

ATHEROSCLEROSIS IN THE MASAI¹

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Mann, G. V. (Vanderbilt Univ. School of Medicine, Nashville, Tenn. 37203), A. Spoerry, M. Gray, and D. Jarashow. Atherosclerosis in the Masai. *Am J Epidemiol* 95: 26-37, 1972.—The hearts and aortae of 50 Masai men were collected at autopsy. These pastoral people are exceptionally active and fit and they consume diets of milk and meat. The intake of animal fat exceeds that of American men. Measurements of the aorta showed extensive atherosclerosis with lipid infiltration and fibrous changes but very few complicated lesions. The coronary arteries showed intimal thickening by atherosclerosis which equaled that of old U.S. men. The Masai vessels enlarge with age to more than compensate for this disease. It is speculated that the Masai are protected from their atherosclerosis by physical fitness which causes their coronary vessels to be capacious.

atherosclerosis; autopsy; cholesterol; coronary artery disease; diet; exercise

INTRODUCTION

Studies of the pastoral Masai people of East Africa have been made to evaluate the soundness of the hypothesis relating diet to the causation of coronary heart disease. The hypothesis proposes that a large dietary intake of saturated animal fat causes hypercholesteremia which aggravates atherogenesis and leads to coronary heart disease (CHD). Keys (1), Kinsell (2), Ahrens (3) and Stamler (4) have been the main proponents. Programs to change the

Abbreviations: CHD, coronary heart disease; BI, brain infarction.

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diet to contain less animal fat and more vegetable fat have been encouraged (5). If this hypothesis were true, it should follow that the Masai people who live almost exclusively on meat and milk would be found to have high levels of cholesterol in blood and extensive CHD. We examined 600 genuine Masai with clinical methods, including 350 men over the age of 40, and found very little evidence of cardiovascular disease (6). We have found only one Masai man with unequivocal ECG evidence of an infarction. High blood pressure was unusual among those people and blood pressure did not increase with age. The level of cholesterol in serum was low, rarely exceeding 150 mg per cent and it did not rise with age in adults. Dietary evaluations showed a daily intake of 300 g of fat and 600 mg of cholesterol among young men, all of them from fermented milk and meat. Physical fitness was measured in 53 men and found to be exceptionally high (7). This could be only partially explained by extensive walk-

ing, which was documented with pedometers in 30 men and youths (8).

These facts reduced the investigation to one fundamental question: Do the Masai not develop atherosclerosis or do they have it but remain immune to occlusive disease because of some other protective circumstance?

The question was answered with autopsy material collected over a five-year period. The Masai do have atherosclerosis but they are almost immune to occlusive disease.

METHODS

A total of 50 hearts and aortae were collected from men dying suddenly. This was done by establishing eight collection stations in Masailand with medical aids equipped and instructed to obtain details of the terminal illness, the cause of death and the hearts and aortae "en block." The cardiovascular tissues were fixed in the field in neutral, buffered formalin and sent to the African Medical and Research Foundation in Nairobi. They were repacked and shipped to the Vanderbilt laboratory where they were dissected and the aortae were stained in Sudan IV in ethanol with the methods of the International Atherosclerosis Project (9). The stained tissue was pinned out on acrylic carpet and photographed in color with side lighting to reveal raised lesions. The color transparencies were projected (magnification $\times 2$) in a darkened room and the image was traced on paper to show the entire area of the vessel and the sudanophilic areas. These stained areas were measured with a planimeter and expressed as per cent of total area. Fibrosis was graded according to extent on a scale of 0-4. Complicated lesions, i.e. ulcerated or calcified, were noted. The aortae were divided for measurement into three regions: 1) *arch*—aortic ring to first intercostals; 2) *thoracic*—to superior mesenteric; 3) *abdominal*—to bifurcation.

The coronary system was examined with the methods of Young, Gofman and Tandy

(10). The coronary vessels were sampled by the plan shown in figure 1. The approximate distances of the coronary sections from the aorta are also shown in figure 1. The vessel rings were embedded in paraffin and sectioned for Gomori's and von Hoeff's stains. These stained sections were projected (magnification $\times 50$) and the image was traced on white paper. The areas were measured with a planimeter as indicated in figure 2. Since intimal thickness is correlated with vessel size (10), it is expressed as per cent of vessel area. Areas are in planimeter units (1 planimeter unit = 6.48 mm² magnification $\times 50$).

Additional portions of tissue were taken from the right and left ventricles, the pericardial surface and the aorta for stain with eosin and hematoxylin. Representative areas of aorta and coronary vessels were processed as frozen sections and stained with Sudan III.

