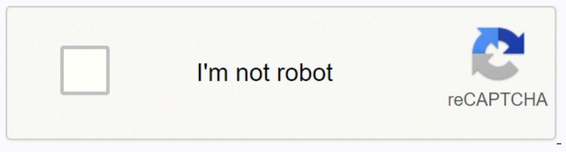
Which of the following is characteristic of amino acid supplements marketed to athletes



Next

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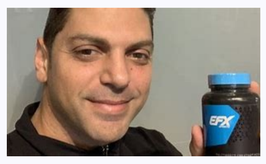

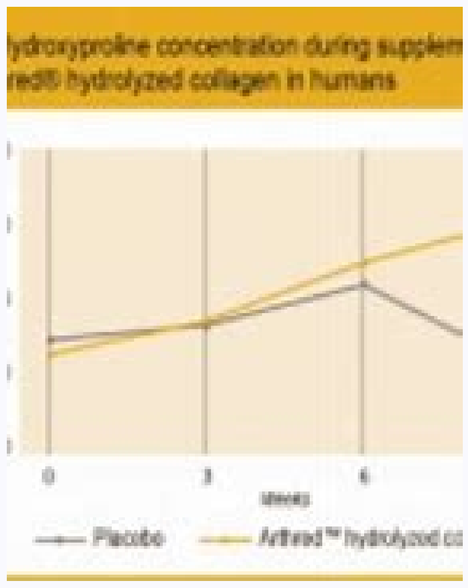

Which of the following is characteristic of amino acid supplements marketed to athletes quizlet.

Most of the major epidemiological studies focused on white men, but some have provided information about women and non-newspapers of both sexes.Total cholesterol (CT) TC is used in this chapter as an abbreviation for Total cholesterol in serum or plasma. The concentration of TC is generally expressed as cholesterol milligrams per 100 ml of serum or plasma (mg / dL). The concentrations of serum tc are about 2% higher than the corresponding plasma measurements (Folsom et al., 1983). Although this difference is considered to compare the results of the studies with each other when a number of subjects are large and small systematic biases may affect the comparison, it does not affect the main results or conclusions From studies discussed in this report where sane or plasma is used in cholesterol analysis. Thus, the TC is used interchangely for the total cholesterol of serum and plasma. In the last one, the TC, instead of lipoprotein cholesterol, it was measured in most epidemiological studies, because all trusted â € - to measure lipoprotein cholesterol in large numbers of people They were available. Therefore, most data on the risk of diseases are based on the level TC.Variação in the MST of TC between populations, the means of the TC vary widely among the populations. In the seven countries studies, the researchers studied 16 populations of middle-aged men residing in seven countries: Finland, Holland, Italy, Yugoslavia, Greece, United States and Japan (Keys, 1970, 1980b). Exam, laboratory procedures and quality control procedures were standardized. TC for men from 50 to 54 years ranged from 157 mg / dl in a Japanese population to 262 mg / dl in East Finland (Chaves, 1980b). In the Ni-Hon-San study, three samples based on the population of Japanese ancestrises were compared. TC's medical levels for men from 50 to 54 years were 182, 219 and 228 mg / dL In Hawaiah and California, respectively (Nichaman et al., 1975). In heart ischemic bone bone study, adjusted age adjusted The CT level in male public employees aged 40 years or more ranged from 195 mg/dl for those born in Africa to 219 mg/dl for those born in Central Europe (Kahn et al., 1969). Similar differences were found about 15 years later for adolescents and male and female adults in the Jerusalem Lipid Research Clinics Prevalence Study (Halfon et al., 1982a,b). Other differences between populations were observed for men in Puerto Rico, Hawaii, and Framingham, Massachusetts (Gordon et al., 1974), and for men and women in London, NÃ¡poles, Uppsala, and Geneva (Lewis et al., 1978). Some of this evidence is reviewed in the report of a Conference on the Effects of Blood Lipoproteins on Health (1979). The results of these various studies, particularly studies with migrants, indicate that the differences in ct© numbers among populations are largely due to environmental factors, especially diet, and not to constitutional factors.Large population differences in ct ©© were also observed among children and adolescents; the pattern of variation of these means a narrow parallelism with adult values, but at lower absolute values (Conference on Blood Cells in Children, 1983).Variation in CT rates among populations There are© also large differences between populations in ct and mortality and in the prevalence and severity of atherosclerosis. For example, in the Study of the Seven Countries, the 10-year focus of the first major CHD events (myodio infarct and coronãria death) among chd-free men at the entrance ranged from 3 in 1,000 in Crete to 107 out of 1,000 in eastern FinlÃ¢ndia (Keys, 1980b). The corresponding numbers for a 10-year CHD mortality were 0 and 68 out of 1,000, respectively. In the Ni-Hon-San Study, the relative risks of the first major CHD event were 0.46, 1.00, and 1.54 for cohorts in JapÃ£o, Hawaii, and CalifÃ³rnia, (Kagan et al., 1981; Marmot et al., 1975; Robertson et al., 1977). Focus of the first major CHD events among middle-aged men in It was double what in Puerto Rico and Havaan (Gordon et al., 1974) .Variação in Atherosclerosis Among populations in the International Atherosclerosis Project, The Extent of Atherosclerosis in the Coronary Arteries and Aortas was measured in 23, 207 Autopied People from 19 Populations in 14 Countries (McGill, 1968B). The percentage of intimate surface with increased injuries ranged from six% in Durban Bantu to 18% in New White Orleans. The differences between populations were perceptible in ages 15 to 24 and marked at ages 25 to 34. With few exceptions, classify the populations according to the extension of the increased lesions corresponded closely the classific Them by the CHD mortality rate. The Correlation Coefficient for the TC MEDICAL level with the 10-year mortality rates of CHD for 16 cohorts in the study