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Super Foods Dietary Approaches for Acute Myocardial Infarction?

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Abstract

The role of diet in acute myocardial infarction is not known. Increased consumption of proinflammatory macronutrients such as w-6 fatty acids, trans fat and saturated fatty acids (SFA) as well as refined carbohydrates may produce oxidative stress, free fatty acids and proinflammatory substances, which results into endothelial dysfunction. Glucose ingestion in normal subjects is associated with increased superoxide generation in leukocytes and mononuclear cells, as well as with raised amount and activity of nuclear factor-κB (NF-κB), a transcriptional factor regulating the activity of at least 125 genes, most of which are pro inflammatory. Increased consumption of refined carbohydrates also causes an increase in two other pro-inflammatory transcription factor, activating protein-1(AP-1) and Egr-1, the first regulating the transcription of matrix metallo-proteinases and the second modulating the transcription of tissue factor and plasminogen activator inhibitor-1. It is possible that simultaneous supplementation with antioxidants such as coenzyme Q10 and w-3 fatty acids can modulate some of these mechanisms, resulting into decrease in these adverse effects.

Keywords: Heart attack, acute coronary syndrome, unstable angina, inflammation.

Introduction

Although, adverse effects of western diet are known [1,2], there is no guideline for dietary advice in patients with acute myocardial infarction(AMI) [1-3]. Most cardiologists and dietitians continue to prescribe refined starches; bread, biscuits, butter, cakes, to these patients without bothering about the adverse effects of such foods [1,2]. A recent study(4), among 54 patients of acute myocardial infarction(AMI), showed that the intake of large meals and large breakfast >1000cal especially rich in trans fatty acids(TFA) was significantly associated

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with AMI compared to control subjects. Those consuming large meals, showed significantly greater levels of tumour necrosis factor-alpha (TNF-α) and interleukin-6(IL-6)) compared to subjects taking small breakfast. In another study (5), among 202 patients, large meals was a trigger for development of AMI among half of the patients. TNF-α and IL-6, incidence of known hypertension and type-2 diabetes were significantly greater among AMI patients compared to healthy subjects. These proinflammatory markers are risk factors of reinfarction and cardiac mortality among these patients [3-8].

AMI is associated with acute hemodynamic disturbances, endothelial dysfunction, and tachycardia, characterized with hyperglycemia, hyperinsulinemia, hypertriglyceridemia, free radical stress, rise in proinflammatory cytokines, free fatty acids and a hypercoaguable state which are predisposing factors for disruption of atheroma plaques, known to result in to reinfarction and death. There appears to be an acute generation of a proinflammatory milieu, in patients with AMI, which in turn may become worst due to increased consumption of proinflammatory foods, resulting into greater incidence of complications and deaths. However, consumption of fruits, vegetable and nuts and oils rich in monounsaturated fatty acids and w-3 fat (Columbus oils(olive +lin seed oils), due to increased content of phytochemicals and slow absorption, may be protective [3,6-13].

Columbus Concept

Columbus foods are natural wild type foods, rich in phytochemicals which are slowly absorbed without causing any abnormal increase in blood glucose, insulin, proinflammatory cytokines and free fatty acids [14-17].The Columbus® Concept stands for a return of alpha-linolenic acid (ALA, C18:3ω3) - herein referred to as *wild*- or *game*-type land-based fatty acid - into the feed ration of land-based bred animals to such an extent that their fat depots (white adipose tissue) exhibit a balanced ratio of *essential* fatty acids, i.e. ω6:ω3 = 1:1, characteristic of fat depots in wild animals or game [14-17]. This return to the wild standard translates into a substantial reduction in long chain omega-6 fatty acids and a moderate species-specific increase in long chain omega-3 fatty acids in organs and peripheral tissues of these domesticated animals or livestock. The ω6:ω3 = 1:1 ratio is also taken as reference for the design of composite plant-derived table oils and fats as these represent other primary sources of energy in the modern human diet. Provided particular attention is drawn to the antioxidant content of such foods, the twofold end results are [17] a return to animal and plant food supplies in better compliance with human genetic heritage and a possible rehabilitation of dietary cholesterol and saturated fats (former CSI, C: Cholesterol, S: Saturated fats, I: Index).

