

sci.med.nutrition: Re: What exercise, vitamins, or food needed for diabetes/cardio risk individual?

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From: Michael C Price (michaelEXCISESPAMprice_at_ntlworld.com)

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Nico Kadel-Garcia writes of my suggestion:

>> *In addition to chromium, try vanadium, niacin, biotin, magnesium*
>> *B1, B6. The other B-vitamins would probably help as well.*
>
> *He may as well eat a bumper as go this route, it will help about as*
> *much and probably be cheaper.*

Cheaper, perhaps, but not as beneficial.

> *There are a lot of vitamin salesfolks in the world who will help you*
> *spend your money.*

Perhaps there are, but I'm not in sales. Just a happy consumer.

> *Talk with your local doctor or dietician about*
> *changes in diet, and get a glucose tolerance test if you think you might*
> *have diabetes. In particular, untreated Type 2 diabetes can cause you*
> *to *gaini* weight.*

Nico,
there's plenty of evidence to support the items I listed.
The fact that people like you and others are so quick
to denounce anything outside your limited knowledge
is a scandal — but very predictable. Check out the
numbered and annotated references in the text below
for a more scientific and informed opinion.

Glycation is the non-enzymic binding of glucose molecules to other molecules
in general. This binding is uncontrolled and destructive, causing the
faster aging exhibited by diabetics, and, since as we age we all tend to
become pre-diabetic^{55, 83i-j} (with reduced glucose tolerance, rising insulin
and increased insulin-resistance and glucose levels), it is implicated in
normal aging⁸³.

Animals fed chromium had lower fasting glucose and insulin levels and

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improved glucose tolerance. A range of studies on pigs, dogs and rodents suggest that glucose processing is optimised when the chromium intake in micrograms (ug) is above 1/5 of the number of calories consumed^{5d}. In humans, chromium benefits both the healthy non-diabetics⁵⁵ and diabetics, types I10d & II10, by improving HDL/LDL profiles, lowering insulin levels, fasting glucose levels and improving glucose tolerance. Both vitamins B3 (niacin)^{55a}, B7 (biotin)^{55d} and the mineral zinc¹³⁷ synergise with chromium to improve insulin-resistance.

Part of the reason why chromium deficiency is so common is that plants do not require chromium; plants can thrive in chromium poor soils, leading to chromium poor diets. Chromium optimises insulin function, which, in turn, lowers fasting glucose levels, improves glucose tolerance and reduces glycation. Chromium's anti-glycation, glucose-regulatory action is mediated entirely via the protein insulin (a hormone); chromium, therefore, is a proteonomic cofactor, but not an enzymic cofactor, with a single target protein, unlike the enzymic cofactors (which are proteonomic cofactors, since all enzymes are proteins), which operate via multiple enzymes. Therefore we have to be cautious in extrapolating the chromium rodent lifespan extension of 27% to humans; the control rats tested showed evidence of pre-diabetic, sub-clinical insulin resistance – their glycated haemoglobin levels increased almost 4-fold more as they aged^{5a}, ^{5c} (as a proportion of their lifespan) than in non-diabetic humans⁸³ⁱ; the anti-aging effect on non-diabetic humans will probably be only a quarter as much, about 7%.

Independently of any synergy with chromium, vitamins B1 (thiamine)^{88c-e}, B3 (niacin)⁸⁵, B6 (pyridoxine)^{88a-e}, and B7 (biotin)⁹³ along with minerals magnesium¹²⁸, zinc⁹¹, in doses greater than the RDA, improve insulin-resistance and lower glycation levels.

Calorie restriction (see later) also lowers glycation and extends lifespan, but whether it will synergise with the above micronutrients is unknown.

Summary: chromium⁵, ¹⁰, ⁵⁵, magnesium¹²⁸ and zinc⁹¹, ¹³⁷, along with vitamins B1 (thiamine)^{88c-e}, B3 (niacin)^{55a}, ⁸⁵, B6 (pyridoxine)^{88a-e} and B7 (biotin)^{55d}, ⁹³, are effective in preventing or ameliorating diabetes and, even in non-diabetics, reducing glycation levels, insulin-resistance and slowing aging.

