SOME BIOCHEMICAL EFFECTS OF A MAINLY FRUIT DIET IN MAN*

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SUMMARY

The effect of a nut-supplemented fruit diet on glucose tolerance, insulin secretion, plasma proteins and plasma lipids of human volunteers was investigated. The results suggest that, at least for the period covered by the experiment and under the prevailing conditions, the diet was adequate with respect to the parameters investigated, and may even have something to commend it.

In a previous paper[43] some physiological effects of a nut-supplemented fruit diet on human volunteers were described. The impact of the diet on a variety of biochemical parameters in these subjects was also investigated. The results of this investigation are now reported.

METHODOLOGY

The biochemical parameters investigated included plasma electrolytes (K^+, Na^+, Cl^-, HCO_3^-), potassium per kg body-weight, liver functions, plasma proteins, α- and β-lipoproteins, blood lipids (a complete lipogram was done in 5 cases), thyroid function (PBI and ^{131}I-uptake) glucose-tolerance curve, insulin curve and renal function (in some cases).

The total potassium content of the body was determined by measuring the ^{40}K content by means of a single crystal whole-body counter. The total plasma protein was determined by the method of Weichselbaum,[61] while the protein fractions were determined electrophoretically on cellulose acetate strips using the Beckman microzone apparatus and technique.[44] Electrograms were stained after fixing with Ponceau S solution.

Cholesterol levels were determined in the auto-analyser (Technicon) using a modification of the method described by Levine and Zak.[36] The micromethod of Young and Eastman[64] was used to determine the triglycerides. The α- and β-lipoproteins were determined by the paper electrophoresis method of Fredrickson *et al.*[22] The protein-bound iodine and 24-hour ^{131}I-uptake were employed as criteria of thyroid function. Fifty grams of glucose, taken by mouth, was used to determine glucose tolerance. The glucose level was determined in the auto-analyser (Technicon) utilizing the potassium ferricyanide-potassium ferrocyanide oxidation-reduction reaction. This method is a modification of the method proposed by Hoffman.[29] Insulin levels were determined by the procedure of method C of the double antibody method of Hales and Randle.[26] The other biochemical parameters were determined by means of standard laboratory methods.

RESULTS

The various biochemical parameters for the subject forming group C were determined at three 20-week intervals and the values given in the tables represent the mean.

Glucose tolerance. The tolerance to 50 g of glucose *per os* was determined for each subject before the project began and subsequently at 6- or 12-weekly intervals. The results are presented in Tables I and II. According to Brown *et al.*[10] schizophrenia does not affect tolerance to glucose. On the other hand Conn[12] reported that diet does affect tolerance to glucose, and the results obtained for the Bantu subjects (group A_1) and the White subjects (group B) are therefore presented separately (Tables I and II).

TABLE I. GLUCOSE TOLERANCE OF GROUP A_1

Patient No.	Glucose (mg/100 ml) Fasting	30 min	60 min	90 min	120 min
At zero time					
1	82	156	181	125	70
2	82	88	143	125	100
3	82	129	133	100	88
4	82	117	104	88	94
5		100	140	94	70
6	70	119	130	110	79
7	95	132	141	143	122
8	79	110	110	100	84
9	100	145	162	124	115
Mean	84·00	121·77	138·22	112·11	91·33
SD	9·36	21·37	23·69	18·15	18·41
After 12 weeks					
1	93	165	201	180	137
2	85	115	120	128	118
3	85	145	156	142	109
4	101	140	161	156	126
5	90	140	109	101	105
6	90	152	156	100	82
7	100	197	209	198	174
8	94	154	144	124	112
9	104	174	213	198	177
Mean	93·55	153·55	163·22	147·44	126·67
SD	6·88	23·36	37·56	38·10	31·52
After 24 weeks					
1	88	135	110	60	82
2	88	55	94	100	75
3	94	128	118	75	60
4	100	108	135		
5	100	110	130	123	88
6	81	139	132	87	91
7	91	186	175	155	132
8	91	153	150	148	118
9	91	167	136	155	132
Mean	91·55	131·22	131·11	112·87	97·25
SD	5·99	38·14	23·23	37·70	26·95

As is clear from Table I, the fasting blood sugar level of the subjects of group A_1 were raised after 12 and 24 weeks on the diet. This change was statistically significant at the 5% level of reliability ($P<0{\cdot}05$). The subjects from group B showed no statistically significant change in the fasting glucose level. The subject in group C showed a sharp increase in her blood sugar level which returned to more or less the fasting level after 2 hours (Table II). Glycosuria was present after 30 minutes but had almost disappeared after 1½ hours.

Insulin secretion. Some of the blood drawn to determine glucose tolerance was stored at −20°C until the experiment was completed and the final insulin levels of all the blood samples collected from individual subjects were then determined simultaneously. The results are presented in Tables III and IV.

*Date received: 9 November 1970.