of the seven countries was.82 (Keys, 1980b). Correlations between National TC and CHD mortality rates for these seven countries by 0, 5, 10 and 15 years after the TC measurement were.86, .90, .93, E.96, Respectively (Rose, 1982). In the international project of Atherosclerosis, there was a correlation of.76 between the extent of atherosclerosis and the concentration of the TC in 19 populations (Scrimshaw and Guzman, 1968). POPULATIONS WITH METHOD LEVERS OFFER TO 180 mg / dl are largely free of atherosclerosis and CHD, while those with MÃ- dial TC levels above 220 mg / dL are characterized by high rates of CHD (conference on health effects of bloody lipoproteins, 1979). These results sustain the conclusion that the variation of CHD rates among populations is determined predominantly by differences in the levels of incidency and mortality between individuals within the prospective studies of men Middle-aged, TC levels above 200-220 mg / dL are positively associated with the risk of CHD in the United States. Project Investigation Group, 1978; Stamler et al., 1986), as well as in Norway (Holme et al., 1981), France (Ducimetiere et al., 1980), Japan (Johnson et al, 1968), Israel (Goldbourt et al, 1985), England (Rose and Shipley, 1980), Italy (Italian National Research Council, 1982), Finland, the Netherlands, the Netherlands, the Netherlands © cia and SAN © Serbian (Keys, 1980b). The association may be weak or absent in some low-level populations © TC and low absolute risk of CHD, for example, in rural areas of B o snia and Croatia (Keys, 1980b; Kozarevic e t al., 1976, 1981).The results have been less consistent in relation t o the association of TC levels below 200 mg/dl with the risk of CHD. In fact, questions were raised as to whether the serum TC association with the risk of CHD © Continued or if there is any serum level TC below which is not related to the risk of CHD (e.g. Goldbourt, 1987). In four of the eight studies of the US Pool Project, the standard CHD age approach for men ages 40 to 64 years was smaller in the second quintile of the TC serum (194 to 218 mg/dl) than in the first Thursday (8194 mg/dl) (Pool Project Research Group, 1978). In the Study of Israel's Islamic Cardiac Diseases, of 9902, civilian male officials forty or more years old, mortality rates of the CHD fifteen years according to the qu ou of the serum (241 mg 2mg/dl) were 4.4.4.4.4.6, 7.10 and 100 respectively (Goldbourt). In the same study, however, the corresponding seven-year CHD mortality rates previously showed a growing pattern: 10, 12, 16, 17, and 30 by 1000 respectively (Yaari et al, 1981). Also © m, in this study, the rates of five-year onset for mycorrhium infarction were 29, 39, and 60 for 1000 for men in the Tertiles © rich from 77 to 189, 190 to 219, and 220 to 500 mg/dl respectively (Medalie et al, 1973). prospective © demonstrated a clear mononic association of CHD with TC paragraphs below 200/mg/dl. In Hiroshima Hiroshima Health study of 4,256 men age 40 and older, six-year-old standardized chd morbidity races associated with three levels of TC (220 mg / dL) were 72, 162, and 333, respectively (100 representing risk for the whole group) (Johnson et al, 1968). 10-year CHD mortality rates between 17,718 British public clients 40 to 64 According to the Quintupar of TC (233) were 28 , 34, 34, 44, and 54 per 1,000, respectively (Rose and Shipley, 1980). In the study Framingham, the Incidency of CHD of twenty years for men ages 33 to 49 According to the TC level (114 to 193, 194 to 213, 214 to 230, 231 to 255, and 256 to 514 mg / dL) was 86, 153, 220, 268 and 306 per 1,000, respectively (Kannel and Gordon, 1982). Between 356,222 men ages from 35 to 57 which were initially examined in the trial of intervention of risk factors of risk, the six-year mortality of CHD increased constantly according to the CT decipirtion from of 3 per 1,000 to TC 263 mg dl the data of this test are shown in Figure 7-1. In this test, the six-year mortality rate doubled between 153 and 226 mg / dl (3.16 to 6.94 per 1,000) and doubled again between 226 and 290 mg / dL (6.94 to 13.05 per 1,000). The weight of the evidence submits the idea that the TC level, at least from 150 mg / dl up, is positively in accordance with the risk of CHD. Since the Incidency of CHD is low on TC levels in 200 mg / dL, occasional exceptions to this rule are more likely due to the statistical artifact than to biological diversity. Results of the observations of the defendants in the intervention judgment of the multiple risk factor also indicated that the association between the risk of death of CHD by 23,490 black men was similar to that of 325,384 white men (Neaton et al, 1984). The association between CT and the relative risk of CHD decreases with age. combined results from five American studies, the relative risk of CHD in the highest fifth Tc (> 256 mg/ dl) in comparison with the lower quintil e (500 mg/ dl), and reduced triglycerides by diet and, if necessary, by drugs © (NIH, 1984) The Diet and Health Committee endorses these conclusions Versumy The relationship between the triglyceride levels © plasma ridges and cardiovascular