COCONUT OIL IN HEALTH AND DISEASE: ITS AND MONOLAURIN'S POTENTIAL AS CURE FOR HIV/AIDS*

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Introduction:

Folkloric and Ayurvedic writings are replete with accounts of the efficacy of the coconut for many ailments - from the cure of wounds, burns, ulcers, lice infestations to dissolution of kidney stones[1] and treatment of choleraic dysenteries[2]. The people of South Asia and the Pacific also look to the coconut as an important provider of food, drink and fuel, not to mention its many uses in industry. Hence, it has been called the *tree of life.*

More recently, Lim-Sylianco et al demonstrated in animals a powerful protecting effect of coconut oil against six powerful muta-carcinogenic chemicals, (such as benzpyrine, azaserine and nitrosamines). The protection was observed not only when coconut oil was given with the diet for several days before the mutacarcinogen but also when it was given in one bolus or dose with the mutacarcinogen[3,4]. In both experiments, coconut oil gave a significantly higher protection than soybean oil.

In another animal study by Lim-Navarro, et al [5], evidence for another protectant effect of coconut oil was obtained, i.e. significant prevention against shock in rats injected with E. coli endotoxin. The mechanism for these anti-inflammatory, antitoxic, antimutacarcinogenic actions are still not known.

Anti-Infective Action

In a series of papers published in the 70s, Jon J Kabara et al [6-10] and other workers studied the anti-microbial activity of various fatty acids. They found that the medium chain fatty acids (MCFA) with 6 to 12 carbons, possessed significant activity against gram positive bacteria, but not against gram negatives; they were also active against lipid coated viruses as well as fungi and protozoa. Saturated fatty acids, longer than 14 carbons long had no such activity. And of the MCFA, lauric acid (C12:0) was most potent, particularly in its monoglyceride form (monolaurin); it was more active than caprilic acid (C-8) caprie acid (C-10) or myristic acid (C-14). The dilaurin and trilaurin (di and triglycerides) had no activity. This finding has found use in the incorporation of monolaurin in cosmetic products and mouth washes; but although classified by the USFDA as GRAS (Generally Regarded as Safe), its oral use for systemic inflections has not been tried.

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HIV-AIDS Patients and the Coconut

According to Mary Enig[11], the AIDS organization, Keep Hope Alive, has documented several HIV-AIDS patients whose viral load fell to as low as undetectable levels, when they took coconut oil or ate coconut (half a coconut a day) or when they added coconut to their anti-HIV medication (anti protease and/or antiretrovirals) that had previously not been effective. The amount of coconut oil consumed (50 ml or 3 1/2 tablespoonfuls) or half of a coconut, would contain 20-25 grams of lauric acid, which indicates that the oil is metabolized in the body to release lauric acid and/or monolaurin.

The Monolaurin Trial on HIV-AIDS

The first clinical trial (pilot study) using Monolaurin for 6 months as monotherapy on 15 HIV patients was just completed [12]. These 15 patients (Table 1) ages 22 to 38 years, 5 males and 10 females, were all regularly reporting to San Lazaro Hospital, the hospital for infectious disease of the Department of Health. None of them could afford or ever received anti-HIV treatment. The males averaged 58 k in weight (49 to 68 k) and the females, 54 k (39 to 65 k). Seven showed elevated liver enzymes (ALT and AST) and 12 had unexplained eosinophilia. Two patients had high serum cholesterol and one had elevated triglyceride. No one had renal dysfunction. Their viral load ranged from 1,960 to 1,190,000 except for one patient (#94-022B) whose load was too low to count (below 400). This fact unfortunately was not determined before the random assignment of the patients to the 3 treatment groups. The monolaurin used was 95% pure. It was given in capsules, each containing 800 mg ML. The coconut oil was administered by tablespoonfuls.

The 3 treatment groups to which the 15 patients were randomly assigned were (Table II):

a) High Dose Monolaurin (HML): 7.2 grams (9 capsules) ML 3 times daily or about 22 grams daily

b) Low Dose Monolaurin (LML): 2.4 grams (3 capsules) ML 3 times daily or 7.2 grams daily.

c) Coconut oil (CNO): 15 ml 3 times daily or 45 ml daily. The ML content of this dose is about the same as HML.