POPULATION

The tissues were collected in regional hospitals and medical stations of Masailand in Northern Tanzania and Southwestern Kenya from the region of the Loita Masai. The collectors were instructed to accept Masai men: 1) who were killed or died within a week of an accident or acute illness; 2) with the distinctive Masai dress and foreskin who were known to have been living in the tribal setting. They were instructed not to collect tissues from subjects who died with wasting disease. Nevertheless, many of the men meeting these criteria were found to have chronic diseases. The clinical forms used inquired about these circumstances. Age was estimated by determining the man's initiation group and is accurate to ± 2.5 years.

RESULTS

The age distribution of the 50 subjects studied is shown in table 1. The causes of death are arranged by frequency in table 2. The weights of the entire hearts, after re-

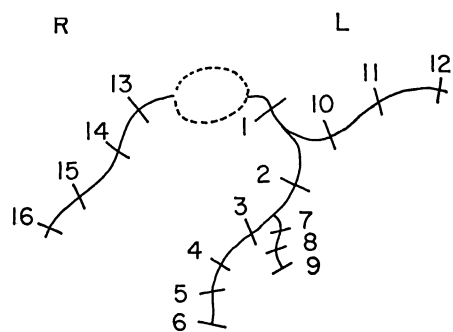


FIGURE 1. Coronary vessel—sampling. Approximate distances from origin. 1 cm from aorta: #1, #13; 3 cm from aorta: #2, #10, #14; 5 cm from aorta: #3, #7, #11, #15; 6 cm from aorta: #4, #8; 7 cm from aorta: #5, #9, #12, #16; 8 cm from aorta: #6.

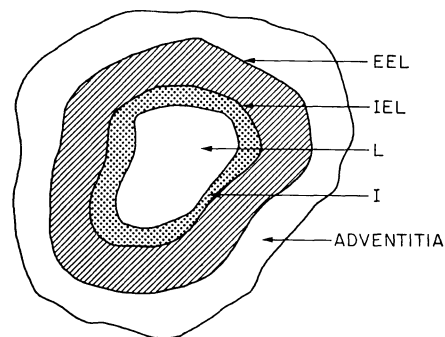


FIGURE 2. Criteria in measurements of coronary vessels. EEL is external elastic lamina. IEL = internal elastic lamina. L = lumen. I = intima and subintima. Vessel size is the area inside the EEL. Intimal area as per cent of vessel area = $(I/EEL) \times 100$.

moving the aorta, are shown in table 3, along with representative Western data. The heights and weights of these particular Masai men are not known but our earlier anthropometry (6) indicated adult Masai men are very near U.S. heights but about 10 kg lighter (171.6 ± 0.36 cm and 57.8 ± 0.372 kg). The Masai hearts, excluding those from subjects dying with malignancy, valvular or pericardial disease or congestive failure are smaller than Western hearts, perhaps a reflection of lower average blood pressure.

Ten of the 50 heart specimens showed evidences of disease or anomalies (table 4). Four were scarred in a manner typical of

rheumatic carditis. Two showed evidences of syphilitic aortitis and two had extensive pericarditis, probably causing their deaths. Three hearts with anomalous patterns of coronary vessels were found but in none was there evidence of myocardial disease.

The coronary vessels were classified according to the balance of the system judged

TABLE 1
Masai autopsy material

Age	No.
10-19	6
20-29	12
30-39	7
40-49	4
50-59	13
60-	8
Total: 50	

TABLE 2
Masai autopsy material

Causes of death	No.
Combat	15
Infection	13
CV renal	8
Accidents	6
Malignancy	3
Intestinal obstruction	2
Suicide	2
Diabetic coma	1
Total: 50	

TABLE 3
Masai heart weights (excluding 16 subjects)*

	No.	\bar{X} (g)	SD	Age (years)	Average age (years)
Masai	34	300	52	16-65	38
Comparative studies:					
Boston (11)†	26	328	62	21-60	40
London (12, 13)	695	345	61	25-55	41
	78	371	64	25-55	41.3
New Haven (14)	187	366	60.9	20-74	44.1

* Exclusions (for heart weight consideration only): malignancy—3; congestive failure—3; valvular heart disease—3; cor-pulmonale—4; pericarditis—2; incomplete specimen—1.

† Numbers in parentheses are references.