diseases © a little controversial and unclear. In most population studies, triglycerin levels © Plasma rides were positively associated with increased risk of cardiovascular diseases, but they were not independently predictive for CHD after statistical adjustment adjustment -Attractions such a s TC, HDL-C, hypertension, cigarette smoking and obesity. indicated above, however, in the framingham heart study, plasma tri©glyceride scans were an independent predictor of CHD in Even so, the total level of plasma triglycerides, rather than a direct cause of atherosclerotic disease, probably reflects the presence of certain atherogenic lipoproteins. It is well known that VLDLs are highly heterogether; Various different types of lipoproteen particles are contained within this density class. Many pathological entities that elevate the levels of triglycerides, such as diabetes mellitus, nephrotic resemblance, chronic renal disease, and certain primary hyperlipidemias, lead to an increased risk of CHD. In these situations, the high triglyceric level can be a clue to the presence of other lipoproteenic anomalies that are more directly associated with CHD, such as low HDL-C, AP B or high LDL-C , or remains of rich atherogenic triglycerides in lipoproteen particles that have not been well defined. Thus, either the VLDL and triglycerides are or not directly involved in the atherogenic process, high levels may be osteable in the identification of people at increased risk of cardiovascular disease. It was identified and characterized. genuine disturbances of lipoproteen metabolism (Brown and Ginsberg, 1987; Stanbury et al., 1983). The study of these disturbances provided many knowledge about structure, metabolism, and regulation of lipoproteins and plasma apolipoproteins. Several of these disturbances are characterized by severe hypercholesterolemia and premature atherosclerosis and CHD. Such disturbances include familiar hypercholesterolemia (FH), familiar combined hyperlipidemia, and familiar disbetalipoismiamia (type 3 hyperloteinemia) .FH provides strong evidence that a high level of LDL per se is a cause of accelerated atherosclerosis and premature CHD. FH is caused by a defect in the coding of the gene for the LDL receiver - the cell surface receiver that usually removes the LDL from the circulation (Brown and Goldstein, 1986). The defective gene causes receiver is absent or not functional. A gene for the LDL receptor usually is © Wow. © of each parent. In the heterozygous form of FH, the patient has a normal and abnormal gene for LDL receptors, and LDL-C levels are approximately duplicated from levels greater than 200 mg/dl.Heterozygous FH occurs in the general population at a frequency of about 1 in 500, making FH one of the most common diseases of certain genes in humans. Fh Heterozygotes have CT levels that usually exceed 300 mg/dl and frequently have tendinous xanthomas, corneal arcus, premature CHD, and a strong family history of hypercholesterolemia. Approximately 5% of patients with myodio infarction before the age of 60 will have heterozygotes. Affected men usually develop CHD in their 30s and 40s; In women with FH, CHD usually occurs in their 50s and 60s. Rarely, about once a million people, individuals inherit two abnormal genes for LDL receptors and are therefore homozygous by FH. Patients with homozygous FH have CT rates ranging from 600 to 1,000 mg/dl, and usually have planar xanthomas and tubes, as well as tendan xantomas. Severe and often fatal coronary illness often develops while these people are in the age. A homozygote had an acute myodio infarction at 18 months of age; another died of an acute myodio infarction at 3 years of age. Very few FH Homomozygotes survive past age 30 (Goldstein and Brown, 1983). The fh animal homologue was identified in a tensit of rabbits called the Hyperlipidemic Rabbit (WHHL) Watanabe Hyperabidemic (Goldstein et al., 1983). These rabbits have very high levels of CT and LDL-C and develop severe atherosclerosis that is © similar to that seen in humans. This animal © model provides more strong evidence of causal relationship between high LEVELS LDL-C and atherosclerosis. High LDL-C levels due to an LDL receptor defect were© also observed in a Rhesus monkey family by Scanu et al. (1988). on FH and the LDL receptor provided insights into the mechanisms that may be involved in the common problem of moderately high levels of blood cholesterol that are observed in the general population. The potential relationships between LDL receptors, diet and atherosclerosis have been discussed by Brown and Goldstein (1986). It is likely that diet© components that increase plasma CT levels and LDL cholesterol (specifically, dietary SFAs© and cholesterol, as discussed in detail later in this chapter) act, at least in part, by suppressing LDL heponic receptor activity. Recent studies (Spady and Dietschy, 1988) in hamsters powerfully demonstrate the role of dietic© saturated triglycerides in increasing the effect of diet© © cholesterol on suppressing LDL hepotic receptor activity and in raising ldl-c plasmic levels. Small-scale tests (angiographic