[5a] Composition and Biological Activity of Chromium-Pyridine Carboxylate Complexes. GW Evans and DJ Pouchnik, *Journal of Inorganic Biochemistry* 49, pg 177-187 (1993). PMID: 8433089

Describes the action of dietary chromium picolinate (relative to chromium chloride and chromium nicotinate) in reducing glycation & plasma glucose levels in rats as they aged.

[5b] Longevity effect of chromium picolinate—'rejuvenation' of hypothalamic function? McCarty MF in *Med Hypotheses* 1994 Oct;43(4):253-65 PMID: 7838011

"The first rodent longevity study with the insulin-sensitizing nutrient

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chromium picolinate has reported a dramatic increase in both median and maximal lifespan.." Gives additional information about the Evans–Meyer–Pouchnik chromium picolinate experiment on rats: Cohort maximum lifespan (last survivor) was 48 months, extending the previous species maximum by 15% to give a total maximum lifespan increase of 26%.

[5c] Chromium picolinate increases longevity. Evans GW, Meyer LK in AGE (the Journal of the American Aging Association) Oct 1992; 15(4), 134.

[5d] Chromium Picolinate. Gary W Evans, (1996) ISBN 0895299119. Gives additional information about the Evans–Meyer–Pouchnik chromium picolinate experiment on rats: Mean lifespan extension of 27% However, the strain of rats used may have been pre–diabetic.

[5e] The Longevity Factor: Chromium Picolinate. RA Passwater, (1993), ISBN 0879836199.

[10a] Elevated intakes of supplemental chromium improve glucose and insulin variables in individuals with type 2 diabetes. Anderson RA, Cheng N, Bryden NA, Polansky MM, Cheng N, Chi J, Feng J in Diabetes 1997 Nov;46(11):1786–91 PMID: 9356027

[10b] Nutritional factors influencing the glucose/insulin system: chromium. Anderson RA in J Am Coll Nutr 1997 Oct;16(5):404–10 PMID: 9322187

[10c] Beneficial effects of chromium in people with type 2 diabetes, and urinary chromium response to glucose load as a possible indicator of status. Bahijri SM, Mufti AM in Biol Trace Elem Res 2002 Feb;85(2):97–109 PMID: 11899964

[10d] [Chromium in the treatment of clinical diabetes mellitus] Ravina A, Slezack L in Harefuah 1993 Sep;125(5–6):142–5, 191 PMID: 8225092
"We gave 243 diabetic patients Cr (200 mcg/d) to study its effect on blood glucose balance. 105 were Type 1 (IDDM) and 138 Type 2 (NIDDM). Cr reduced insulin, sulfonylurea or metformin requirements in 115 patients. The success rate was greater in those with NIDDM (57.2%) than in those with IDDM (33.6%). More women, of either type, reacted than men (62.5 vs 50% in NIDDM and 37.6 vs 28.6% in IDDM). A placebo was ineffective."

[55a] Evidence for synergism between chromium and nicotinic acid in the control of glucose tolerance in elderly humans. Urberg M, Zemel MB in Metabolism 1987 Sep;36(9):896–9 PMID: 3626867
"Sixteen healthy elderly volunteers were divided into three groups and given either 200 micrograms Cr, 100 mg nicotinic acid, or 200 micrograms Cr + 100 mg nicotinic acid daily for 28 days and evaluated on days 0 and 28. Fasting glucose and glucose tolerance were unaffected by either chromium or nicotinic acid alone. In contrast, the combined chromium–nicotinic acid supplement caused a 15% decrease in a glucose area integrated total (p less than .025) and a 7% decrease in fasting glucose."

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[55b] Effect of chromium supplementation on glucose tolerance and lipid profile. Bahijri SM in *Saudi Med J* 2000 Jan;21(1):45–50 PMID: 11533750
"Improved glucose control, and lipid profile following chromium supplement suggests the presence of low chromium status in the studied population. However, serum chromium could not be recommended for use as an indicator of chromium status as subjects with widely varying levels responded favorably to the chromium supplement."