TABLE 4
Anatomical evidences of abnormalities in Masai hearts

Acc. No.	Sex	Age	Cause of death	Comment
Rheumatic heart disease				
10	M	60	Uremia	Mitral stenosis
30	M	50	Liver abscess, amebic	Aortic stenosis
33	M	12	Pulmonary edema	Mitral stenosis
36*	M	26	Cerebral malaria	Mitral stenosis
Syphilitic aortitis				
12	M	50	Congestive failure	Aortitis with dilatation
18	M	65	Volvulus	Aortitis
Pericarditis—constrictive				
15	M	25	Congestive failure	Cause unknown
41	M	60	Congestive failure	Cause unknown
Anomalous coronary vessels				
31	M	55	Renal cancer	Both coronary vessels arise on left
36*	M	26	Cerebral malaria	Predominant left anterior descending artery
42	M	60	Pneumonia	Absent right coronary artery

* Single specimen.

from vessel caliber, length and distribution (table 5). The Masai hearts were quite like those described in Western Europe and the U.S. (15-17). There was no evidence of genetic peculiarity of vessel pattern which might explain the Masai situation with respect to CHD.

Aorta—sudanophilia and fibrosis

The per cent of aortic surface involved with sudanophilia, i.e. fatty streaking, is shown in figure 3. The measurements indicate about equal involvement in each of the three regions of the vessels. Variance analysis showed no evidence of regional differences for either specific ages or for all ages combined. While there is a suggestion of a decrease of sudanophilia with age from ages 10 through 30, this is not statistically supportable with the variance encountered within these age groups. Neither was it possible to show an increase of sudanophilia after age 30. It is remarkable, however, that this degree of sudanophilia is found in a population where cholesteremia averages 125 mg per cent. Typical aortic surfaces are shown in figure 4.

Aortic fibrosis is classified for discussion as mild, i.e. grades 1-2 or severe, grades 3-4. Figure 5 shows a great increase of severe

TABLE 5
Masai-coronary artery balance: % distribution

	No.	Rgt. Dom.	Rgt. = Lft.	Lft. Dom.	Origin
Masai	50	47	30	23	Hamitic
%					
Comparative studies:					
Fulton (15)*	59	50	30	20	Scotland
Campbell (16)	100	72	14	14	London
Schlesinger (17)	225	48	34	18	Boston

* Numbers in parentheses are references.

fibrosis after age 40 ($\chi^2 = 16.0$). Up to 20 per cent of the subjects under 40 also showed extensive fibrosis. Complicated lesions in the aorta were very rare. Only three instances of calcified lesions were found and no ulcerated lesions were seen.

Coronary vessels

The intimal thickness of the coronary arteries is shown by age in figure 6 for all vessel areas combined. There is a significant gradient with age.

$$y = 0.22x + 25.7$$

The slope b with a value of 0.22 has a SD of 0.044 so that the value of t is 5.0 for the null hypothesis. The intimal thickness in

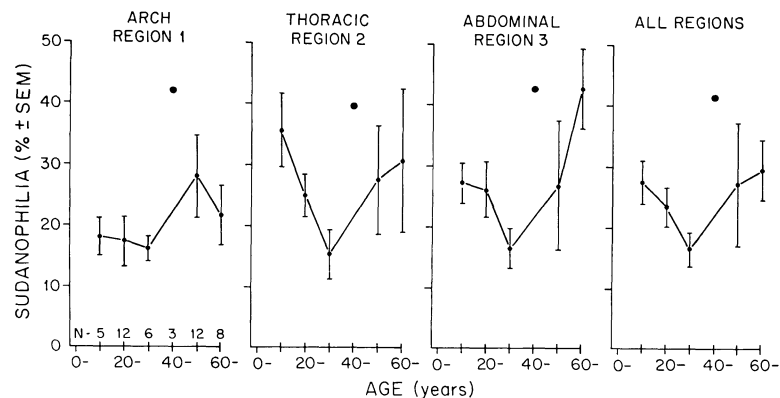


FIGURE 3. Masai aorta sudanophilia. Sudanophilia expressed as percent of area involved. The filled point for 3 men in the fifth decade does not follow the pattern of the others because of one exceptionally involved aorta in a man of 40 years, who died of pneumonia and liver cirrhosis.

the Masai material exceeds that recorded by the same methods by Young et al. (10) for U.S. subjects, age 60-69 years, shown to the right in figure 6.

The intimal thickness is shown by vessel in figure 7. This is expressed as I/E, the intimal area/area within the external elastic lamina, called E in the figure ordinate. All vessels show a gradient with age but some more than others. The coronary vessels in the oldest Masai are equal to the thickness found in California elderly subjects ($t = 1.03$, $t_{.05} = 2.06$) (figure 6).

Young et al. (10) emphasized the strong inverse relationship between intimal thickness of coronary arteries and the distance from the aortic origins. In figure 8, I/E expresses the intimal area/area within external elastic lamina. By assigning approximate distances to the sampling sites (figure 1), the relationship of I/E to distance shown in figure 8 was found. The Masai material also resembles the U.S. material in this respect. Intimal thickness is more extensive in the superficial and proximal coronary vessels.