All patients were observed daily for any side effects. Baseline, 3-month and 6-month laboratory examinations included: viral load (by PCR method), CD4 and CD8 counts (by-flow-cytometric method), complete blood count, tests for liver function (ALT, AST), renal function (urea N and creatinine), blood lipids (cholesterol, triglycerides, HDL) and body weight (k). Treatment benefit was defined as reduction in viral load and increase in CD4 count.

Tables II and III summarize the effects of the 3 treatment groups on the viral load, CD4 and CD8 counts. On the 3rd month, 2 showed decreased viral count with HML, 2 with LML and 3 with

CNO for a total of 7 patients benefited. The other patients all had increased viral load. Patient #94-022A continued to have undeterminable viral load and was excluded from the computation. On the 6th month, and end of the study. 8 of the 14 patients had decreased viral count, (2 of the 4 given HML, 4 of the 5 given LML and 3 of the 5 given CNO). The decrease in viral count was, however, significant only in 3 patients using the log Baseline-log 6th month ≥ 0.5 criterion. Two of these significant decreases were in the CNO group and one in the LML group.

The CD4 and CD8 counts (Table III) increased only in 5 patients and did not quite correlate with the fall in viral load, decreasing even when the viral load fell and increasing when the viral load rose. Patient #93006 had a steady viral load during the first 3 months but suffered a severe secondary infection in the 5th and 6th month, which caused the HIV infection to worsen despite fairly good CD4/CD8 response.

AIDS (CD4 less than 200) developed in 3 patients on the 3rd month of LML therapy (2 patients) and CNO therapy (l patient). The last mentioned patient (#86-001) died 2 weeks after the termination of the study. The patient under LML, however, fared better; one (# 93028) recovered by the 6 th month, and the other (#95052) was showing improvement of both CD4 and CD8 counts at the end of the study.

Eleven (l1) subjects gained weight - from l k to 23 k - including the 2 who developed AIDS and were recovering. The single AIDS fatality lost 6 k. The other 3 who failed to gain weight had decreasing viral and rising CD4 counts.

About one-half of the subjects in this study complained of feeling of warmth and a greenish hue to their urine (Table IV A), Both occurred at the beginning of the study and did not interfere with its continuation. Another 3 subjects had flaring up of their acne.

There were 11 subjects with eosinophilia at the start and 7 subjects with some liver dysfunction (Table 1). The treatment caused a rise of the eosinophilia in 7 of the 11, and a rise in ALT/AST in 3 of the 7 (Table IVA).

The patients with normal liver and kidney functions showed no effect from the treatments.

At the beginning, 2 subjects had elevated cholesterol and another one had high serum triglyceride (Table IVB). After 6 months, 4 patients had abnormal cholesterol and triglyceride, 3 had high cholesterol only and 2 had high triglyceride only.

Conclusion from the Study

This initial trial confirmed the anecdotal reports that coconut oil does have an anti-viral effect and can beneficially reduce the viral load of HIV patients. The positive anti-viral action was seen not only with the monoglyceride of lauric acid but with coconut oil itself. This indicates that coconut oil is metabolized to monoglyceride forms of C-8, C-10, C- 12 to which it must owe its anti-pathogenic activity.

More and longer therapies using monolaurin will have to be designed and done before the definitive role of such coco products can be determined. With such products, the outlook for more efficacious and cheaper anti HIV therapy is improved.

Anti-pathogen Mechanism of Monotriglycerides of MCT

The fact that monolaurin's activity is limited to lipid coated organisms (gram positive bacteria, enveloped viruses) suggests strongly that the relatively short C-12, C-10 or C-8 [Icelandic scientists have recently reported on the effectiveness of monocaprin (C-10) against HIV virus] probably exert their action on the lipid-layered coat or plasma membrane to destabilize it or even to cause its rupture. If this mechanism proves correct, monolaurin (and monocaprin and monocaprylin) could be bactericidal and could act synergistically with the present anti-HIV agents (the antiretrovirals and protease inhibitors).