TABLE II. GLUCOSE TOLERANCE OF GROUPS B AND C

Patient No.	Glucose (mg/100 ml) Fasting	30 min	60 min	90 min	120 min
At zero time					
1	102	164	124	90	60
2	80	107	55	88	80
3	95	174	177	60	65
4	95	135	170	100	95
5	95	130	110	95	110
6	95	130	72	45	71
7	80	90	90	65	65
8	90	177	193	65	40
Mean	91·50	138·37	123·87	76·00	73·25
SD	7·80	31·38	51·41	19·76	21·68
After 6 weeks					
1	95	165	140	118	65
2					
3	110	180	87	63	67
4	100	163	173	133	105
5	87	118	110	107	110
6					
7	78	88	58	65	74
8	95	168	150	80	66
Mean	94·16	147·00	119·66	94·33	81·16
SD	10·94	35·86	42·75	29·20	20·70
After 12 weeks					
1	97	166	145	84	72
2	77	102	77	50	66
3	71	136	95	40	64
4	71	174	136	71	65
5	80	127	100	77	70
6	84	122	80	45	66
7	71	83	75	58	50
8	106	160	155	134	131
Mean	82·12	133·75	107·87	69·87	73·00
SD	13·07	31·89	32·57	30·26	24·33
Group C	90	195	215	150	101

TABLE III. INSULIN RESPONSE OF GROUP A_1

Patient No.	Insulin ($\mu U/ml$) Fasting	30 min	60 min	90 min	120 min
At zero time					
1	25	246	321	263	41
2	22	61	331	267	126
3	21	20	96	48	38
4	17	350	173	45	49
5	18	44	225	224	
6	20	33	48	36	27
7	13	35	51	63	31
8	28	18	69	114	100
9	17	87	102	47	37
Mean	20·11	99·33	157·33	123·00	56·12
SD	4·54	117·51	111·61	99·54	36·37
After 12 weeks					
1	15	54	77	48	35
2	13	41	56	47	46
3	11	36	60	35	17
4	13	78	85	68	41
5	10	39	41	25	18
6	11	49	52	49	26
7	17	58	54	57	57
8	24	70	72	64	60
9	23	77	86	65	63
Mean	15·22	55·78	64·78	50·89	40·33
SD	5·17	16·17	15·84	14·37	17·64
After 24 weeks					
1	17	90	49	19	13
2	15	19	31	47	21
3	13	26	52	28	19
4	19	44	123		
5	13	32	39	112	22
6	19	46	49	27	26
7	18	93	89	38	47
8	22	33	30	88	53
9	23	129	59	97	68
Mean	17·67	56·89	57·89	57·00	33·62
SD	3·57	37·86	30·12	36·32	19·74

As will be noticed, the insulin responses were high in 4 of the Bantu subjects when the project began. The reason for these high values is not clear. The average maximum insulin level for the corresponding control group was 121 μU insulin/ml serum, and for control persons without medication 220 μU insulin/ml serum.

After 12 and 24 weeks on the fruit diet most of the Bantu subjects showed a drop in insulin levels (Table III). This drop was significant at the 5% level of reliability. Only 3 of the 9 Bantu subjects showed statistically significant positive correlation ($P<0{\cdot}05$) between blood glucose levels and insulin levels during all of the follow-up test runs.

Only 2 of the 8 normal White subjects showed a slight drop in their insulin levels, while the maximum insulin level increased in 4 of the subjects after 6 and 12 weeks. Only 3 of the 8 subjects showed a good correlation ($P<0{\cdot}05$) between glucose and insulin levels (Table IV). In the case of the subject in group C the blood insulin and glucose levels (Table IV) correlated well ($P<0{\cdot}01$).

Total serum lipids. The total serum lipids of the subjects of both groups had decreased significantly ($P<0{\cdot}01$) after 12 weeks on the diet, and the reduced lipid levels were maintained in group A_1 throughout the 24 weeks that the experiment lasted (Tables V and VI and Fig. 1). The serum lipid level of the single subject in group C was even lower than that of groups A_1 and B. This was the case notwithstanding the fact that fat supplied 45% of her total calorie intake and 36·7 and 27·6% of the total calorie intake of groups A_1 and B respectively.

Cholesterol. The mean serum cholesterol of group A_1 decreased during the first 12 weeks of the experiment and the decrease was significant ($P<0{\cdot}05$) after 24 weeks. In group B the cholesterol level had decreased significantly ($P<0{\cdot}01$) after 6 weeks on the diet. After 12 weeks the mean cholesterol level was still below the control values, but the decrease was no longer statistically significant. This can be ascribed at least in part to an excessive consumption of nuts with a relatively high saturated fatty acid content by some of the subjects. The subject in group C had a very low serum cholesterol level.

Triglycerides. The mean serum triglyceride levels of group A_1 had decreased significantly after 12 weeks on the diet ($P<0{\cdot}01$) and the reduced values were maintained except in 4 subjects in whom the triglyceride levels tended to rise again after 12 weeks. This might be ascribed partly to a reduction in avocado intake to 100 g instead of 200 to 400 g/day. The reduction was due to a short supply of avocados during the 3 weeks before the follow-up tests.

The control mean serum triglyceride level of group B was considerably lower than that of group A_1, and unlike group A_1, no statistically significant changes in the triglyceride levels occurred in group B. The serum triglyceride level of the subject of group C was low.

TABLE IV. INSULIN RESPONSE OF GROUPS B AND C

Patient No.	Insulin ($\mu U/ml$) Fasting	30 min	60 min	90 min	120 min
At zero time					
1	16	70	60	29	17
2	13	62	28	13	11
3	10	35	60	21	15
4	10	36	68	37	31
5	19	50	45	47	48
6	23	300	101	27	23
7	19	83	78	34	24
8	10	74	178	26	13
Mean	15·00	88·75	77·25	29·25	22·75
SD	5·01	87·11	46·07	10·31	12·15
After 6 weeks					
1	18	161	300	195	26
2	22	83	26	19	17
3	18	310	35	24	21
4	22	157	300	170	58
5	19	37	34	37	24
6	19	43	39	21	17
7	16	75	20	35	24
8	13	131	121	26	18
Mean	18·37	124·62	109·37	65·87	25·62
SD	2·97	88·89	121·84	72·56	13·53
After 12 weeks					
1	26	167	350	40	26
2	20	69	49	19	18
3	15	93	28	15	17
4	21	118	85	21	19
5	16	47	65	20	16
6	23	285	80	21	21
7	14	23	31	26	16
8	12	92	103	21	16
Mean	18·37	111·75	98·87	22·87	18·62
SD	4·86	82·58	104·80	7·54	3·46
Group C	23	214	218	102	38