Dissection of these Masai hearts gave the impression that the coronary vessels are ectatic and redundant. The measurement of vessel size, i.e. the area within the external elastic membrane, designated EEL in figure 2, is shown by age for the Masai material in figure 9. The vessels do get larger with

age; the increment is greater for the proximal large vessels, i.e. the left main and the right arteries, than for the more peripheral vessels. For all vessels the increase over the age span 2-7th decade is by a factor of 2.2. The slope of a line fitted by least squares to the mean vessel area for all sites by age is $12.3 \pm SD 3.1$. However, an *effective* increase of vessel size must be of lumen. This is shown in figure 10 by age. Lumen area increases with age but more for the initially large, proximal vessels than for the small, distal ones. The slope of the fitted line is $3.5 \pm SD 1.8$. The intimal thickness was also increasing with age and the slope (all sites) for that increase was $4.2 \pm SD 0.66$. The net effect of these increases is summarized in table 6. Thus vessel size increases enough with age in the Masai to more than compensate for the increase of the intimal and muscular layers of the vessels. The lumen is in fact larger in the old Masai than in the young. These relationships are shown graphically in figure 11.

DISCUSSION

Biss et al. (18) have published their findings with 10 autopsies of "Masai" done at the Narok District Hospital in Kenya. They described "a paucity of atherosclerosis" with only "occasional fatty streaks and fibrous plaques" in subjects presumed to be