endpoints)Tests have been accumulated and several studies are ongoing to determine the effect of CT reduction by diet, drugs, or ileal bypass surgery in the progression or regression of athertoral injuries©© of coronary © and major, as determined by angiography and ultrasound. As demonstrated angiographically, in the first randomized controlled trial reported, patients with intermittent claudication had less angiographic progression and more plaque regression in the group treated with diet and drugs than a control group during a 19-month period. This was directly related to the change in the concentration of LDL-C plasma (Duffield et al., 1983). In a recently completed randomized controlled trial, in which angiographic techniques were used, patients treated with diet and various medications for CT reduction had less progression and more actual regression of plaques in natural and bypass vessels compared with controls during a 2©-year period (Blankenhorn et al., 1987). These results and those of other system studies cited below are generally with the conclusion that the reduction of LDL-C in hyperlipidal patients © mics reduces the rate of progression (and may induce regression) of atherosclerosis according to The degree of reduction of the lipids in the blood (Arntzenius et al., 1985; Brensike et al., 1984; Cohn et al., 1975; Kuo et al., 1979; Nash et al., 1982; Nikkila et al., 1984).Large-scale prevention tests (CHD End Points)Nine randomly controlled trials on the primary prevention of CHD were performed. The key elements of your drawings are summarized in Table 7-1. In five trials, several methods© were used to change the plasma concentrations of files as the only intervention; In two, a diet was used; And in three, drugs. The other four studies, in which the diet was also used, were multi©factor trials in which the effects of change in plasmic cells were confused with the effects of change in smoking and arterial pressure. Only one of the six trials involving changes in the diet the Los Angeles Veterans Administration Domiciliary Studyandorinha Haha was conducted under double-blind conditions (Dayton et al, 1968). In two studies, the Multifactor Trial of the World Health Organization (WHO European Collaborative Group, 1983) and the Finnish Mental Hospital Study (Hjermann et al., 1981) were randomly distributed. In the others, the individuals were anatorios. The number of participants in these studies ranged from approximatel©y 800 to nearly 50,000.Results of the trials summarized in Table 7-2 were listed according to the percentage difference of healthy © cholesterol that was created between the treatment and acommena commencing groups. (Wilhelmsen et al., 1986) at -15% in the Study of the Finnish Mental Hospital (Hjermann et al., 1981; Miettinen et al. (A negative sign here indicates that the experimental group had a lower © than the control group.) These results indicate that the magnitude of the chd risk difference varied directly with the magnitude of the difference in ct ©. Studies that have reached little or no differences in ct © had little or no difference in chd rate, while studies that a bigger difference in hand © day to day As indicated in Table 7-2, the differences in the CHD rates were statistically significant in five of the seven studies that achieved a difference of 8% or more in the CST © day. The number of major events in the other two studios in the entire co-operation of men in the Domestic Study of the Administration of Veterans of Los Angeles and in the sample of women in the Finnish Mental Hospital Study were relatively small, and the percentage differences in the CHD rates, Although comparable in magnitude to those that were statistically significant, they did not reach the criteria © of statistical significance.Combined with evidence from epidemiological studies, classics and laboratory animals, this evidence set unequivocally establishes that the reduction of CT alone © rich, particularly of the LDL-C TC, reduces the incidence of CHD on middle-aged men, hypercholesterols © mimes. The high-risk populations were used in most of these studies for very practical reasons; the cost and problems of conducting such a test in the population in general would be very large and the results might well be inconclusive. However, in light of the total set of tests, it seems reasonable to extend this conclusion to the general population of men and women with 200 mg/dl serum TC or superior. The results of the Test Domitionality of the Los Angeles Veterans'Administration support the conclusion that the conclusion can also extend © m to people over 65 years old, perhaps with a slightly smaller effect, but the evidence supporting this important issue is still weak. Most of the trials listed in Tables 7-1 and 7-2 showed an excess of deaths not related to DCH in the intervening group, in comparison to the comparator group. How much of this can be attributed to chance and if any of them reflect adverse effects of intervention are complex issues that can never be fully resolved. An analysis of 20 randomised controlled classical tests that Changes in serum TC as the