[55c] The safety and efficacy of high-dose chromium. Lamson DS, Plaza SM in *Altern Med Rev* 2002 Jun;7(3):218–35 PMID: 12126463
"The beneficial effects of chromium on serum glucose and lipids and insulin resistance occur even in the healthy."

[55d] High-dose biotin, an inducer of glucokinase expression, may synergize with chromium picolinate to enable a definitive nutritional therapy for type II diabetes. McCarty MF in *Med Hypotheses* 1999 May;52(5):401–6 PMID: 10416947

[55e] Beneficial effect of chromium-rich yeast on glucose tolerance and blood lipids in elderly subjects. Offenbacher EG, Pi-Sunyer FX in *Diabetes* 1980 Nov;29(11):919–25 PMID: 7000589
"Thus, chromium-rich brewers' yeast improved glucose tolerance and total lipids in elderly subjects, while chromium-poor torula yeast did not. An improvement in insulin sensitivity also occurred with brewers' yeast supplementation. This supports the thesis that elderly people may have a low level of chromium and that an effective source for chromium repletion, such as brewers' yeast, may improve their carbohydrate tolerance and total lipids."

[55f] Effects of chromium supplementation on fasting insulin levels and lipid parameters in healthy, non-obese young subjects. Wilson BE, Gandy A in *Diabetes Res Clin Pract* 1995 Jun;28(3):179–84 PMID: 8529496
"However, those individuals [6/15] within the chromium group with initial fasting IRI levels greater than 35 pmol/l had a significant decrease in IRI level after supplementation ($P < 0.03$) despite no significant changes in serum lipids. These subjects may benefit from chromium supplementation by improving insulin sensitivity and cardiovascular risk over time."

[85] Safety of high-dose nicotinamide: a review. Knip M, Douek IF, Moore WP, Gillmor HA, McLean AE, Bingley PJ, Gale EA in *Diabetologia* 2000 Nov;43(11):1337–45 PMID: 11126400
Discusses the potential of 3g/d vitamin B3 in preventing the onset of Type I (insulin-dependent) diabetes.

[88a] Amadorins: novel post-Amadori inhibitors of advanced glycation reactions. Khalifah RG, Baynes JW, Hudson BG in *Biochem Biophys Res Commun* 1999 Apr 13;257(2):251–8 PMID: 10198198
"This in turn led to a unique and rapid post-Amadori screening assay for putative "Amadorins," which we define here as inhibitors of the conversion of Amadori intermediates to AGEs in the absence of excess free or reversibly bound (Schiff base) sugar. Our screening assay then led to the

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identification of pyridoxamine (Pyridorin) as the first member of this class of Amadorin compounds. Rather unexpectedly, the assay also led to the clear demonstration that the well-known AGE inhibitor aminoguanidine, currently in Phase 3 clinical trials for treatment of diabetic nephropathy, has negligible Amadorin activity. In view of the importance of Amadori compounds as intermediates in AGE formation in vivo, the therapeutic potential of Pyridorin is currently being investigated and is now showing highly promising results in different animal models."

[88b] Glycooxidation and lipoxidation in atherogenesis. Baynes JW, Thorpe SR in *Free Radic Biol Med* 2000 Jun 15;28(12):1708–16 PMID: 10946212
"advanced lipoxidation end-products (ALEs). The ALEs and their precursors affect the structure and function of the vascular wall, setting the stage for atherogenesis. The increased risk for atherosclerosis in diabetes may result from additional carbonyl production from carbohydrates and additional chemical modification of proteins by advanced glycation end-products (AGEs). Failure to maintain homeostasis and the increase in oxidizable substrate (lipid) alone, rather than oxidative stress, is the likely source of the increase in reactive carbonyl precursors and the resultant ALEs and AGEs in atherosclerosis. Nucleophilic AGE-inhibitors, such as aminoguanidine and pyridoxamine, which trap reactive carbonyls and inhibit the formation of AGEs in diabetes, also trap bioactive lipids and precursors of ALEs in atherosclerosis. These drugs should be effective in retarding the development of atherosclerosis, even in nondiabetic patients."