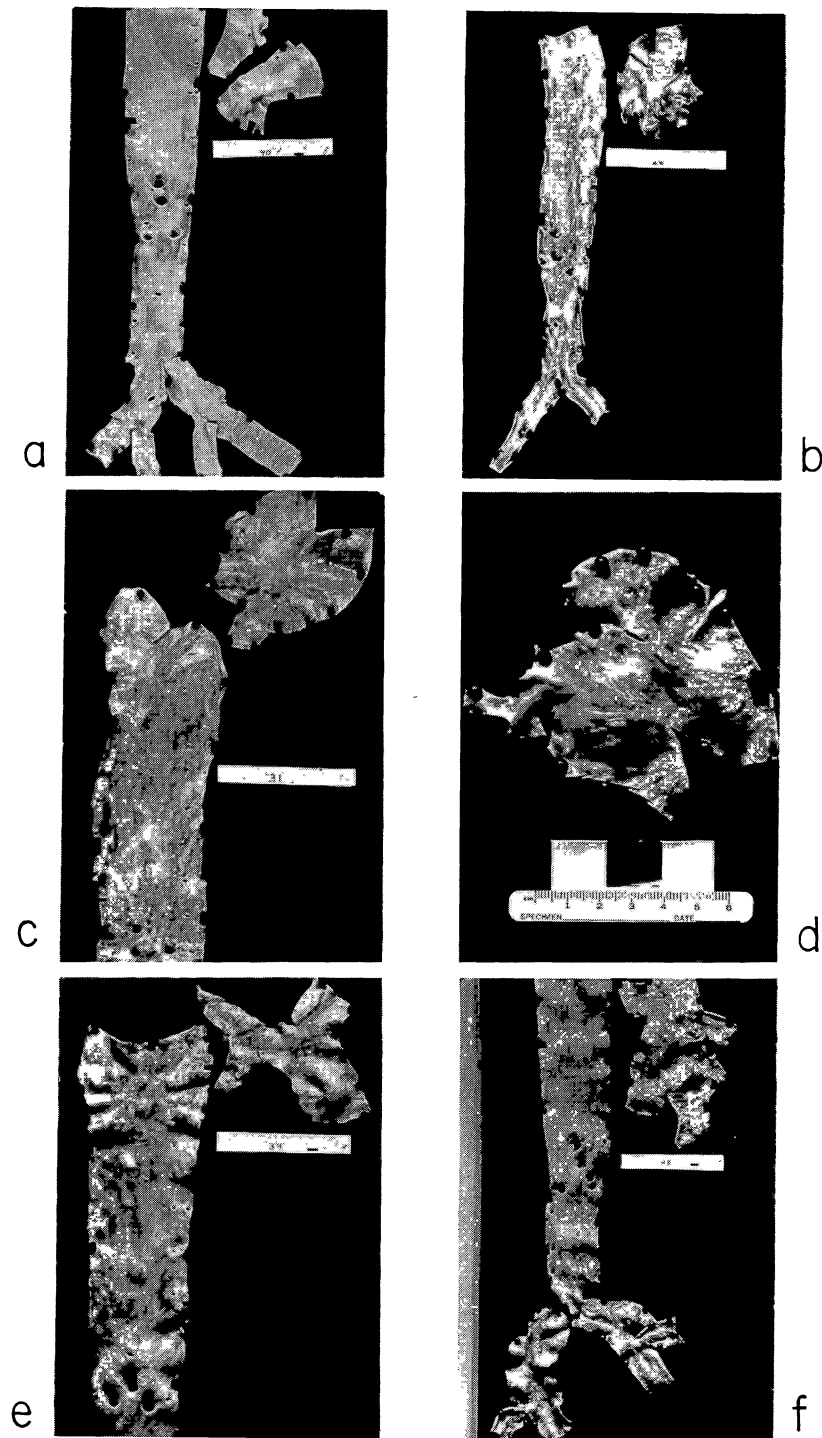


FIGURE 4. Representative examples of Sudan stained aortic tissue
 a) Mild sudanophilia, 25 year old male, acute leukemia.
 b) Moderate sudanophilia, 18 year old male, combat.
 c) Extensive sudanophilia, 55 year old male, renal cancer.
 d) Raised lesions, 55 year old male, pulmonary tuberculosis and hemoptysis.
 e) Extensive sudanophilia, fibrosis and raised lesions, 40 year old male with pneumonia and liver cirrhosis.
 f) Severe fibrosis and raised lesions, 65 year old male, uremia.

Masai. The authors did not give details of selection of the subjects, a description of the causes of death, the methods of evaluation or even the ages. They measured the thickness of the coronary arteries with a caliper and found that "the Masais' coronary arteries had much thinner walls than those of whites in the U.S., matched for age and sex." Those measurements were not shown nor was the comparison population

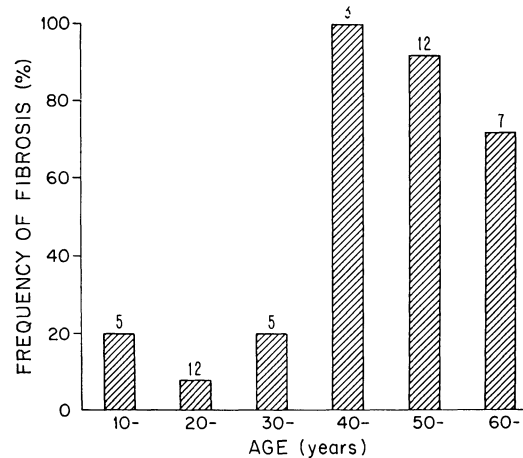


FIGURE 5. Masai—aortic fibrosis. Frequency of severe fibrosis, grades 3 and 4 by age.

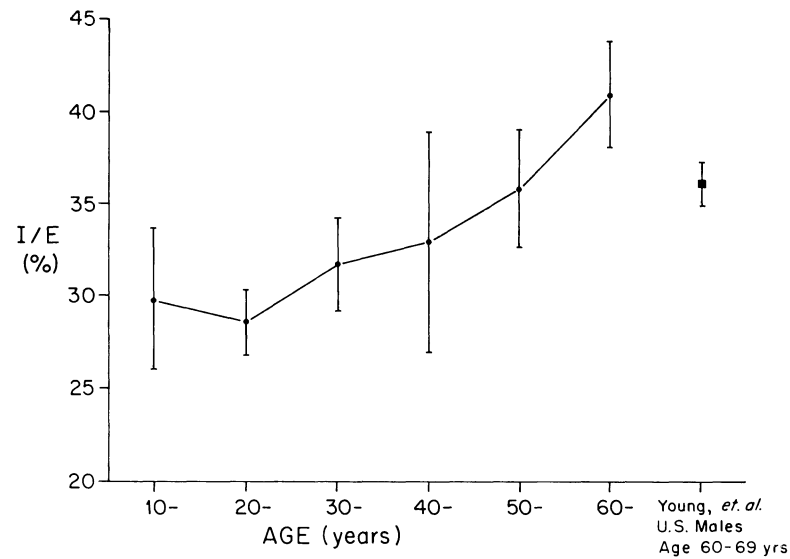


FIGURE 6. Masai coronary arteries—all sites—intimal thickness (\pm SEM). The progression of intimal thickness in the coronary arteries with age is shown for all coronary sites sampled. The U.S. experience described by Young et al. is shown on the right.

further described. The present data do not confirm either of Biss' statements. We find the Masai vessels do show extensive atherosclerosis; they show coronary intimal thickening which is equal to that seen in elderly Americans. The unique anatomical feature of the Masai material is that the coronary vessels enlarge with age so that the lumina are not compromised by intimal thickening.