only systematic intervention indicates that no Significant effect on the risk of death not caused by CHD (Richard Peto, University of Oxford, personal communication, 1987). In the trials presented in Table 7-2, none of the percentage differences in risk of death not related to CHD or total mortality was statistically significant, except in the case of the WHO Clofibrate Test. Following the final mortality, however, the Committee© of principal investigators (1978) of this study found that the excess mortality in the group treated with clofibrates did not continue after the end of treatment, and did not find a reasonable explanation for the excess mortality that occurred during the period of active treatment. The excess deaths were due to a variety of causes not related to CHD and had no apparent association, either with the extension of the reduction of the TAC or with the duration of clofibrate treatment. These results do not exclude the possibility of a toxic effect, but seem more consistent with the hypothesis that the excess of deaths in the treated group was the result of random sampling variation. The possible adverse effects of the drugs used in these trials are not strictly relevant to a report on diet and health and are not discussed anymore. In chapter 28 of this report, the security of© dietary changes to the lower © is discussed. There is no concrete evidence to indicate that any of the d©ietary interventions used in the trials described above increased the risk of deaths not caused by the HRC. In the Los Angeles Veterans Administration Home Test, there was an excess of cancer deaths in the experimental group that ate a diet high in PUFAs (in my© heart, 15% of calories such as PUFAs), but the difference was greater in the subgroup that adhered poorly to the prescribed diet than in the subgroup that adhered well (Pearce and Dayton, 1971). In addition© m, the results of other clinical trials did not indicate a cancer deaths in groups attributed to a high PUFA diet (Ederer et al, 1971). These findings support the conclusion that the excess of cancer deaths observed in the The study of Angeles did not result from the diet of high-PUFA.Rose et al. (1974) analyzed the data gathered from six prospective studies of CHD and found that the mother © the day of the TC of men who subsequently developed canon cancer was significantly lower than expected. The extensive literature however developed on this peak was reviewed by McMichael et al. (1984). In some studies, an inverse association was found during a prolonged period; in others, an inverse association was observed during the first years of follow-up, but disappeared during the continuous follow-up; in others still, no association was observed between the level © initial rich CT and subsequent risk of cancer. Some of the results may have reflected a proper effect ©- Classic serum cancer and some may have been due to metabolic characteristics (e.g. increase in the number of Boers) of people keeping the serum down, despite a high diet in SFAs and cholesterol. There is no evidence of studies in humans that the low consumption of SFAs and cholesterol are positively correlated with mortality rates for cancer of the dog (Liu et al., 1979). This evidence © discussed further in this chapter and in Chapter 28. Richard Peto (Oxford University, Personal Communication, 1987) states that the primary information to be obtained from the randomized classical trials © the time it takes for changes in the CT to have an effect on the CHD rates. He obtained information on 18 randomized trials published and 2 did not published which involved changes in the CT as the only systematic intervention, regardless of the mode of intervention (diet or medicine) and regardless of whether it was trials of disease prevention or treatment. Analysis of all data from these trials indicates that a 10%reduction in CT was associated with a manual reduction © 16% risk of CHD (confidence interval of 95% de 20.4%) in trials lasting 4 years. Peto also © m that the reduction of risk varied according to the duration of treatment: 11% 11% In 13 shorter trials and% 21 (�na, seeks 4%) in longer tests, for a reduction of ten% in CT. There were no tests that lasted a dance or more, but the results of the observation studies suggest that a reduction of ten% in tc over the dumps would be associated with a 33% Reduction of the Fatal CHD rate. Peripheral Vascular Developments (PVD) There are few systematic data on the relationship of bloodstream and lipoproteins with the prevalence or risk of PVD or disease The peripheral arterial of large vessels (pad). In the study framingham, the cumulative incidence of 26-years of intermittent claudication showed a concentration of cases in the part and superior distribution for TC values in men, but not in women (IE, 11.6% on the higher fifth versus 5.3% on the bottom) (Kannel and McGee, 1985). The results are, however, inconsistent between the studies. For example, serious cholesterol was strongly related to some studies (Bothig et al., 1976; Reunanen et al., 1982), but only weak or not related to others (Cavallo-perin et al., 