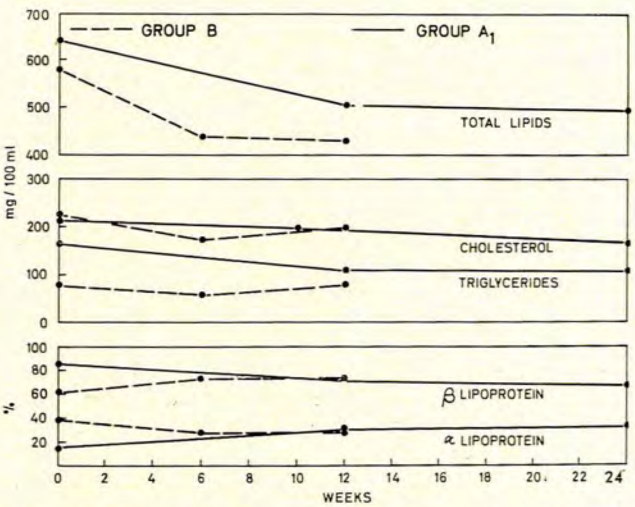

Fig. 1. Changes in the serum lipid levels of the Bantu (group A_1) and White (group B) subjects.

Phospholipids. The serum phospholipid levels were determined only in the subjects of group A_1, and decreased significantly ($P<0·05$) in these cases.

Alpha- and beta-lipoproteins. The results obtained for groups A_1 and B are contradictory. In the case of group A_1 the mean β-lipoprotein level decreased significantly,

TABLE V. PLASMA LIPID LEVELS OF GROUP A_1

Patient No.	Total lipids mg/100 ml 0 weeks	12 weeks	24 weeks	Cholesterol mg/100 ml 0 weeks	12 weeks	24 weeks	Triglycerides mg/100 ml 0 weeks	12 weeks	24 weeks	Phospholipids mg/100 ml 0 weeks	12 weeks	24 weeks	α-lipoproteins % 0 weeks	12 weeks	24 weeks	β-lipoproteins % 0 weeks	12 weeks	24 weeks
1	635	497	339	178	157	129	219	152	84	238	188	126						
2	573	449	383	162	158	148	197	103	75	214	188	160						
3	523	637	467	196	236	183	115	146	97	212	255	187						
4	711	623	463	216	230	203	211	144	59	284	249	201						
5	526	596	534	180	210	208	108	123	104	238	263	222						
6	599	418	419	220	200	130	107	77	92				16	22	50	84	78	50
7	675	352	447	234	160	168	214	59	208				15	34	36	85	66	64
8	836	561	570	306	244	216	182	92	149				14	27	11	86	73	89
9	675	418	419	232	186	132	128	83	106				16	34	32	84	66	68
Mean	639·22	505·67	449·00	213·78	197·89	168·56	164·56	108·78	108·22	237·20	228·60	179·20	15·25	29·25	32·25	84·75	70·75	67·75
SD	99·24	102·78	71·28	42·88	34·64	35·38	49·02	33·84	44·92	29·00	37·39	37·30	0·95	5·85	16·13	0·95	5·07	16·13

TABLE VI. PLASMA LIPID LEVELS OF GROUPS B AND C

Patient No.	Total lipids mg/100 ml 0 weeks	6 weeks	12 weeks	Cholesterol mg/100 ml 0 weeks	6 weeks	12 weeks	Triglycerides mg/100 ml 0 weeks	6 weeks	12 weeks	α-lipoproteins % 0 weeks	6 weeks	12 weeks	β-lipoproteins % 0 weeks	6 weeks	12 weeks
1	732	542	457	252	202	200	103	91	121	38	18		62	82	
2	485	390	371	182	136	160	50	47	82	44	29	20	56	71	80
3	590	466	476	250	192	240	41	22	47	51	47	44	49	53	55
4	656	466	523	248	184	220	53	40	59	44	30	39	56	70	61
5	495	390	333	248	160	146	57	54	51	42	15	13	58	85	87
6	675	419	381	260	170	164	117	77	102	25	23	14	75	77	86
7	409	380	409	146	140	152	62	60	59	27	31	30	73	69	70
8	599	466	494	210	196	308	148	75	101	35	27	35	65	73	65
Mean	580·12	439·87	430·50	224·50	172·50	198·75	78·87	58·25	77·75	38·25	27·50	27·85	61·75	72·50	72·00
SD	109·45	55·38	66·89	41·31	25·31	55·76	38·75	22·35	27·72	8·90	9·76	12·34	8·90	9·76	12·56
Group C		376			137			69·6			30			70	

while it increased significantly in group B ($P<0.05$). The results are presented in Tables V and VI.

Plasma proteins. The values obtained for the total plasma protein, albumin, globulin and different globulin fractions at different time periods of the experiment are summarized in Tables VII and VIII. The changes which occurred are presented graphically in Figs. 2 and 3.

As is clear from Table VII, the mean total plasma protein level of group A_1 decreased initially, but then increased again to reach a mean value which was only slightly below the initial value. The decrease was mainly due to a decrease in globulins from 3·7 to 3·4 g/100 ml. This decrease was not statistically significant, unlike the decrease in α_1 and α_2-globulin. After 24 weeks the mean albumin level was 0·319 g/100 ml higher than the mean initial value. The changes in total protein, albumin and globulin fractions are illustrated in Fig. 3.