Taken within a larger context, the Columbus® Concept stands for the return of a specific healthy cholesterol into men's food supply and blood stream, the so-called wild or game cholesterol that is associated with a dietary balanced essential fatty acid ratio (ω6:ω3 = 1:1). As observed from the overall distribution of fats in a natural untamed environment, nature recommends the consumption of a balanced ratio of saturated and polyunsaturated fatty acids as part of a dietary lipid pattern rich in monounsaturated fatty acids (P:M:S = 1:6:1). The Columbus® hypothesis is that when our diet complies with nature's recommendation in terms of *essential* nutrients (including some minerals and vitamins, amino and fatty acids), then dietary cholesterol becomes healthy; this would also stand for blood cholesterol and other lipoprotein fractions. Since the industrial revolution, we have a tendency to consume more saturated and less polyunsaturated fatty acids, and the latter are nearly exclusively represented by the omega-6 family. Therefore, our diet is deficient in ω3 fatty acids as well as in antioxidant vitamins and minerals as we get modernized.

Cereal grains as staple food are relatively recent addition to the human diet and represent a dramatic departure from those foods to which we are genetically programmed and adapted. Prior to the Agricultural Revolution, humans ate an enourmous variety of wild plants, whereas today about 17% of plant species provide 90% of the human food supply, with the greatest percentage contributed by cereal grains [17].Grains are high in starch and omega-6 fatty acids and low in omega-3 fatty acids and other phytochemicals, compared to green leafy vegetables. Recent studies indicate that low fat high carbohydrate

diets increase insulin resistance and hyperinsulinemia, that are known to increase the risk of coronary artery disease(CAD), type 2 diabetes mellitus and hypertension(17).However, if the cereal grains are used as dry whole grains, such as in South Asia, these grains may retain w-3 fat and are protective against diabetes and cardiovascular disease(CVD)because they are slowly absorbed and do not cause hyperglycemia and hyperinsulinemia but can decrease inflammation [18].

Mechanisms

Increased consumption of proinflammatory macronutrients such as w-6 fatty acids, TFA and saturated fatty acids(SFA) as well as refined carbohydrates may produce oxidative stress, free fatty acids and proinflammatory substances, which results into endothelial dysfunction [1,2,8,13].Glucose ingestion in normal subjects is associated with increased superoxide generation in leukocytes and mononuclear cells, as well as with raised amount and activity of nuclear factor-κB (NF-κB), a transcriptional factor regulating the activity of at least 125 genes, most of which are pro inflammatory(1,2) . Increased consumption of refined carbohydrates also causes an increase in two other pro-inflammatory transcription factor, activating protein-1(AP-1) and Egr-1, the first regulating the transcription of matrix metallo-proteinases and the second modulating the transcription of tissue factor and plasminogen activator inhibitor-1 [1,2,13]. It is possible that simultaneous supplementation with antioxidants such as coenzyme Q10 and w-3 fatty acids can modulate some of these mechanisms, resulting into decrease in these adverse effects. A mixed meal from a fast-food chain has also been shown to induce activation of NF-κB associated with the generation of reactive oxygen species (ROS) by mononuclear cells. Superoxide anion appear to be an activator of at least two major pro-inflammatory transcription factor , NF-κB and AP-1.These observations are consistent with previous findings, demonstrating that after oral or intravenous glucose challenges, in both normal subjects and patients with type 2 diabetes mellitus, there is an increased generation of ROS and raised circulating levels of proinflammatory cytokines, such as TNF-α,IL-6 and IL-18 [1,2,8]. In apparently healthy subjects, a single high-fat meal produces endothelial activation, as evidenced by increased concentrations of the adhesion molecules VCAM-1 (vascular cell adhesion molecule-1) and ICAM-1 (intercellular adhesion molecule-1), in association with raised plasma concentrations of IL-6 and TNF-α.[7-9].A high-fat meal(1,2) may increase the circulating levels of IL-18, a pro-inflammatory cytokine supposed to be involved in plaque destabilization associated with the simultaneous decrease of circulating adiponectin, an adipocyte-derived protein with insulin sensitizing, anti-inflammatory, and antiatherogenic properties. Further proof for the Columbus paradigm has been provided in more recent studies on the effect of meals on risk factors of atherothrombosis. Consumption of a high-fat meal together with vegetable foods rich in natural antioxidants largely prevent the negative effects on endothelial function [1,2,6-8]. In particular, endothelial dysfunction acutely triggered by the consumption of a high-fat meal rich in saturated fatty acids is reduced by the simultaneous consumption of a vegetable serving including pepper (100g) tomatoes (100g), and carrots (200g). It seems that these foods are slowly digested and absorbed without causing any significant increase in free radical stress and free fatty acids which is characteristic of Columbus foods.