1984; Davignon et al., 1977; Greenhalgh et al., 1971; Hughson et al., 1978; Isacsson, 1972; Sirti et al., 1974). You may be more related to a pad than HDL or LDL. In a British control of Control and cases, there was a suggestion that the levels of -betalipoprotein (ie, VLDL) and subjects were disproportionately high in patients with PVD, leading the authors To speculate on the differences in the course of time or gravity of atherosclerosis with this standard of bleeding in the blood, compared to the coronary disease (Greenhalgh et al., 1971). In any case, the overlapping was considerable, as a third of his patients with PVD also had CHD. The intermittent claudication aggregation with other atherosclerotic diseases was also very strong in the study framingham. Excessive coronary disease, cerebrovascular disease, or congestive cardiac insufficiency were found in a three cases of intermittent claudication at the time of diagnosis, indicating the strong common risk characteristics (Kannel and McGee, 1985) The connection in the basic processes leading to the manifest of PVD and CHD © Once again suggested by the discovery of a strong curvy relationship between a Framingham risk index involving several additional risk characteristics for coronary heart disease and the incidence over the intermittent cheering time manifested, mainly due to an excessive risk of intermittent claudition in the upper fifth of the coronary country (Kannel and McGee, 1985). Similar predominance of hypertriglyceridaemia has been observed in older men and women among healthy women. © ris of Swedish patients with PVD (Leren and Haabrekke, 1971). Given that the underlying pathogenicity of PVD and DCC © an atherothrombotic disease, a © Probable, but not established, that the same sketchy evidence for the diet relationship with the CHD blanket © m for developing too © Mm-hmm. That too. © m m © suggested by epidemiological evidence on developing countries, i.e. the positive relationship of the intermittent claudition with the index of risk of coronary illness in the Framingham population. Finally, although the pathogen of atherosclerosis is similar in the different arterial beds, the relative importance of different risk factors in the cause of the disease process clearly differs between different anatomical sites. This issue © discussed later on in Chapter 19.StrokeThere are few systematic data relating blood lipids and lipoproteins to stroke incidence. A U-shaped relationship between lipids and stroke was suggested in the Chicago Stroke Study, in which a lower risk of stroke was found in the central part of the TC (Ostfeld et al, 1974). This was most striking following Framingham from twenty years of the older population, ages 65 to 74, in which the greatest risk of stroke and acute brain attack was observed in the levels TC below 190 and above 295 mg/dl. This U-shaped relationship was stronger for stroke acute than for all strokes. In the same breath © there was a negative relationship between LDL-C and as well as for HDL-C and the general course (Kannel and Wolf, 1983). A similar U relationship was also © have been observed in the Honolulu Hearts Program, both for CT and LDL-C, while the HDL-C was negatively related and the VLDL was positively but not significantly related to the risk of stroke (D. Reed et al., 1986). The left side of the U-V relationship with the stroke in

the Honolulu Study was mainly due to the reverse relationship between TC and haemorrhagic stroke (Kagan et al., 1980). In later studies involving a better ability to discriminate between intracerebral bleeding and thrombosis via © The use of computerized tomography (TAC), emerge s a clearer epidemiological picture. For example, in recent data from the Myth Risk Factors Intervention Test in more than 360.000 screenees observed over ten years, there is a clear, positive, monotonic and linear relationship between the level of TC and the attributable brain thrombosis death (Iso et al., 1988). This explains the right-hand side of the TC U-shaped distribution to the general risk of stroke. A strong negative linear relationship was found between the TC level and the brain haemorrhage encoded deaths, the main explanation for the left side of the U-shaped stroke risk curve. The negative relationship between TC and brain haemorrhage was limited to people with CT levels below 160 mg/dl and diastolic pressures greater than 90 mm Hg (ISO et al., 1988). Hypercholesterolaemia has been the common denominator of experimental atherosclerosis in a variety of foams. © animal species since shortly after the turn of sound © culo (revised by Anitschkow, 1967; Duff, 1935; Jokinen et al., 1985; Katz and Stamler, 1953; Roberts and Straus, 1965; Strong, 1976; Wissler and Vesselinovitch, 1987a,b). In most of the species © cies, hypercholesterolaemia © Induced by a cholesterol-rich diet, SFAs, or both. Some foam © Animals, such as rats and dogs, are resistant to hypercholesterolaemia by diet and develop atherosclerical lesions when hormonal or other They are used in conjunction with the diet to induce hypercholesterolemia. When hypercholesterolemia is prolonged