The total plasma protein of normal White volunteers increased significantly ($P<0.01$) over the first 6 weeks of the experiment, but returned to almost normal after 12 weeks. Similar changes occurred in the albumin levels. The total globulin levels also increased in this group during the first 12 weeks and the increase was statistically significant ($P<0.05$ after 6 weeks and <0.01 after 12 weeks). The increase in total globulin was mainly due to an increase of α_1-globulin during the first 6 weeks ($P<0.01$) and to an increase of β-globulin during the second 6 weeks ($P<0.01$).

The total plasma protein and albumin levels of the one subject constituting group C did not differ significantly from those of the normal volunteers (Table VIII).

Bantu control persons. No indication was found that medication or the type of psychoses from which the patient suffered significantly affected any of the biochemical parameters evaluated.

Glucose Tolerance

Different methods are used to assess the tolerance of subjects to glucose. According to Notelovitz[45] the specificity and sensitivity of subjects to the standard method of 50 g of glucose *per os* give results which compare very well with the results obtained when the glucose is administered by the intravenous route. Apparently the standard method also gives superior results as far as insulin secretion is concerned.[42] Consequently the standard method was employed in the present experiment. With this method only the Bantu subjects showed a statistically significant increase in fasting blood glucose levels after 12 and 24 weeks respectively. Dalderup *et al.*[35] noticed a similar tendency towards raised fasting blood sugar levels in rats maintained on a high intake of refined monosaccharides and disaccharides.

Bantu are habituated to a low-fat, high-carbohydrate diet while the present diet was a high-sugar, relatively high-fat diet. Several authors have demonstrated that the amounts of circulating glucose in rats increased if they were put on a high-fat diet.[7,62] The present diet also contained large quantities of fructose and impaired glucose utilization had been observed in the livers of human subjects who consumed considerable amounts of fructose[58] as well as in rats fed on fructose.[20] This might be due to a decrease in the glucokinase activity of the liver or to an interference with the mechanism responsible for trans-

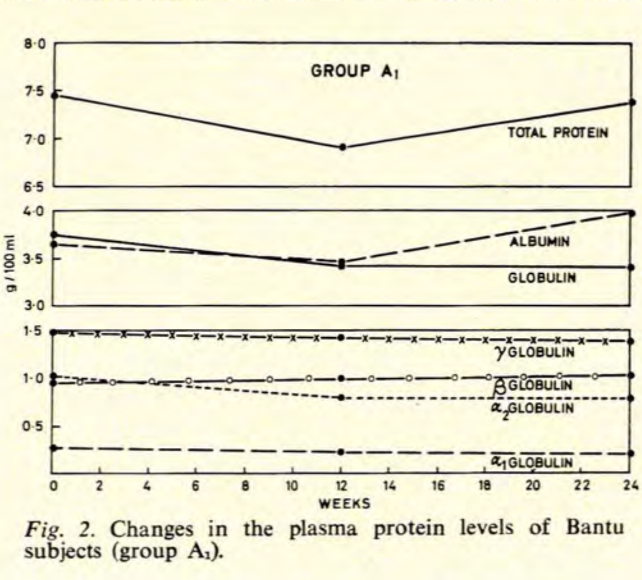

Fig. 2. Changes in the plasma protein levels of Bantu subjects (group A_1).

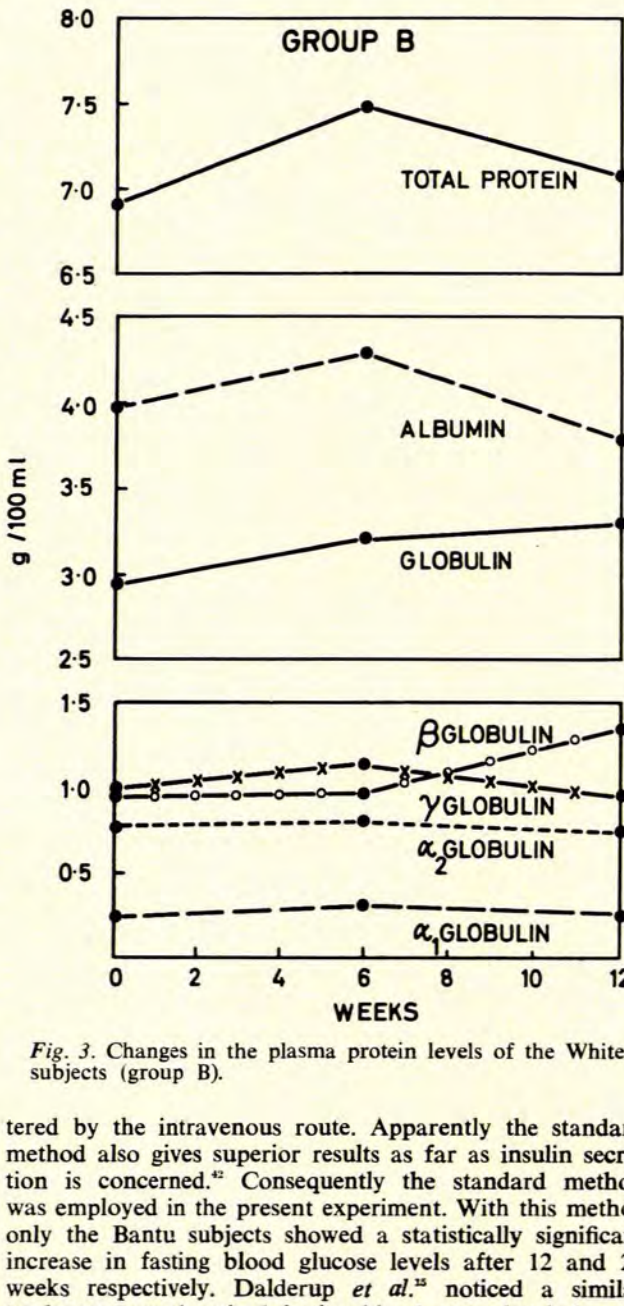

Fig. 3. Changes in the plasma protein levels of the White subjects (group B).

porting glucose to the site of glucokinase activity in the cell. This is, however, still a subject of much controversy.