[88c] [Diabetes and vitamin levels] Tamai H in *Nippon Rinsho* 1999 Oct;57(10):2362–5 PMID: 10540887
"Administration of vitamins to diabetic patients reduces insulin requirement and attracts much attention for improvement of vascular complications. Vitamins play as not only nutritional supplements for deficiency, but pharmacological agents for treatment."

[88d] In vitro kinetic studies of formation of antigenic advanced glycation end products (AGEs). Novel inhibition of post-Amadori glycation pathways. Booth AA, Khalifah RG, Todd P, Hudson BG in *J Biol Chem* 1997 Feb 28;272(9):5430–7
PMID: 9038143
"Of several derivatives of vitamins B1 and B6 recently studied for possible AGE inhibition in the presence of glucose (Booth, A. A., Khalifah, R. G., and Hudson, B. G. (1996) *Biochem. Biophys. Res. Commun.* 220, 113–119), pyridoxamine and, to a lesser extent, thiamine pyrophosphate proved to be novel and effective post-Amadori inhibitors that decrease the final levels of AGEs formed."

[88e] Thiamine pyrophosphate and pyridoxamine inhibit the formation of antigenic advanced glycation end-products: comparison with aminoguanidine. Booth AA, Khalifah RG, Hudson BG in *Biochem Biophys Res Commun* 1996 Mar 7;220(1):113–9 PMID: 8602828
"Among the inhibitors, pyridoxamine and thiamine pyrophosphate potently inhibited AGE formation and were more effective than aminoguanidine, suggesting that these two compounds may have novel therapeutic potential in

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preventing vascular complications of diabetes."

[93a] Urinary biotin analogs increase in humans during chronic supplementation: the analogs are biotin metabolites. Mock DM, Heird GM in *Am J Physiol* 1997 Jan;272(1 Pt 1):E83–5 PMID: 9038855

[93b] Biotin supplementation improves glucose and insulin tolerances in genetically diabetic KK mice. Reddi A, DeAngelis B, Frank O, Lasker N, Baker H *Life Sci* 1988;42(13):1323–30 PMID: 3280936

[93c] Oral glucose tolerance test after high-dose i.v. biotin administration in normoglycemic hemodialysis patients. Koutsikos D, Fourtounas C, Kapetanaki A, Agroyannis B, Tzanatos H, Rammos G, Kopelias I, Bosiolis B, Bovoleti O, Darema M, Sallum G in *Ren Fail* 1996 Jan;18(1):131–7 PMID: 8820510

[93d] [Enhancement of glucose-induced insulin secretion and modification of glucose metabolism by biotin] Furukawa Y in *Nippon Rinsho* 1999 Oct;57(10):2261–9 PMID: 10540872

[93e] Biotin administration improves the impaired glucose tolerance of streptozotocin-induced diabetic Wistar rats. Zhang H, Osada K, Sone H, Furukawa Y in *J Nutr Sci Vitaminol (Tokyo)* 1997 Jun;43(3):271–80 PMID: 9268917

[93f] A high biotin diet improves the impaired glucose tolerance of long-term spontaneously hyperglycemic rats with non-insulin-dependent diabetes mellitus. Zhang H, Osada K, Maebashi M, Ito M, Komai M, Furukawa Y in *J Nutr Sci Vitaminol (Tokyo)* 1996 Dec;42(6):517–26 PMID: 9089478

[128a] Magnesium supplementation reduces development of diabetes in a rat model of spontaneous NIDDM. Balon TW, Gu JL, Tokuyama Y, Jasman AP, Nadler JL in *Am J Physiol* 1995 Oct;269(4 Pt 1):E745–52 PMID: 7485490

"We examined the effects of a magnesium-supplemented (Mg-S) diet in the male obese Zucker diabetic fatty rat, a model of non-insulin-dependent diabetes mellitus (NIDDM). Obese rats were maintained on either a control (0.20% Mg) or magnesium-supplemented (Mg-S; 1% Mg) diet for 6 wk beginning at 6 wk of age. The rats maintained on the Mg-S diet had markedly lower fasting and fed-state blood glucose concentrations and an improved glucose disposal. By 12 wk of age, all of the eight animals on the control diet became diabetic, whereas diabetes developed in only one of eight animals on the Mg-S diet. Insulin and C-peptide concentrations, in addition to pancreatic GLUT-2 and insulin mRNA expression, were higher in the male obese Mg-S rats than in their control-fed counterparts. A subgroup of rats on the control diet with established diabetes was switched to a Mg-S diet for an additional 4 wk. The Mg-S diet did not reverse diabetes once already established. These data indicate that an increased dietary Mg intake in male obese rats prevents deterioration of glucose tolerance, thus delaying the development of spontaneous NIDDM."