The International Study of Atherosclerosis concluded (9), after reviewing the findings of about 23,207 autopsies collected from 19 geographic and ethnic groups around the world, that race per se did not determine susceptibility to atherosclerosis. Those workers could not show a relationship between atherosclerosis and diet or body size. They did see a sequential relationship between vascular fatty streaks, fibrosis and raised lesions and the prevalence of clinical CHD. They found a high negative correlation between the extent of coronary vessel lesions and the distance from the aortic origin both in the type and the severity of the lesions. They presumed that fibrotic plaques are derived from fatty streaks. The study indicated the development of atherosclerosis

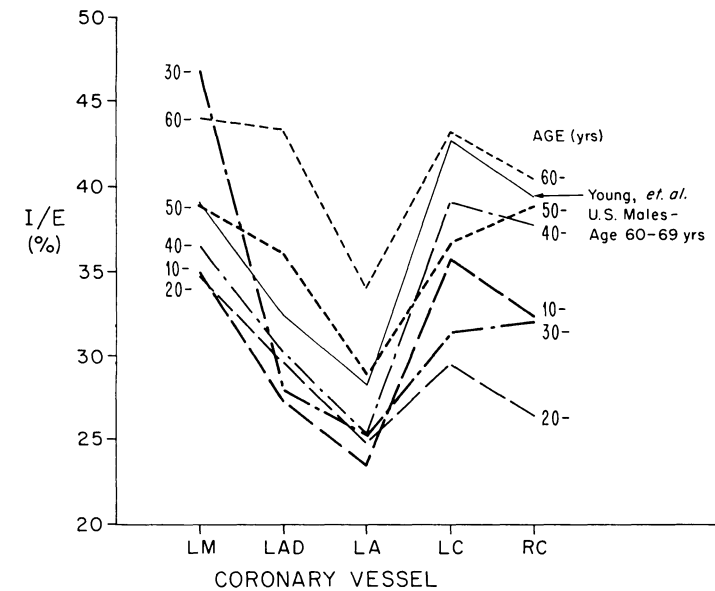


FIGURE 7. Masai coronary arteries—intimal thickness by site and age. The coronary vessels are: LM, left main; LAD, left anterior descending; LA, left anterior; LC, left circumflex; RC, right. Young's U.S. data are shown as the solid line. I/E = intimal area/area within external lamina.

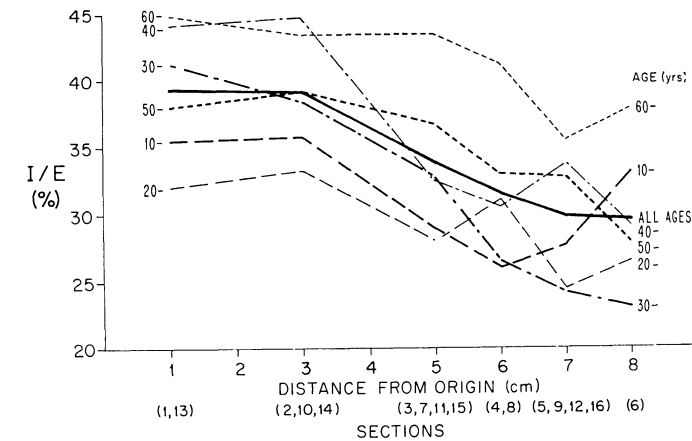


FIGURE 8. Masai coronary arteries—intimal thickness. Intimal thickening by approximate distance from origin of the vessel at the aorta. Section numbers in parentheses refer to the code in figure 1.

is similar in all geographic locations studied and, to a degree, in all races. The differences between groups were thought due to environment and certain unidentified factors which influence the quantity of atherosclerosis more than the quality. The Masai data conform with those findings.

The most striking thing about the Masai

data is the extent of arterial lesions in a population with consistently low levels of cholesteremia. Only the Ituri pygmies (19) have been shown to have lower levels but no autopsy material is available for those people.

The age trend of intimal thickening in the Masai is very similar to British material

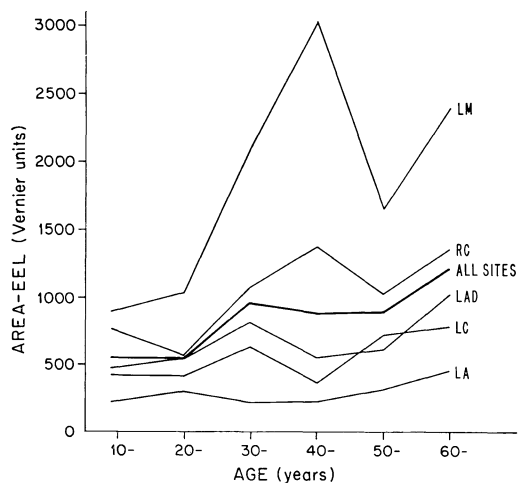


FIGURE 9. Masai coronary arteries—vessel size. Vessel size is the area in vernier units within the external elastic lamina.