The relatively high potassium intake of the subjects is apparently not responsible for their higher fasting blood glucose levels, for according to Conn[13] 'a large body of experimental evidence indicates that potassium deficiency leads to deterioration of carbohydrate tolerance, but the mechanisms involved are not clear. The potassium is now known to be an essential activator of many intracellular enzymatic reactions and especially those involved with the transfer of high energy phosphate in carbohydrate metabolism.' Seedat,[52] likewise, noticed an inverse relationship between the plasma potassium and the blood sugar in his patients.

The fasting blood glucose levels of the normal White volunteers were not affected by the fruit diet. This could perhaps have been expected, for these subjects were used to high sugar and fat intakes before their collaboration in this project and their fruit consumption was likewise considerable.

The fruit diet did not produce a statistically significant change in the tolerance of glucose of Bantu and Whites. Persson *et al.*[46] reported a reduced tolerance to glucose in human subjects who consumed a low-carbohydrate diet. Conn[12] made similar observations.

Insulin Secretion

The fruit diet produced a statistically significant reduction in the plasma insulin response after glucose intake in some Bantu while in some Whites there was an inexplicably high insulin secretion at some stage during the experiment. Blazquez and Lopez[7] reported an increase in plasma glucose and hepatic glycogen together with a simultaneous marked decrease in the concentration of plasma insulin and in the utilization of glucose by the tissues of rats fed on foods with a high fat content. The sugar intake in the present diet was high, but so was the fat intake. It has also been reported that adrenaline inhibits insulin secretion in the presence of high glucose levels[14,37] and mental patients apparently often have elevated adrenaline secretions.

In the presence of a relatively high potassium intake, as was the case in the present experiment, one would have expected an increased insulin secretion by the pancreas, for according to Conn[13] a low plasma potassium level prevents the release of insulin from the pancreas and according to Hales and Milner[25] an increase in potassium stimulates insulin secretion. The fruit diet produced a considerable change in the insulin response to oral glucose but the time required to produce these responses differed from subject to subject.

Only some of the subjects showed a significant correlation between plasma glucose and plasma insulin levels. Buchanan and McKiddie[11] and Abrams *et al.*[1] reported similar inconsistent results, while Seymore and De Bruin[53] found that 50% of their cases showed a significant correlation.

Another factor to consider in connection with the plasma insulin response of the subjects in the present experiment is their high avocado intake, for according to Viktora *et al.*[50] avocado suppresses insulin secretion. This effect of avocado has been ascribed to its high content of a 7-carbon sugar, namely D-mannoheptulose which con-

TABLE VII. PLASMA PROTEIN VALUES OF GROUP A_1

Patient No.	Total protein g/100 ml 0 weeks	12 weeks	24 weeks	Albumin g/100 ml 0 weeks	12 weeks	24 weeks	Total globulin g/100 ml 0 weeks	12 weeks	24 weeks	α_1-globulin g/100 ml 0 weeks	12 weeks	24 weeks	α_2-globulin g/100 ml 0 weeks	12 weeks	24 weeks	β-globulin g/100 ml 0 weeks	12 weeks	24 weeks	γ-globulin g/100 ml 0 weeks	12 weeks	24 weeks
1	8·3	7·0	8·5	4·68	3·39	4·55	3·62	3·58	3·96	0·32	0·28	0·30	1·03	0·85	0·81	0·86	1·11	1·23	1·41	1·34	1·62
2	7·8	7·4	8·1	4·34	3·93	4·29	3·46	3·47	3·80	0·24	0·21	0·24	0·99	0·80	1·05	0·77	1·18	0·97	1·46	1·28	1·45
3	8·1	6·7	8·3	4·20	3·22	4·32	3·49	3·49	3·97	0·19	0·23	0·27	0·81	0·71	0·91	0·92	1·05	1·14	1·57	1·50	1·67
4	6·6	6·4	8·8	3·69	3·09	4·93	2·92	3·16	3·88	0·27	0·23	0·32	0·65	0·56	0·59	0·79	1·02	1·26	1·21	1·35	1·71
5	7·1	7·4	7·9	2·28	3·46	4·80	4·83	3·95	3·10	0·47	0·27	0·21	1·21	0·92	0·77	1·03	1·18	0·98	2·12	1·58	1·14
6	7·6	6·9	6·9	3·57	3·48	3·54	4·03	3·42	3·34	0·26	0·23	0·18	0·93	0·70	0·61	1·24	0·87	0·91	1·60	1·62	1·64
7	6·9	6·4	6·2	3·42	3·23	3·09	3·48	3·17	3·10	0·22	0·19	0·15	1·17	0·97	1·00	1·11	0·85	0·95	0·98	1·16	1·00
8	7·2	6·7	6·0	3·35	3·35	3·17	3·85	3·34	2·81	0·28	0·22	0·08	1·23	0·84	0·65	0·98	0·90	1·07	1·35	1·38	1·01
9	7·4	7·2	5·8	3·38	4·01	3·09	4·02	3·19	2·70	0·26	0·16	0·13	1·19	0·76	0·59	0·91	0·76	0·75	1·66	1·51	1·23
Mean	7·44	6·90	7·39	3·656	3·462	3·975	3·744	3·418	3·406	0·278	0·224	0·208	1·023	0·790	0·775	0·956	0·991	1·028	1·484	1·413	1·385
SD	0·56	0·38	1·17	0·705	0·316	0·756	0·533	0·262	0·510	0·083	0·031	0·083	0·200	0·122	0·178	0·154	0·151	0·167	0·320	0·151	0·294