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[128b] Synergistic interaction of magnesium and vanadate on glucose metabolism in diabetic rats. Matsuda M, Mandarino L, DeFronzo RA in *Metabolism* 1999 Jun;48(6):725–31 PMID: 10381146

"Based on these results, MgV is superior to either V alone or Mg alone in improving insulin sensitivity and glycogen synthesis in diabetic rats."

[128c] The effect of magnesium supplementation in increasing doses on the control of type 2 diabetes. Lima Mde L, Cruz T, Pousada JC, Rodrigues LE, Barbosa K, Cangucu V in *Diabetes Care* 1998 May;21(5):682–6 PMID: 9589224

"In the placebo and in the 20.7 mmol [0.5g] Mg groups, neither a change in plasma and intracellular levels nor an improvement in glycemic control were observed. Replacement with 41.4 mmol [1g] Mg tended to increase plasma, cellular, and urine Mg and caused a significant fall (4.1 +/- 0.8 to 3.8 +/- 0.7 mmol/l) in fructosamine (normal, 1.87–2.87 mmol/l). CONCLUSIONS: Mg depletion is common in poorly controlled patients with type 2 diabetes, especially in those with neuropathy or coronary disease. More prolonged use of Mg in doses that are higher than usual is needed to establish its routine or selective administration in patients with type 2 diabetes to improve control or prevent chronic complications."

[128d] Improved insulin response and action by chronic magnesium administration in aged NIDDM subjects. Paolisso G, Sgambato S, Pizza G, Passariello N, Varricchio M, D'Onofrio F in *Diabetes Care* 1989 Apr;12(4):265–9 PMID: 2651054

"In eight aged non–insulin–dependent diabetes mellitus (NIDDM) subjects, insulin response and action were studied before and after chronic magnesium supplementation (2 g/day) to diet. [.] In conclusion, our data suggest that NIDDM subjects may benefit from therapeutic chronic administration of magnesium salts."

[128e] Daily magnesium supplements improve glucose handling in elderly subjects. Paolisso G, Sgambato S, Gambardella A, Pizza G, Tesauro P, Varricchio M, D'Onofrio F in *Am J Clin Nutr* 1992 Jun;55(6):1161–7 PMID: 1595589

"aged subjects were enrolled in a double–blind, randomized, crossover study in which placebo (for 4 wk) and chronic magnesium administration (CMA) (4.5 g/d for 4 wk) were provided. [.] CMA vs placebo significantly increased erythrocyte magnesium concentration and improved insulin response and action. Net increase in erythrocyte magnesium significantly and positively correlated with the decrease in erythrocyte membrane microviscosity and with the net increase in both insulin secretion and action. In aged patients, correction of a low erythrocyte magnesium concentration may allow an improvement of glucose handling."

[128f] Magnesium Intake and Risk of Type 2 Diabetes in Men and Women. Lopez–Ridaura R, Willett WC, Rimm EB, Liu S, Stampfer MJ, Manson JE, Hu FB in *Diabetes Care*. 2004 Jan;27(1):134–140. PMID: 14693979

"Our findings suggest a significant inverse association between magnesium intake and diabetes risk."

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[137] Potential antioxidant effects of zinc and chromium supplementation in people with type 2 diabetes mellitus. Anderson RA, Roussel AM, Zouari N, Mahjoub S, Matheau JM, Kerkeni A in J Am Coll Nutr 2001 Jun;20(3):212-8
PMID: 11444416

"CONCLUSIONS: These data suggest the potential beneficial antioxidant effects of the individual and combined supplementation of Zn and Cr in people with type 2 DM. These results are particularly important in light of the deleterious consequences of oxidative stress in people with diabetes."

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Cheers,
Michael C Price

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