It is possible that foods act as drug. We want to eat those foods which are inspired by our brain. In this mechanism taste is very important, which is observed in experiments with rats. Rats prefer choklates, although several foods were available to them in the cage. It seems that food is an intoxication similar to alcohol, which we eat at the inspiration of our brain. Our brain allows us to eat only those foods, whose taste is already known to the brain.

Intervention Trials

Clinical trials, using the Columbus food approach so far produced are also in line with above experimental and epidemiological evidence. In the Lyon Diet Heart Study [6], 605 patients who had a myocardial infarction were randomly assigned to a ‘Mediterranean-style’ diet or a control diet resembling the American Heart Association Step I diet. The Mediterranean diet model supplied 30% of energy

from fats and < 10% of energy from saturated fatty acids, whereas the intake of 18:3 (n-3) (α-linolenic acid) provided >0.6% of energy. After a mean follow-up of 27 months, the risk of new acute myocardial infarction and episodes of unstable angina was reduced by ~ 70% by the Mediterranean diet, Moreover, total mortality was also reduced by 70%.Singh et al(7) tested an 'Indo-Mediterranean diet' in 1000 patients in India, with existing coronary disease or at high risk for coronary disease. When compared with the control diet, the intervention diet characterized by increased intake of mustard or soyabean oil, nuts(walnuts, almonds), vegetables, fruits and whole grains-reduced the rate of fatal myocardial infarction by one-third and the rate of sudden death from cardiac causes by two-thirds.

It is therefore suggested that a western foods that are rapidly absorbed, should be avoided in patients with acute coronary syndromes(ACS),and administer diet which is beneficial to vascular endothelium and myocardium. There is limited evidence regarding the role of dietary intervention in patients with AMI [3-7,9-13].The aim of the Indian experiment was to determine the effects of a diet rich in w-3 fatty acids, vitamins, minerals and antioxidants (fruits, vegetables, legumes, walnuts, almonds, fish, mustered and soyabean oils)and low in refined carbohydrates, in patients with (AMI, tables 1-3)).All patients with a diagnosis of ACS were assigned to an intervention diet(n=204) or a control diet(n=202)within 48hours of the onset of the symptoms of AMI[9-12].The intervention group was advised to consume 600g/day of fruits, vegetables, legumes and almonds and walnuts, in a soup or semisolid form. Tomato soup, skim milk and curd(yogurt)were commonly used to mix crushed almonds and walnuts and other foods, which were grilled with mustered oil. The control group was advised a low fat diet consistent with National Cholesterol Education Program. (Tables 1,2).Clinical characteristics, time elapsed from symptom onset to the index infarction, site of infarction, drug therapy and final diagnosis were comparable between the two groups. Intake of foods and selected nutrients was assessed during the 1 week and after 1 year. After 1 week, plasma lipid peroxides, vitamin C and lactate dehydrogenase levels were determined. Compared with the control groups, patients allocated to the dietary intervention consumed significantly greater amounts of fruit, vegetables, pulses, almonds, walnuts, oils and fish, both during the first trial week and 1 year after AMI [12]. The consumption of n-3 fatty acids was also significantly greater in the intervention group than in the control group(1.8±0.66 versus 0.65±0.4g day^{-1}P<0.01).The consumption of proinflammatory foods, such as butter and clarified butter, refined starches and sugar were significantly greater in the control group than in the intervention group. Plasma lipid peroxide level decreased significantly in the intervention group compared with the control group, indicating a decrease in oxidative stress which is protective against proinflammatory IL-6 and TNF-alpha as well as endothelial disfunction, although these data were not measured in our study.Lactate dehydrogenase (LDH) level increased less in the intervention group than in the control group, indicating that myocardial damage was prevented by the cardioprotective diet. The increased intake of n-3 fatty acids from mustard and soy bean oil associated with the Mediterranean diet might be responsible for the significant reduction in the cardiac enzyme LDH and lipid peroxides in the intervention compared with the control group. Total cardiac events, including fatal and non-fatal myocardial infarctions and sudden cardiac deaths, were significantly lower in the intervention group compared with the control group, both after 6 weeks [10] as well as after one year [11].