TABLE VIII. PLASMA PROTEIN VALUES OF GROUPS B AND C

Patient No.	Total protein g/100 ml 0 weeks	6 weeks	12 weeks	Albumin g/100 ml 0 weeks	5 weeks	12 weeks	Total globulin g/100 ml 0 weeks	6 weeks	12 weeks	α_1-globulin g/100 ml 0 weeks	6 weeks	12 weeks	α_2-globulin g/100 ml 0 weeks	6 weeks	12 weeks	β-globulin g/100 ml 0 weeks	6 weeks	12 weeks	γ-globulin g/100 ml 0 weeks	6 weeks	12 weeks
1	7·4	7·7	7·5	4·14	4·22	4·03	3·26	3·48	3·48	0·18	0·40	0·20	0·93	0·83	0·75	1·16	1·01	1·64	0·99	1·24	0·89
2	6·7	7·7	6·8	3·91	4·23	3·31	2·78	3·55	3·49	0·21	0·27	0·29	0·78	0·92	0·71	0·74	0·84	1·31	1·05	1·52	1·18
3	7·2	7·2	7·0	4·13	4·17	3·68	3·07	3·03	3·32	0·25	0·24	0·29	0·81	0·87	0·88	0·94	0·74	1·11	1·07	1·18	1·04
4	6·5	7·5	7·0	3·86	4·37	3·70	2·65	3·13	3·30	0·25	0·32	0·31	0·75	0·83	0·95	0·81	0·86	1·09	0·84	1·12	0·95
5	7·0	7·2	6·5	3·97	4·20	3·35	3·04	3·00	3·23	0·23	0·30	0·23	0·92	0·87	0·73	0·80	0·66	1·33	1·09	1·17	0·94
6	7·2	8·1	7·2	4·51	5·08	4·05	2·69	3·02	3·15	0·24	0·32	0·26	0·55	0·58	0·52	0·79	1·09	1·48	1·11	1·03	0·89
7	6·3	7·2	6·8	3·52	4·02	3·58	2·78	3·17	3·21	0·33	0·28	0·33	0·66	0·67	0·65	0·90	1·09	1·18	0·90	1·13	1·05
8	7·0	7·2	7·8	3·76	3·97	4·66	3·25	3·23	3·19	0·27	0·37	0·21	0·78	0·77	0·80	1·32	1·38	1·53	0·88	0·71	0·65
Mean	6·91	7·47	7·07	3·975	4·282	3·795	2·940	3·201	3·296	0·245	0·312	0·265	0·772	0·792	0·748	0·932	0·958	1·333	0·991	1·137	0·948
SD	0·37	0·33	0·41	0·294	0·344	0·441	0·244	0·209	0·126	0·044	0·044	0·044	0·122	0·114	0·130	0·202	0·230	0·200	0·104	0·223	0·154
Group C		7·5			4·07			3·46			0·22			0·76			1·18			1·29	

stitutes approximately 1·3% of the pulp of avocado.[55] In 5 of the 8 human subjects studied by Viktora *et al.*[59] mannoheptulose reduced insulin secretion. No effect was apparent in the other 3 subjects. In some of these subjects the depression of the plasma insulin resulted in benign melituria. According to these workers, feeding of mannoheptulose to animals produced a low steady blood mannoheptulose level which was maintained for extended periods. Mannoheptulose accumulates in the liver[54] and Viktora *et al.*[59] suggested that the maintenance of a steady mannoheptulose blood level for a considerable number of hours is due to slow absorption of this sugar from the intestines, and later the slow release of the sugar from the liver. Our subjects consumed a minimum of 225 g of avocado per day, so that their minimum intake of mannoheptulose was about 2·7 g per day. It is, however, doubtful whether the D-mannoheptulose played any part in the reduced insulin response to oral glucose in the present study, for the glucose-tolerance test was performed 12 hours after the last meal and the reduced insulin response was only noticed in the Bantu subjects.

When this experiment commenced the subject of group C had eaten considerable amounts of avocado daily for the past 12 years. Although she developed glycosuria during the glucose-tolerance test, she had no symptoms or signs of diabetes mellitus, and her glucose-tolerance curve was not of the diabetic type. Her melituria was therefore apparently benign.

Lipids

To some extent the plasma levels of cholesterol, triglycerides and phospholipids are interrelated. Hence conditions affecting the level of one often also affect the levels of the other two. Fat intake is the most important single factor affecting the plasma lipid level, but carbohydrate intake also affects the plasma lipid levels.[19,47] In this respect it should be pointed out that not only is the total quantity of dietary carbohydrate important, but also the nature of the carbohydrate which is consumed.[3,41] This is of special importance in a fruit diet for most fruits contain considerable quantities of fructose, glucose and sucrose.