Reprise

With all the opprobrium cast against it, it bears repeating again and again that no evidence has ever been presented to prove that coconut oil causes coronary heart disease in humans. All the evidences presented have been in various species of animals who were given coconut oil along without the necessary dose of essential fats or PUFA that should be given, just like the essential vitamins and minerals. On the contrary, the human epidemiologic evidence proves that coconut oil is safe. Coconut eating peoples like the Polynesians (Table V) and Filipinos (Fig. 1) have low cholesterol, on the average, and very low incidence of heart disease.

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Table I. HIV SUBJECTS: INITIAL FINDINGS ON ENTRY

	Patient	Age	Sex	Wt(k)	Cholesterol	Eosinophil	ALT/AST
1.	86001	38	F	39	3.7	8	20/14
2.	87006	33	F	44	3.4	5	20/11
3.	87015	36	F	64	5.0	2	20/12
4.	91008A	25	M	66	5.8	5	87/82
5.	93006	27	F	58	3.6	8	36/30
6.	93021	32	M	49	4.8	1	20/13
7.	93028	33	F	52	4.3	1	26/19
8.	93030	32	F	65	4.15	7	45/39
9.	93030B	31	M	56	3.69	6	39/32
10	94022	23	F	49	4.43	8	70/65
11.	94022A	27	M	52	3.95	5	26/20
12.	95017B	34.	F	64	4.7	1	460/450
13.	95052	31	M	68	4.3	4	38/31
14.	98056B	31	F	58	4.6	9	25/19
15.	98113	22	F	49	5.5	6	14/8
Mean					4.4	11	

Table II. EFFECT ON HIV VIRAL COUNT								
Patient		Viral Count (x $l0^3$)			% Change		(log) B – 6m	
		Baseline	3 mo	6mo	B-3m	B-6m		
HML								
87-015	36F	79.0	47.7	59.0	- 39.6	- 25.3	-0.13	n.s
91-008A	25M	18.1	12.6	54.7	- 30.4	+202.2	+0.48	sig
93-006	27 F	124.0	125.0	993.0	0	+700.8	+0.48	sig
94-022A	27M	< 0.4	< 0.4	< 0.4	--	--	--	
95-017B	34F	143.0	169.0	97.2	+ 18	- 32-0	-0.16	n.s
LML								
93-021	32M	365.0	705.0	308.0	+ 93.2	- 15.6	-0.07	n.s.
93-028	33F	22.0	105.0	42.5	+377.3	+ 93.2	+0.29	n.s
93-030	32F	105.0	172.0	94.0	+62.9	- 10.5	-0.26	n.s
95-052	31M	1,190.0	402.0	169.0	- 66.2	- 85.8	. 0.85	sig
98-113	22F	76.0	61.0	52.2	- 19.7	31.3	-0.16	n.s
CNO								
86-001	38F	808.0	683.0	463.0	- l 5.5	42.7	-0.24	n.s
87-006	33F	4.5	5.9	9.22	+ 31. 1	+104.9	+ 0.31	n.s
93-030B	31M	74.0	112.0	26.9	+ 51.4	- 63.6	-0.44	sig
94-022	23F	1.96	0.49	2.21	- 5.0	+ 12.8	+0.05	n.s
98-056B	31F	415.0	262.0	160.0	-531.3	-285.5	- 1.41	sig
				HML	(2)/4	(2)/4		
				LML	(2)/5	(4)/5		
				CNO	(3)/5	(3)/5		
					(7)/14	(8)/14	(3)/14	