The effects of 1 year of treatment with fish oil (122 patients, eicosapentaenoic acid, EPA1.08g day^{-1}) mustard oil (120 patients), alpha-linolenic acid 2.9g day^{-1}), and no treatment (118 patients, placebo group) on the outcome of patients with suspected AMI were compared in a randomized, placebo-controlled trial)[12].Treatment were administratered within, on average 18h of onset of symptoms. Clinical characteristics, extent of cardiac damage and rise of cardiac enzymes and lipid peroxides were comparable among the three groups at study entry. After randomization, angina pectoris (18.0 and 21.6 versus 42.3%), arrhythmias (13.1and 13.3 versus 28.7%) and poor left ventricular function (22.8 and 26.6, versus 47.4%) were significantly lower in the fish oil and mustard oil treatment groups compared with the placebo group.

Table 1. Effect of w-3 fatty acid rich diet in patients with acute myocardial infarction [11,19](tubers not included)

	Intervention group (n=204)		Control group (n=202)	
Foods and nutrients	**4-7 days**	**After 1 years**	**4-7 days**	**After 1 years**
Fruits and vegetables (g. day-1)	**508.4±28.66****	**575±91.4****	**254.4±17.2**	**220.5±19.6**
Legumes and pulses (g. day-1)	**80.5±6.6****	**95.0±8.9****	**52.5±4.6**	**45.6±5.6**
Almonds and walnuts (g. day-1)	**82.4±5.7****	**75.5±5.2****	**-**	**-**
Fish(g. day-1)	**52.5±6.5****	**22.4±4.1****	**20.2±3.1**	**10.5±3.5**
Chicken(g. day-1)	**-**	**10.2±3.2***	**76.2±6.5**	**66.5±10.5**
Mustard or soybean oil	**18.4±3.9***	**31.5±5.5****	**10.5±2.3**	**6.8±2.8**
Butter or clarified butter(g. day-1)	**2.5±0.6****	**3.3±0.71***	**10.5±2.6**	**12.6±3.5**
Skim milk (ml day-1)	**161.2±12.0**	**152±14.5***	**150.2±8.0**	**165.5±16.1**
Wheat chapati	**5.5±1.6****	**30.6±5.5**	**50.6±6.6**	**55.6±7.8**
Bread,biscuits (g. day-1)	**10.6±2.2***	**25.5±6.2****	**230.6±20.1**	**212.2±18.1**
Rice and wheat cereals(g. day-1)	**25.6±2.4**	**30.6±5.5**	**30.2±3.1**	**35.6±4.8**
Honey or raisins(g. day-1)	**2.6±0.8**	**5.5±1.2**	**-**	**-**
Sugar(g. day-1)	**16.4±3.7***	**12.6±3.4***	**25.5±5.4**	**30.5±7.6**
Alpha-linolenic acid(g. day-1)	**1.0±0.46****	**1.8±0.66****	**0.5±0.11**	**0.65±0.14**
Ascorbic acid (mg. day-1)	**474±70.5****	**440.6±100.6****	**150.2±10.6**	**160±38.0**

P values were obtained by comparison of intervention and control groups after 1 week and after 1 year.*=P<0.05,**P<0.01.

Table 2. Effect of dietary intervention on oxidative stress,vitamins and cardiac damage in relation to dietary alpha-linolenic acid [9,19]

	Intervention group (n=204)		Control group (n=202)	
	Baseline, 3rd day	**7th day**	**Baseline, 3rd day**	**7th day**
Dietary alpha-linolenic acid(g/day)	**1.0±0.46**	**1.8±0.66***	**0.5± 0.11**	**0.65±0.4**
Plasma ascorbic acids (23.2±3.2, μmol.1^{-1})	**7.38±1.7**	**30.6±4.7****	**7.9±1.7**	**15.8±3.8**
Plasma lipid peroxides(1.4±0.41,pmol.1^{-1})	**3.56±0.71**	**2.9±0.62***	**3.48±0.7**	**3.38±0.62**
Lactate dehydrogenase (216.5±36.5,IU.1^{-1})	**228.8±48.6**	**656.5±75.6***	**224.2±45.4**	**785.4±81.2**

P value was obtained by comparison of intervention and control groups on the seventh day after onset of symptoms of myocardial infarction.Values are mean+SD, *=P<0.05, **=P<0.01.