over several years, these animals develop all complications observed in advanced atherosclerosis in humans and possibly myocidadium infarction and other clinical manifestations of atherosclerosis in humans (Taylor et al, 1963). Not only hypercholesterolemia has been the sine qua non of experimental atherosclerosis, but the relation of the specific plasma lipoproteins with atherosclerosis in animals is similar human beings. For example, in humans and non-human primates, LDL is positive, and HDL is negatively associated with atherosclerosis. In parts of multiple regression, the plasma concentrations of LDL and HDL represent as much% of the variation in the extent of atherosclerosis (McGill et al., 1981a, 1985; Rudel, 1980). In some experiences, fansical characteristics of LDL, as the molecular size, they are also associated with atherosclerosis (Rudel et al, 1985). When diet-induced hypercholesterolemia is combined with other conditions known to increase atherosclerosis in humans, such as hypertension, the experimental lesions are also increased in extensions and gravity (McGill et al, 1985) . Thus, the association of altered concentrations of plasma lipoproteins in laboratory animals is not only strong and consistent, but the pattern of the association is similar to that of the human. In addition, many studies have demonstrated that the reduction of plasma cholesterol concentrations by withdrawal of cholesterol or fat, or both of the diet, or by drug administration, leads to Regressing of experimental atherosclerosis (magazine by Malinow, 1983). These regression studies, which were conducted in non-human primates, gave considerable support to the concept that the progression of atherosclerosis in humans can be retarded and possibly that the lesions can be reversed by the treatment of With diet and drugs. Drugs. atherosclerosis has provided an important basis for in vitro studies dealing with the cellular and molecular mechanisms of © neasing. These in vitro studies sought to determine how LDL and other athero©genic lipoproteins could lead to the formation of macrÃ³phages© (foam cells)© filled with colesterile and other elements of the atherorectal lesion. The results of such studies have, in turn, supported data on humans and animals that suggest a primary etiological role for LDL and related lipoproteins in the development of atherosclerosis. A prominent and early feature of atherlastic lesions © the© cell. In order to understand the mechanisms of foam© ing cells, the researchers studied the interaction of plasma lipoproteins with various types of cultivated macrophages. Native LDL does not lead to the accumulation of colesteril Ã©ster in many types of macrophages (reviewed in Brown and Goldstein, 1983). This lack of accumulation of native LDL co©sters is due, at least in part, to the decrease in LDL receptor regulation in these cells© by small amounts of excess cellular cholesterol, thus avoiding a large influx of LDL cholesterol. However, modified forms of LDL, such as acetyl-LDL, oxidized LDL, and malondialdehyde-LDL, lead to maciato-maciã§a© ster accumulations in cultured macrophages. These modified forms of LDL enter the cell© by a receptor (called scavenger receptor) that is© distinct from the LDL receptor and which is not subject to down-regulation regulation (Brown and Goldstein, 1983, 1986). Thus, the cellular influx of these modified forms of LDL continues at a high rate, leading to a significant accumulation of colesteril ©steres. These in vitro observations have given rise to widespread demand for evidence of LDL modification in vivo. Several recent studies have shed light on this tee. Pitas et al. (1983) demonstrated that macrÃ³phages of foam cells© of rabbit aorta atheroma explants have necrophagous receptors capable of binding modified LDL. Modified. et al. (1987) and Carew et al. (1987) took advantage of the in vitro discovery that drug probucol, which is © an antioxidant carried in LDL, prevents oxidative modification of LDL. These researchers demonstrated that treatment with WHHL rabbit stems with deficiency in LDL receptors decreased LDL absorption in foam© cell lesions and reduced the rate of development of these lesions regardless of plasma cholesterol levels. These results are consistent with the hypothesis that oxidative modification of LDL may be important in the development© of lesions of foam cells.Haberland et al. (1988) found immunological evidence for the existence of LDL-malondialin in atheosccerotic aortas. Malondialdede is © by-product of the metabolism of the aquidonic area, which is © active process in the arterial wall. Whether ldl-malondialis is involved in the formation of foam© cells in vivo has not yet been determined. A potential role for native LDL in the formation of foam cells has recently been suggested by findings that another cultured macro©phage, the J774 macrophage, accumulates large amounts of colesterÃlic Ã©ster in the presenãa native LDL (Tabas et al, 1985). In this case, the LDL receptor is poorly regulated by LDL-C due to intracellular deviation of cholesterol away from the regulatory pathway to an overactive cholesterol