In the present experiment the subjects from groups A_1, B and C received 36·7, 37·6 and 45% respectively of their calories from fat and 64·8, 64·7 and 52% respectively from carbohydrate.

Numerous factors affect the plasma cholesterol level, but the level is surprisingly constant in any one person. However, although the plasma cholesterol level is unaffected by minor day-to-day variations in diet or routine, it is changed gradually over several weeks by a change in the nature of the diet. According to Davidson *et al.*[17] the full effect of a change in diet on the plasma cholesterol level is usually apparent only after 2 weeks, and persists for at least 8 weeks. Whether these changes are permanent when a new dietary habit is maintained, is not yet known. The general pattern of the cholesterol changes noticed in the subjects of group A_1 seems to point in this direction.

While the initial mean cholesterol level of 224 mg/100 ml for our White subjects (group B) is comparable to the mean value of 234 mg/100 ml reported for White males in Cape Town by Brönte-Stewart *et al.*,[9] the initial mean value of 214 mg/100 ml for our Bantu subjects (group A_1) was considerably higher than the mean value of 166 mg/100 ml for Cape Town Bantu obtained by Brönte-Stewart *et al.*[9] This difference might be due to the fact that our Bantu subjects were on a balanced hospital diet for about 3 months before their participation in the experiment. In man the cholesterol content of the diet has apparently only a negligible effect on the plasma cholesterol level, while the fat content of the diet will gradually alter the plasma cholesterol level. According to Hegstedt *et al.*[28] controlled variations in the quantity and quality of dietary fats produce rather well-described and predictable changes in plasma cholesterol levels in man, while several workers have concluded that the type of dietary carbohydrate may participate in the control of the plasma cholesterol level.[3,39,41] In general, sucrose is thought to have a hypercholesterolaemic effect as compared to starch—or at least when an isocaloric substitution of sucrose is compared with complex carbohydrate from various plant sources, e.g. fruits, leafy vegetables and legumes.

Sugar intake was not restricted in our subjects. Being on a fruit diet they occasionally developed a craving for salt rather than for sugar. Hence their intake of refined sugar was quite low, their main source of carbohydrate being the various fruits.

Reaven *et al.*[47] suggested that the ability of a high-carbohydrate diet to stimulate hepatic triglyceride production and secretion may be directly related to the insulin response produced by the diet. The acute effect of glucose[23] or insulin in the postabsorptive state is to lower glyceride levels,[49] probably by facilitating peripheral clearance of glycerides through an increase in lipoprotein lipase activity.[33] However, there is some experimental evidence that insulin may increase hepatic triglyceride production.[48] Insulin may therefore have a dual role with the blood glucose level being an important balancing factor. According to Lees and Fredrickson[35] a high-carbohydrate diet containing a minimum of 7 g carbohydrate per kg body-weight per day resulted in a progressive rise in the serum triglycerides in normal people reaching peak levels after 7 - 14 days, but then gradually returned towards normal despite a continued high-carbohydrate intake.

In the present study the subjects of group A_1 ingested about 7 g carbohydrate/kg body-weight/day and the results obtained for the 5 subjects agree with the results reported by Reaven *et al.*[47] in that their triglyceride levels and insulin response decreased significantly. However, none of our groups showed a close correlation between triglyceride levels and the sum of the insulin response. The triglyceride levels of group B were much lower than those of group A_1 and showed no specific trend. The higher control triglyceride levels in the Bantu than in the White subjects are unexpected, for it is generally assumed that serum triglyceride levels are lower in Bantu and Coloureds than in Whites.[4,63] The high values of our Bantu subjects can probably be ascribed to their 3 months of hospital diet, their physical inactivity during hospitalization and perhaps their medication.

According to Ford *et al.*[22] there may also be a separate correlation between glucose and triglyceride concentrations, being relatively independent of body-weight and insulin levels. These workers inferred that triglyceride levels were dependent upon blood glucose as well as insulin levels. In the present study no significant correlation between glucose response and triglyceride levels was, however, noticed.

The decrease in the serum triglyceride levels of the subjects in group A_1 in the present study may in part be due to the high intake of unsaturated fatty acids. The avocado and nuts consumed supplied about 100 g of fat per day and approximately 63% of this fat consisted of unsaturated fatty acids. In the case of group B the intake of unsaturated fatty acids varied considerably from subject to subject. The consumption of high-fat diets rich in unsaturated fatty acids apparently maintains low plasma triglyceride levels, and lowers the lipid content.[4,56] The absence of significant changes in the triglyceride levels of the subjects in group B may be due to their low initial triglyceride levels and the relatively short period covered by the present experiment. According to Antonis and Bersohn[4] triglyceride levels are not significantly affected by the composition of the diet consumed over short periods.

The limited phospholipid studies carried out in the present experiment confirm the observations of Antonis and Bersohn[4] that a high-calorie, high-unsaturated-fatty-acid diet which lowers the serum cholesterol level, lowers the phospholipid level even more.

Plasma Proteins

The average protein intake of about 50 g per day, supplying 7·5% of the total calorie intake, was relatively low, and all the protein consumed was of plant origin. Yet the total serum protein did not decrease significantly in the Bantu subjects while a significant ($P<0·01$) temporary increase was noticed in White subjects. This raises the question as to what may be regarded as an adequate protein intake. The observations of Darling *et al.*[16] and Hegstedt *et al.*[27] suggest that the protein requirements of man are less than is generally accepted, and according to Best and Taylor[6] there is no accepted norm for protein intake. Since significant underweights (perhaps 10 - 15% less than the weight/height tables indicate) and hypoproteinaemia did not develop in our subjects, gross protein intake, in the main, could not have been seriously low.

