement, had no appreciable effect on the lipid levels. An effort will be made to attempt to lower the blood lipids of the resistant individuals and then to determine if these individuals will respond to lecithin therapy. Thyroid may serve to help lower the lipid level. It is possible also that those patients with a lower phospholipid level also improved under lecithin therapy. In most instances the free cholesterol did not change a great deal.

From a period of at least six weeks (in case B 3 weeks) observation it was seen that three of the patients improved with lecithin feeding. Patient B had improved some weeks previously and then relapsed when the lecithin was discontinued. This is in accord with our previous experiences with lecithin. In this instance, however, the patients were hospitalized and kept under more rigid control with only boric ointment used from time to time. In this study also, the patients were kept on a fat poor diet. In our next control series a regular diet will be used. However, it is our impression at present that lecithin is more effective when the patient is on a fat poor diet.

CONCLUSIONS

1. A method for studying the fat metabolism of patients with psoriasis is presented. This study included: 1) Determinations of cholesterol, cholesterol esters, and phosphatides and consequently the lipid ratios of Schaaf in conjunc-

tion with fat tolerance tests. 2) Lipokrit index. 3) Liver function tests including the cephalin-cholesterol flocculation test.

2. A study was started of the effect of soybean lecithin feeding on the lipid levels in five patients with psoriasis of varying severity. These patients were observed in the hospital and were on a fat poor diet at the same time. From the studies completed at present, in spite of improvement of the patients under lecithin feeding there was no marked change in the lipid levels, and no definite changes in the abnormal lipid ratios. Those three patients who had relatively low lipid levels improved under lecithin feeding while those two patients with relatively high lipid levels did not improve under the lecithin. Further studies are needed to clarify these findings. Similar experiences have been recorded before with lipocaic.

The lipokrit determinations and curves were done by Ruth Hazlewood, B.A., in the laboratory of Dr. Edward J. McGrath of the Department of Surgery of the Cincinnati General Hospital.

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DISCUSSION

DR. PAUL GROSS, *New York, N. Y.:* Dr. Goldman referred to the work on certain lipotropic substances in the treatment of Psoriasis, which Dr. Kesten and I presented at the Meeting of the American Academy of Dermatology in December 1941. We are interested in the results reported here since we have carried on a similar investigation.

The statement of Dr. Goldman that soyabean lecithin is a substitute for lipocaic, requires further clarification. The recent reports on the effect of lipocaic in depancreatized dogs, show that its action differs from that of inositol and choline. The so-called soyabean lecithin is a mixture of cephalin, containing inositol, and lecithin, containing choline. Nevertheless, we have found that a defatted wheat germ, which is a good source of inositol and its esters, and contains the entire Vitamin B Complex plus minerals, has a therapeutic effect on psoriasis similar to that of soyabean lecithin. This, despite the fact that defatted wheat germ is practically free of choline. In the action on high serum cholesterol, the soyabean lecithin is more effective than the wheat germ. Further studies are necessary to explain these facts, but we should not view a normal serum cholesterol as absolute evidence of a normal fat metabolism.

DR. ARTHUR C. CURTIS, *Ann Arbor, Michigan:* I think Dr. Gross made a mistake when he said I had done extensive work on fat metabolism with psoriasis. I have followed about twenty patients with psoriasis on lecithin feeding, with my interest directed more toward the disturbance in fat metabolism and its changes on such a regimen than upon the involution of the psoriasis lesions.

In these patients, the cholesterol values were usually elevated but the total blood lipids were either normal or only slightly abnormal. The spread of fat, cholesterol, and phospholipid levels were so little changed, compared to an equal number of normal people, that we feel our figures were not significant. Diseases such as hyperthyroidism, hypothyroidism and myxedema would produce similar changes.

In xanthalsma high total blood lipid levels are to be expected and were found. In some cases we have followed, lecithin seemed to reduce the blood fat level but again we have not followed enough cases for a long enough period to report these results as final at that time.

DR. DAVID KAHN, *Chicago:* Six months ago I briefly reviewed the records of 46 psoriatic patients who had received lipocaic. The various responses to this therapy did not appear to be definitely correlated with changes in the total blood lipids, cholesterol, or cholesterol esters. A more detailed study is in progress.

DR. DUDLEY C. SMITH, *University, Va.:* Several years ago I had the occasion to discuss the treatment of psoriasis with a physician in general practice. He stated that he had had four cases over the period of a number of years and had "cured them all". All of these patients had a distaste for milk and butter. He insisted that they take ¼ lb. of butter and 1 qt. of milk daily. I did not know how to interpret these statements because the diagnosis may have been incorrect and psoriasis is a variable clinical condition.

Shortly after this a patient with typical psoriasis was seen in the Clinic. When asked if he drank milk and ate butter he answered in a vigorous tone that "he never took either in his life". The next day a physician who had been seen several times previously because of psoriasis came in. When he was asked the same question he replied that he had never liked nor used milk or butter. We re-examined 35 cases of psoriasis and found that about 75% of these either had a distaste for dairy products or were unable to obtain these in adequate quantities.

Therapeutic trials with milk and butter, cod liver oil and carotene in oil caused no special relief in any of these cases except that one was apparently cured. The high percentage of cases though whose history indicate this deficiency would seem to be significant. Experimental work similar to that reported here may lead eventually to the scientific explanation of the above observations.

DR. LEON GOLDMAN, *Cincinnati:* Some of the work done previously in diets with psoriasis may be correlated by the fact that the bad effects of a high protein diet certainly have some relationship to cholesterol metabolism.

At present, although the compounds are certainly different, lipocaic and lecithin feedings seem to have similar effects on the fat metabolism.

However well the fat metabolism theory may sound for psoriasis, practice alone will show its true value. "Syndrome" was used because we believe that psoriasis is a type cutaneous reaction of different etiologies.