Table 3. Effect of cardioprotective diet on cardiac events after one year [11,19]

	Intervention group (n=204)	Control group (n=202)
Non-fatal myocardial infarction	**30(15)****	**48(24)**
Fatal myocardial infarction	**13(6)***	**19(9)**
Sudden cardiac death(<1h)	**7(3)***	**15(7)**
Suspected cardiac death	**-**	**1(0.5)**
Death due to stroke	**1(0.5)**	**2(1.0)**
Death due to cancer	**-**	**1(0.5)**
Total cardiac mortality	**20(10)****	**34(17)**
Total mortality	**21(10)****	**38(19)**
Total cardiac events and deaths	**51(25)****	**86(43)**

P value was obtained by comparison of intervention and control group by a Z test for proportions.P<0.05,**=P<0.01.

Sudden cardiac deaths (1.6 and 1.6 versus 6.6%), total cardiac deaths(11.4 and 13.3 versus 22.0%) non-fatal infarctions (13.0and 15.0, versus 25.4%), and total cardiac events 24.5 and 28.2 versus 47.4%) were also significantly lower in the two intervention groups. A modest improvement in dyslipidaemia and a decrease in oxidative damage were observed in the fish oil and mustard oil groups but not in the placebo group. On the third and the fifth day after AMI, serum glutamic oxalo-transaminase (SGOT) and LDH cardiac enzymes showed greater decline in the fish oil and mustard oil groups compared with the placebo group. In a recent study [14], a total of 117 volunteers completed the 12-week trial. Participants in the 1-, 3-, and 6-portions/d groups reported consuming on average 1.1, 3.2, and 5.6 portions of fruit and vegetables, respectively, and serum concentrations of lutein and β-cryptoxanthin increased across the groups in a dose-dependent manner. For each 1-portion increase in reported fruit and vegetable consumption, there was a 6.2% improvement in forearm blood flow responses to intra-arterial administration of the endothelium-dependent vasodilator acetylcholine (P=0.03). There was no association between increased fruit and vegetable consumption and vasodilator responses to sodium nitroprusside, an endothelium-independent vasodilator. Further clinical and epidemiological studies (15-20) indicate a significant association of w-3 fatty acid status and risk of ventricular premature beats, AMI and inflammation. Evolutionary aspects of diet also indicate that w-3 fat was quite rich during human evolution [21]. Intervention trials with w-3 rich eggs found no adverse effects on the risk factors of CAD [22-24].

Further trials with fruits, vegetables and whole grains have been found to reduce the risk of CAD [25-29]. The nurses health study showed that regular consumption of sugar sweetened beverages was associated with a higher risk of CAD in women, even after other unhealthful lifestyle or dietary factors are accounted for [30].

These intervention trials [6-8,27-29] indicate that further studies should be conducted with Columbus diet and Columbus oil(olive oil+lin seed oil)in patients with AMI, to demonstrate, cytokine and endothelial function mediated mechanisms, in the pathophysiology of complications and deaths,among these patients.Similar dietary intakes are being advised in the Columbus paradigm(www.Columbus-concept.com).

We do not have any guideline from any other health agency, for dietary advice, in patients with AMI, which may be protective against recurrent cardiac events [13]. A Columbus soup(tomatoes, grapes, vegetables, walnuts, almonds+ lin seed and olive oil) or a columbus yogurt containing, walnuts, almonds, black raisins, 20g each, crushed with a mixer could be prepared for ready use, for nonpharmacological intervention, among patients of AMI [13].Such recepies have been commonly used by us in our studies and clinical practice [7,9-13].We need to plan more studies with diet rich in w-3 fatty acids, coenzyme Q10,soluble fibre and other phytochemicals, to provide proof for the role of diet in AMI as diet can modulate cardiac dysfunctions, during acute coronary syndromes.After recovery from acute phase, eating 400g/day of whole grains including legumes and another 400g/day of fruits, vegetables and nuts(almonds and walnuts,50g/day) inconjunction with 30-50g/day of canola oil or Columbus oil(olive oil+lin seed oil)may be protective against endothelial dysfunction in patients after AMI.

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