esterification pathway. Since LDL receptors are abundant in in© vivo foam cells (see below), indicative of a weak regulation of the LDL receptor, this mechanism of formation© of foam cells can help explain the athero©genic effects of native LDL©Foam cell formation has also been demonstrated in vitro by lipoproteins that do not native and modified forms of LDL. One of these lipoproteins, til-VLDL, ã© a lipoprotein enriched with cholesterol in the plasma of animals fed cholesterol and in the plasma of humans with dysbetalipoproteinemia familiar from the effectuation TIL-VLDL causes massive cholesterial ster deposit in macrómagos in vitro (Brown and Goldstein, 1983; Koo et al., 1986) and and the macrophage attaching itself to the same receiver as the native LDL, and this receiver demonstrates a hand regulation down (Koo et al., 1986). Receptors for MPL (and therefore for native LDL) are located in dog © ateroma foam squids of the rabbit's aorta (Pitas et al., 1983). These observations, together with the strong correlation between B-VLDL plasm a levels and atherosclerosis in cholesterol-fed animals and humans © Myths, indicate that squared VLDL is © atheogne © Single-disc-VLDL was found only in humans with family dysethoxyproteinaemia. However, the remains of kilometers, which occur normally in humans, interact with macrophages such as the anus-V-L and cause the accumulation of © cholesterile esters as well as a large amount of triglycerol © ridos (Van Lentern et al., 1985). These and other findings led some researchers to speculate that prandial lipoproteins, in particular cholesterol-enriched particles, such a s quil a remains, may be atheogenic. © the investigation of dog © foam squids © an active in vitro research a rea exploring the relationship between LDL and atherogen © nese. However, other areas of study also © concentrated on this relationship (revised in Steinberg, 1983). For example, LDL, whether native or oxidized, causes endothelial cell injuries in vitro. The endothelial slug, in turn, was placed the hypothesis of starting an atherogen © Causing the adhesion of platelets to the vessel wall and the release of growth factors. Hello. © In addition, the LDL has been reported to directly increase platelet aggregation and stimulate dog growth. © Smooth muscle cells, two important characteristics of atherosclerical slug. It's obvious, hello © m that, what other components of the dog © pots and walls, not yet well defined, are also © m important factors in atheognomy © nese. The Daddies © is potential growth factors, endothelial slug and different dogs © Blood cell arteries in the pathogen © Athrosclerosis was revised by Ross (1986). In summary, numerous in vitro studies demonstrated the ability to and related lipoproteins to cause dog formation © foam squids and other elements of atherosclerosis. Although there may be other important metaphorical, cellular and vessel wall factors in atheogenesis, in vitro evidences strongly suggest an important causal role for LDL and related lipoproteins. © plasma lipid and lipoprotein and CHD rates. The mom levels © population bodies TC also © differ between adults and children; children in parallel to adults with absolute lower values. The population correlations between the risk of CHD and the level of © TC dio are strong, but are weak for LDL levels, triglyce © The TC media and the CHD rates among migrants change to those in the adopted country, whether they are higher or lower than the countries of origin. positive and contiguous. The individual HDL-C level is inversely related to the risk of CHD in studies in the United States, Norway and Israel, but not in Finland and the USSR. HDL-C population means that they are weakly correlated, if any, with population levels TC and LDL-C resulting from altered diets and medicines and decreases in the incidence of CHD, the tests show a consistent relationship between TC and LDL-C decreases in levels of diets and medicines; The magnitude of the risk difference varies directly with the magnitude of the difference between the CST © dio and the experimental. The differences in the CHD rates between the experimental and control groups were statistically significant in five of the seven trials which reached a range of 8% or more in PMC © Hate. The magnitude of the benefit that can be expected from the reduction of TC and LDL levels can be estimated from epidemiological studies of observation and classical trials. The estimates of these different types of studies are consistent in projecting that for individuals with TC levels initially in the range at 300-mg / dl, every reduced or 1% I TC TC Reduces CHD rates for approximately 2%. Thus, observation studies and clinical trials indicate that relatively small differences or changes in the TC's medical level have a relatively large population effect or impact on the public health on disease. In addition, the tests indicate that the risk can be reduced in individuals with a high moderate risk and that much of the effect of a lower risk on the risk is seen only after a few years. years old.

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