Table III. EFFECTS ON VIRAL LOAD, CD4 AND CD8 COUNTS						
			B	3 mo	6 mo	B-6m CD4/CD8
HML	87 015	Viral Ct.	79.0	47.7	59.0	
		CD4	553	343	508	dec/dec
		CD8	1395	723	1190	
	9_1 008A	Viml Ct.	18.1	12.6	54.7	
		CD4	506	671	638	inc/inc
		CD8	842	1484	1044	
	93006	Viml Ct.	124.0	125.0	993.0	
		CD4	305	272	364	inc/inc
		CD8	1215	996	1362	
	9 022A	Viral Ct.	< 0.4	< 0.4	< 0.4	
		CD4	1065	888	896	dec/0
		CD8	659	619	662	
		95 017B	Viral Ct.	143.0	169.0	97.2
		CD4	432	457	544	inc/0
		CD8	1324	1246	1353	
LML	93 021	Viml Ct.	365.0		705.0	308.0
		CD4	575	377	512	dec/dec
		CD8	1698	1263	1660	
	93028	Viral Ct.	22.0	105.0	42.5	
		CD4	547	141	459	dec/dec
		CD8	1597	412	1423	(AIDS)
	93030	Viral Ct.	105.0	172.0	94.0	
		CD4	455	321	252	dec/dec
		CD8	1671	-628	448	
	95052	Viral Ct.	1190.0	402.0	169.0	
		CD4	470	168	L86	dec/dec
		CD8	1550	585	1025	(AIDS)
	98 113	Viral Ct.	76.0	61.0	52.2	
		CD4	396	386	501	inc/dec
		CD8	1187	737	226	
CNO	86 001	Viral Ct.	808.0	683.0	463.0	
		CD4	326	176	174	dcc/dec
		CD8	772	387	623	(AIDS) t
	87006	Viral Ct.	4.5	5.9	9.2	
		CD4	248	419	573	ine/Inc
		CD8	570	682	1267	
	93 030B	Viral Ct.	74.0	112.0	26.9	
		CD4	723	459	379	dec/dec
		CD8	1562	1056	826	
	94022	Viral Ct.	1.96	0.49	2.21	
		CD4	494	701	760	inc/inc
		CD8	795	844	920	
	98 056B	Viral Ct.	415	262.0	160.0	
		CD4	776	432	902	inc/inc.
		CD8	1663	943	2312	

Table IV-A. ADVERSE REACTIONS

	No.	%
Feeling of warmth (transient)	8	53
Greenish urine (transient)	7	47
Acne flare-up	3	20
Effect on Eosinophilia (l 1)		
Increase	7	
Decrease	4	
Effect on Liver Dysfunction (7)		
Improved	1	
Worsened	3	
No change	2	
No Effect on normal liver		
No effect on renal function		

Table IV-B. CHOLESTEROL/TRIGLYCERIDE/HDL										
		Baseline			**3 mo.**			**6 mo.**		
		Chol	**TG**	**HDL**	**Chol**	**TG**	**HDL**	**Chol**	**TG**	**HDL**
HML										
	87015	5.02	1.2	0.66	5.44	1.2	.02	5.81	1.0	0.8
	91008A	5.78	1.2	0.57	5.35	1.0	0.96	6.09	2.5	0.62
	93006	3.63	0.3	1.1	3.18	0.5	0.44	4.33	2.1	0.89
	94022A	3.95	3.2	0.63	5.15	2.3	0.86	4.82	4.6	0.6
	95017B	4.68	1.0	0.56	6.12	2.2	1.01	6.01	1.9	0.68
	Mean	4.6						5.4.		
LML										
	93021	4.82	0.6	0.63	3.71	0.9	0.88	5.0	.1.2	0.65
	93028	4.32	1.20	0.57	3.71	0.6	0.51	5.17	1.2	0.735
	93030	4.15	0.72	0.72	3.56	1.7	0.74	5.75	2.8	0,53
	95052	4.3	2.7	0.65	4.13	0.95	0.62	4.0	1.70	0.24
	98113	5.46	1.6	0.69	4.77	2.3	2.96	5.95	2.3	0.74
	Mean	4.6						6.2		
CNO										
	86001	3.71	1.5	0.63	2.57	1.1	0.62	5.71	3.2	0.36
	87006	3.35	0.6	0.57	7.24	1.4	0.87	4.65	6.0	0.93
	93030B	3.69	1.0	0.59	4.57	2.0	0.92	4.57	1.8	0.59
	94022	4.43	1.7	1.17	6.0	1.4	1.7	5.33	0.66	1.25
	98056b	4.59	1.8	0.75	5.14	1.0	0.72	5.13	1.0	0.63
	Mean	4.0						5.1		