On the other hand Louw *et al.*[38] found hardly any correlation between serum protein levels and dietetic factors, and these workers regard the total serum protein levels as a poor criterion of the nutritional status of a human subject. However, they did find a positive correlation between serum albumin levels and the intake of protein from animal origin. This correlation did not exist between serum albumin levels and the intake of plant protein.

Our subjects consumed only plant protein, yet the albumin levels increased significantly and in Bantu subjects the increase was maintained. Similarly the subject forming group C had a normal albumin level. This suggests that the supply of essential amino acids was also adequate. About 35% of the protein consumed was of nut origin and nut protein is regarded as first-class protein.

According to James and Hay[32] a decrease in protein intake is immediately followed by a reduction in the rate of albumin synthesis. However, the body tries to preserve its blood albumin level and this is done by transferring albumin from the extravascular to the intravascular compartment, and reducing the rate of albumin catabolism. The observations of James and Hay have been confirmed by Kirsch *et al.*[34] Although from a nutritional point of view one is probably not justified in applying results obtained on plasma albumin *pari passu* to body proteins in general, the circulating albumin mass reflects at least to some extent the quality and quantity of protein consumed in the present experiment.

According to Waterlow[60] if albumin constancy is not achieved, 'then one may suppose that the physiological range of adaptation has been exceeded. By contrast, the finding of a normal intravascular albumin mass with a reduced rate of catabolism would suggest a successful adaptation.' In the present study the albumin level was not only adequately maintained in Whites, but very efficiently raised and maintained in Bantu. As the circulating albumin mass in the normal White volunteers was most probably optimal when the experiment began, the explanation of their transient increase in circulating albumin is not clear. Our results are at variance with those of Louw *et al.*[38] who associate high circulating plasma albumin levels with a high animal-protein intake and a high plant-protein intake with low serum albumin levels.

The total circulating plasma globulin and β-globulin levels did not increase significantly in the Bantu subjects, but showed a statistically significant increase in the White subjects over the first 12 weeks of the experiment. Mack[40] reported a positive correlation between total circulating globulin mass and plant-protein intake. The absence of a significant increase in circulating total and β-globulin in Bantu subjects must probably be ascribed to the already high circulating mass of total and β-globulin in Bantu, for obviously there is a limit to which the different plasma protein fractions can increase. Once this limit has been reached no additional protein intake of whatever composition will induce further rise.

Scrimshaw[51] recently emphasized the importance of an adequate intake of essential amino acids together with an adequate intake of utilizable nitrogen for the synthesis of non-essential amino acids. For obvious reasons the amino-acid composition of animal protein corresponds more closely to the amino-acid composition of the human body than plant protein. Most plant proteins are deficient in one or more essential amino acids. The deficient amino acids can be supplied by other plant proteins supplying complementary amino acids; taking the deficient amino acids in synthetic form; animal protein; or consuming excessive amounts of plant protein so as to make up for the low levels in which some of the amino acids are present.

According to Scrimshaw[51] the average young man who is leading a fairly active life requires a daily protein intake of less than 30 g. This amount of protein is even adequate for young men who are physically very active provided that the total calorie intake is such that part of the protein is not required for calorigenic purposes and the protein is of good quality.

In the present experiment the nut supplementation of the fruit diet supplied amino acids on a complementary basis. In addition a great variety of fruit and a few types of vegetables could be eaten *ad libitum*. This probably explains why the subjects did so remarkably well on the diet.

Looking at the plasma proteins from a different point of view also suggests that the nut-supplemented fruit diet supplied protein, adequate in quantity and in quality. The plasma protein pattern of black Africans often differs from that of healthy white Africans, the main difference being a tendency to lower circulating plasma albumin and higher total globulin, especially of γ-globulin, in Blacks than in Whites. The total plasma protein is often reduced in black Africans, but sometimes the rise in globulins is enough to compensate for the low albumin. These racial differences appear after the first year of life.[31] Suggestions put forward to account for these racial differences include inadequate protein intake,[30] chronic liver disease,[5,24] tropical diseases,[50] and true genetic differences.[18]

In Bantu children suffering from malignant malnutrition or from nutritional oedema the total serum protein level is far below normal, mainly because the albumin level is greatly reduced; α- and β-globulin levels are usually about normal, while γ-globulin is both absolutely and relatively normal or higher than normal.[2] The serum albumin level rises rapidly on protein repletion.[8] In malnourished adults Stanier and Holmes[57] noticed a rise in serum albumin after a period, when these subjects were fed on foods with a high-protein content. However, the high γ-globulin level did not fall invariably. Schofield[50] studied West African residents in Britain and concluded that with time the protein pattern approaches that of healthy Europeans.

When the present study began the Bantu subjects had about normal total protein levels, but the albumin level was low and the globulin level, especially the γ-globulin, was high. The A/G ratio was less than unity. Although the fruit diet did not alter the mean total protein level of the Bantu substantially, their mean albumin level increased, while the mean total globulin level decreased. When these observations together with those of the other workers referred to are considered, one is justified in concluding that the present diet was neither inadequate in total protein nor deficient in individual amino acids—in fact it tended to 'westernize' the plasma protein pattern of the Bantu. In support of this conclusion is the slight change in the mean albumin level of Whites (Table VIII).

In conclusion it should be pointed out that under different nutritional circumstances the biochemical effects of a fruit diet would not necessarily be comparable to those found in this study, for it has been well established that the effects of a change in diet would depend on the degree of change (quantitative and qualitative), the duration, and on the individual concerned.

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