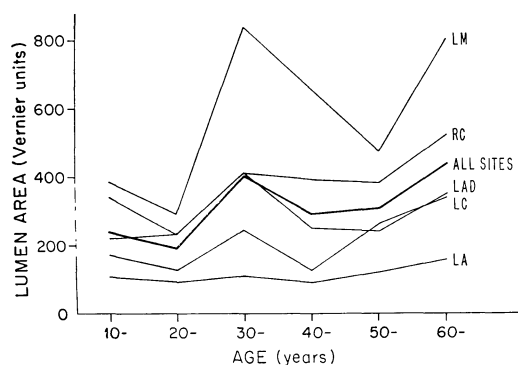


FIGURE 10. Masai coronary arteries—lumen. Lumen size by age averaged for the multiple samplings of each vessel.

(20) and in old age is equal to that of U.S. subjects of similar ages. However, the trend of decreasing sudanophilia in the second and third decades of life leads us to a more detailed examination of the Masai material in these and younger ages (figure 12).

The men are in the muran cohort from about age 12-30 years and during this time they adhere strictly to a diet of meat and milk. They are also maximally active at that time. Those men have significantly lower levels of cholesterol ($t = 4.08$, $t_{.05} = 1.97$) than do subjects after muranhood. We obtained blood from 44 Masai children

from ages two to 12 and those values with SEM are also plotted in figure 12. Children show a high (for Masai) cholesteremia in infancy which declines until about age four, to near adult levels. The babies are typically nursed to age two to three years and then fed the food available for women and elders. Cholesteremia increases in older children until puberty and reaches levels well above those of the muran. (Ages five to 12 vs. ages 14 to 29 years: $t = 4.00$, $t_{.05} = 1.99$). After

TABLE 6
A summary of the changes of coronary vessel parameters with age

Measurement	Slope of line expressing change with age \pm SD
Vessel size	12.3 \pm 3.1
Intimal area	4.2 \pm 0.66
Muscularis area	4.5 \pm 0.97
Lumen area*	
Predicted	3.6
Observed	3.5 \pm 1.8

* Average lumen area was predicted by subtracting the slopes of muscularis and intimal areas from vessel size. The observed area was as measured.

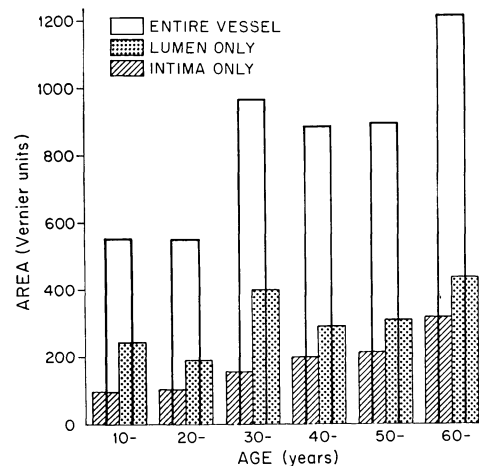


FIGURE 11. Masai coronary arteries—all sites. Each area measurement is plotted from zero. Entire vessel indicates area within EEL, the external elastic lamina. The area of muscularis is not plotted so that lumen plus intima does not equal EEL.

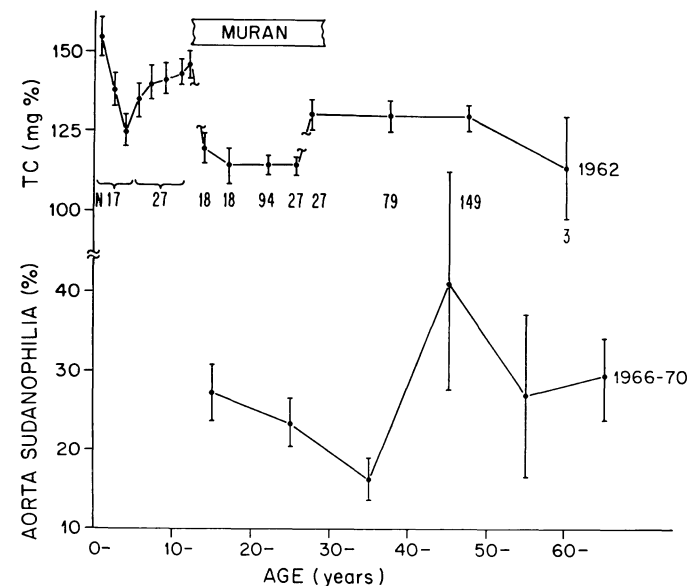


FIGURE 12. Masai-cholesteremia/Sudanophilia ($\bar{X} \pm$ SEM). The cholesterol data for 1962 are described in reference 6 and in subsequent unpublished studies (1964-1965). All subjects in the boma were measured. Number of subjects is shown for each mean value.

initiation and during the 15 years of warrior life, cholesteremia is at the lowest level. This is the time when fibrosis is about constant, but when coronary intimal thickness is increasing.