HML - 22 g/d monolaurin
LML - 7.2 g/d monoiaurin
CNO - 45 ml coconut oil

Normal Values
Chol ≤ 5.2
TG ≤ 2.0
HDL ≥ 1.4

Table V
Coconut Diet - Polynesian Atolls

	Males		Female		Remark
	Pukapuka	**Tokelau**	**Pukapuka**	**Tokelau**	
Kcal	2120	2520	1810	2100	
Protein (g)	31	34	53	63	Mostly fish
Fat (total g)	83	156	80	131	Mostly coconut
% of total calories	35.2%	55,7%	39.8%	56.I%	
Fat, saturated (g)	63	137	64	120	Mostly Coconut
Fat, unsaturated. (9)	7	6	4	4	
Cholesterol (mg)	73	51	70	48	
Carbohydrate (g)	283	229	230	189	
Serum cholesterol (mg)	170	208	176	216	

I. A. Prior, et. al.
Am. J Clin Nutrition
34: 1552-61, 1981

Table VI

ATHEROSCLEROSIS

ATHEROGENESIS

FATTY STREAK

↓

Trauma
Platelet Aggregation
Monoclonal
Migration
Cholesterol
Deposition

FIBROUS PLAQUE

↓

Cellular Migration
Toxic Peroxidation

↓

SOFTPLAQUE

Necrosis
Plaque rupture

Thrombosis

Vasospasm

↓

ISCHEMIA

Occlusive
Thrombotic Plug

↓

INFRACTION

RISK FACTORS

Arterial pressure & turbulence	Smoking
Dyslipoproteinemia	Diabetes
Male gender	Hypertension
Menopause	Stress
Genetic	Lack of exercise
	Obesity

HEREDITY

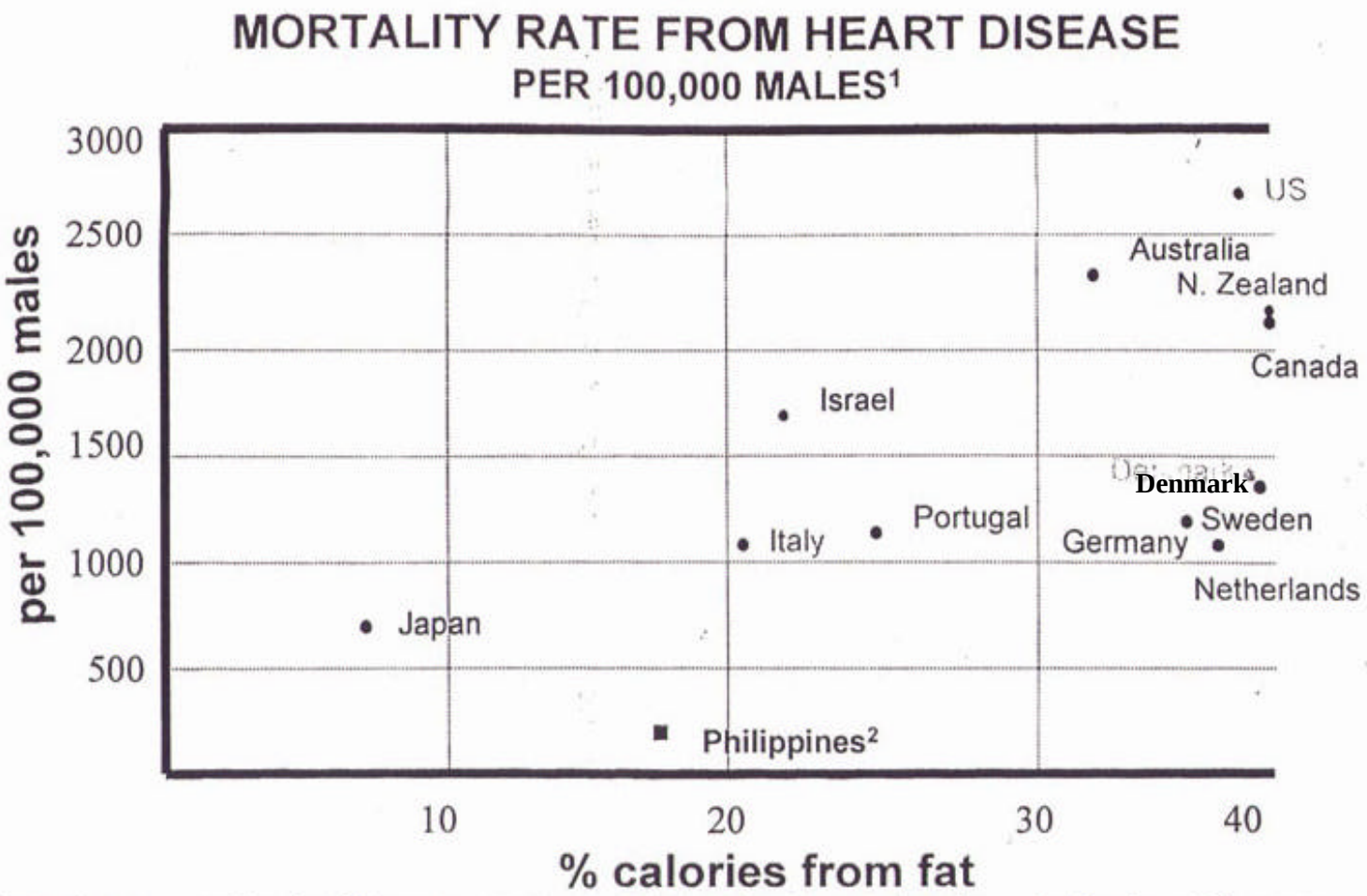
HDL-Low count
Small dense LDL - High count
Lipoprotein (a) - High count
Fibrinogent ↑

Excess of Polyunsaturated Fatty Acids (PUFAs) liable to peroxidation
Oxygen-free radicals

ENDOTHELIAL DYSFUNCTION

<u>Pro-Inflammation</u>
Adhesion Factors
Growth Factors
<u>Pro-Thrombosis</u>
Pro-coagulant factors
Anti-fibrinolytic factors
<u>Pro-vasoconstriction</u>
Endothelin-secretion
Nitric oxide-inhibition
<u>Coagulative Process</u>
↑ Fibrinogen
↓ Antithrombin

Figure 1



[1] 1950-52 Average Yearly of Hypertensive Heart, Rheumatic, Atherosclerotic, and other heart diseases.
[2] 1987 Phil. Health Statistics: Heart Disease (67.7) + Diseases of the Vascular System (52.1) = 119.8/100,000 population or 240 per 100, 000 males. (M:F = 1:1)

COCONUT OIL IN HEALTH AND DISEASE: ITS AND MONOLAURIN'S POTENTIAL AS CURE FOR HIV/AIDS

By

Dr. Conrado S. Dayrit*

ABSTRACT

The coconut is called the tree of life for it has been providing us, humans, food and drink, materials for housing, fuel and many industrial uses. And its medicinal uses are many and varied. The latest medical potential of products of the coconut first identified by Jon Kabara and others in the 70s, is the anti-bacterial, anti-viral and anti-fungal activity of its medium chain fatty acids, particularly lauric acid (C12:0) in its monoglyceride form (monolaurin or ML).

The first clinical trial ever of ML was on 15 HIV-infected patients reporting regularly at the San Lazaro Hospital, Manila who, never having received any anti-HIV medication, were randomly assigned to 3 treatment groups: 7.2 g ML, 2.4 g ML and 50 ML of coconut oil daily for 6 months. The San Lazaro Hospital Team was led by Eric Tayag.

Viral, CD4 and CD8 counts, complete blood counts, blood lipids and tests for liver and kidney functions were done at the beginning of the study and after 3 and 6 months of treatment. In one patient, the viral load was too low to count.

By the 3rd month, 7 of the patients (50%) showed reduced viral load and by the 6th month 8 patients (2 receiving 7.2h ML, 4 receiving 2.4 g ML and 3 receiving, coconut oil had a lowered viral count. The CD4/CD8 counts showed a favorable increase in 5 patients. There were no serious side effects observed.

Three patients developed AIDS on 3rd month of therapy when their CD4 count dropped below 200. One of these three, who was in the coconut oil group, died 2 weeks after the study. The two other AIDS patients were in the 2.4 g ML group; one recovered fully on the 6th month and the other showed a rapid return towards normal CD4 and CD8 counts.

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