There are at least three possible explanations for these relationships: 1) The exclusive milk-meat diet may be displacing some dietary agent which is available to the children and the elders and that agent causes hypercholesteremia. 2) The warrior's life style causes the fatty streaks to develop fibrous caps which thereafter are immune to proliferation and occlusion. 3) The warrior's high level of activity and fitness causes hypocholesteremia and delipidation of fatty streaks which then return after age 30. The immunity to occlusive disease in later life is due to this postponement of the process for 15 years.

We believe the first is a probable explanation: namely, that the muran escapes some noxious dietary agent for a time. Obviously, this is neither animal fat nor cholesterol. The old and the young Masai do have some access to such processed staples

as flour, sugar, confections and shortenings through the Indian dukas scattered about Masailand. Those foods could carry the hypothetical agent.

One obvious explanation for the Masai immunity to occlusive disease is the compensatory increase of coronary vessel size with age resulting in a net increase of lumen size despite a tripling of intimal thickness during this time. This we attribute to high levels of exercise but we have no direct proof. There are scattered bits of experimental evidence to support this thesis (21-23).

The striking tendency of coronary disease to affect the proximal, superficial portions of the coronary vessels is a corroboration of the studies of Young (10) and the International study (9). This no doubt accounts for the early success of surgical bypass for occlusive disease of coronary arteries.

We have found no anatomical evidence of myocardial infarction in these hearts and we have seen only one man with ECG evidence (age 50, serum cholesterol 154 mg

TABLE 7
Population

	U.S. suburban (29)*	U.S. farmer (27)	Japanese (28)	Masai elder	Masai muran
Factor:					
Diet fat	+	+	0	+	+
Fitness	0	+	±	±	+
Cholesteremia	+	+	0	0	0
Blood pressure	+	+	+	0	0
Consequences:					
CHD	+	±	-	-	-
BI	±	±	+	-	-

* Numbers in parentheses are references.

per cent). The rarity of occlusive disease, coupled with the frequency and extent of atherosclerosis, suggests that exercise and fitness are the crucial protectants.

Taylor et al. (24) have repeatedly discussed their belief that the Masai immunity to CHD and hypercholesteremia is genetically determined. For at least 10,000 years the main occupation of Masai warriors has been raiding the neighboring tribes for cattle and women. Their success is evidenced by their huge herds and the variety of Masai physical appearances. They range in size and color far more than their neighbors. Casual observers mistake the Masai dress and manner for uniformity of physique. The Masai are one of the most genetically mixed groups in East Africa. The genetic argument is worthless.

The three noteworthy factors to be considered in the Masai immunity to CHD are fitness, which is remarkable, the hypothetical noxious dietary agent which is unidentified and the fibrous capping explanation which proposes that 15 years of muranhood in young adults allows early lesions to be capped and this gives immunity to subsequent progression. It would be of the greatest interest to add to the murans' diet artificial agents now present in the Western diet and to follow their cholesteremic response. The muran offer a unique test group because of their unusually simple diet.

These findings with the Masai material need to be considered in the context of other

cross-cultural information which bears on the problem of hypercholesteremia, atherogenesis and occlusive vascular disease. Kuller and Reisler (25) have proposed a general hypothesis to explain the behavior of CHD and stroke, i.e. brain infarction (BI). This suggests that the important determinants are the levels of blood pressure and blood lipids. When both are low, as in Central American Indians (26), both disorders are rare. When both factors are high, as in the U.S. Negro, both heart attack and stroke are common (27). When only blood pressure is high as in Japan, stroke is common but heart attacks are rare (28). When blood lipids are high and blood pressure is low, as in U.S. whites, CHD is common (29).

The hypothesis can now be amplified to include some new testable propositions (table 7).

These relationships suggest three conclusions: 1) High blood pressure is conducive to stroke; 2) high blood lipids are conducive to heart attacks; and 3) fitness protects against occlusive vascular disease in both the heart and brain.

The three intriguing variables that need study are the roles of fitness, the hypothetical dietary agent which leads to hypercholesteremia and an examination of the effects of early treatment of high blood pressure on the behavior of CHD. Fries' work (30) shows clearly that late treatment prevents congestive failure and stroke, but it has not changed the behavior of CHD.

The Masai evidence suggests that three preventive measures will be necessary to control occlusive vascular disease in the Western world: 1) prevent HBP; 2) maintain high levels of fitness; 3) prevent hypercholesteremia. Of these, the first is now a professional option requiring early detection and vigorous treatment, long continued. The second is immediately available as a personal option. The methods are well established (31). The prevention of hypercholesteremia is unsatisfactory. This will require more research.

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