Beneficial Effect of Chromium-rich Yeast on Glucose Tolerance and Blood Lipids in Elderly Subjects

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SUMMARY

Twenty-four volunteers, mean age 78, including eight mildly non-insulin-dependent diabetics, were randomly allocated to one of two groups and were fed (daily for 8 wk) 9 g of either chromium-rich brewers' yeast (experimental) or chromium-poor torula yeast (control). Before and after yeast supplementation, the serum glucose and insulin response to 100 g oral glucose was measured at 30 min intervals for 2 h. Fasting serum cholesterol, total lipids, and triglycerides were also determined. In the total experimental group (normals + diabetics) and in both the diabetic and nondiabetic experimental subgroups, glucose tolerance improved significantly and insulin output decreased after supplementation. Cholesterol and total lipids fell significantly after supplementation in the total experimental group. The cholesterol decrease was particularly marked in hypercholesterolemic subjects (cholesterol > 300 mg/dl). In the control group, no significant change in glucose tolerance, insulin, triglycerides, or total lipids was found. Cholesterol was significantly lowered in the nondiabetic but not in the diabetic group.

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. The improvement in serum cholesterol in some control subjects, as well as in the total experimental group, also suggests the presence of a hypocholesterolemic factor other than chromium in both brewers' and torula yeast. DIABETES 29:919–925, November 1980.

Trivalent chromium, in a dinicotinato, glutathione-like complex,[1] is considered an essential element for insulin function and glucose disposal in mammalian nutrition. This biologically active molecule is known as glucose tolerance factor (GTF).[2] In vitro, insulin-sensitive tissues require higher insulin levels, in the absence of chromium, for equivalent glucose utilization.[3] In laboratory animals, chromium deficiency elevated serum glucose, insulin, and cholesterol and produced aortic plaques and corneal opacities.[4–6] When chromium-deficient animals were supplemented with inorganic chromium or GTF extracts, elevated glucose, insulin, and cholesterol levels were reduced.[4–7]

There is evidence to suggest that marginal dietary intake of chromium over decades in man can lead to a depletion of the body's chromium content.[8–10] In the U.S., where chromium intake is generally low,[10–14] many elderly people may be affected.[15] Such a chromium deficit was proposed as a contributing factor in some cases of maturity-onset diabetes and atherosclerotic disease.[3,10–15] Improved glucose tolerance and lower blood cholesterol occur in about 50% of elderly subjects who have been supplemented daily with 150 to 250 μg inorganic chromium.[16–18] A few studies were also done using brewers' yeast as a GTF-chromium supplement.[11,19,20] None of the human studies to date has included control or placebo groups, and only one quantified the amount of chromium given.[20]

The single-blind, controlled study described here was undertaken to measure the effect on glucose tolerance and on serum insulin and lipid levels of a chromium-rich brewers' yeast of known chromium content, fed for 8 wk to half of a monitored group of elderly subjects, while a chromium-poor torula yeast was fed to the other half.

SUBJECTS AND METHODS

A volunteer group of people, aged 63 yr and older, was recruited at a home for the retired elderly. Medical and dietary

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records were screened before subjects were accepted into the study. Among the criteria for exclusion were ketosis-prone insulin-dependent diabetes, marked obesity, hyperuricemia, major intestinal disease, and mental incompetence. An initial clinical and laboratory assessment included complete blood count and tests of liver and kidney function. Weights and heights were recorded. Informed consent was obtained.

An oral glucose load (Glucola, Ames Laboratories) was administered after an 8 h overnight fast. The daily carbohydrate intake of each subject for 3 days preceding the test was monitored to be at least 250 g. Subjects were given 100 g of glucose if they weighed more than 100 pounds or, if they weighed less, they were given 1.75 g per kilogram body weight. Blood was drawn before and at 30, 60, 90, and 120 min after the glucose load. Blood samples were collected in Vacutainer tubes, clotted at room temperature for 20 min, and then refrigerated, centrifuged, and the serum separated.

Serum glucose and insulin were measured on each of the five blood samples. Glucose was measured on the same day with a Beckman glucose analyzer utilizing an oxygen consumption method for glucose determination.[21] Serum cholesterol,[22] triglyceride,[23] and total lipid[24] levels were measured on fasting sera; the sera were then frozen at −20°C. Insulin determinations for all subjects were made at the conclusion of the study, when a second set of sera was obtained; this was done so that all insulin determinations for each individual could be performed in the same assay. The single antibody, charcoal absorption method of Herbert et al.[25] was used, utilizing a human insulin standard.

Subjects were classified as diabetic or nondiabetic by the criteria of Fajans[26] and, within these categories, were randomly assigned to experimental and/or control groups. All subjects in each group were fed 9 g of either chromium-rich brewers' yeast (experimental) or chromium-poor torula yeast (control) every day for 8 wk. Both yeasts contained 0.5 mg niacin per gram and were similar in other nutrient composition, except for their chromium contents. The brewers' yeast (Amber Laboratories, Wisconsin) had a chromium content of 1.2 μg per gram,* providing 10.8 μg chromium in the daily 9 g of yeast dose. The chromium content of the torula yeast (type B, Lake States Division, St. Regis Paper Co., Wisconsin) was <0.05 μg per gram,* providing <0.45 μg of chromium per day.

The yeast was administered in fruit juice. Batches of the beverages were prepared several times weekly, refrigerated or frozen, and served daily, under supervision, usually at the beginning of the lunch meal. The subjects did not know which yeast they received.

After the period of supplementation, the glucose tolerance tests were repeated in the same place, by the same personnel, and in the same manner as before. Twenty-four subjects, including eight nonketotic diabetics, satisfactorily completed the study. These subjects ranged in age from 63 to 93, mean age 78. The experimental and control groups each contained eight nondiabetic subjects (six women and two men) and four diabetic subjects (three women and one man). Distribution by decade was, 60–69: 4F, 1M; 70–79: 3F, 4M; 80–89: 8F, 1M; and 90+: 3F.

* These levels were confirmed by us in collaboration with Drs. W. Wolf, C. Veillon, and J. Kumpalainen at the Human Nutrition Center, U.S.D.A., Beltsville, Md.

A certain amount of chronic disease and use of medication is to be expected in such an elderly population. Diagnosed disease in the total group (single or concurrent) included hypertension (2), osteoarthritis (3), Paget's disease of bone (1), mild Parkinsonism (1), mild pulmonary insufficiency (1), and arteriosclerotic heart disease requiring some diuretic therapy (3). Chronic medication in the group (single or concurrent) included chlorothiazide and KCl (3), furosemide and KCl (2), digoxin (3), alphamethyldopa (1), Tofranil (1), and Valium (1). As only subjects with stable disease and medication patterns were allowed in the study, the medications listed were not changed during the course of the study. There was also no preponderance of disease or medication in one group over the other.

Mean serum glucose and insulin values were calculated for the blood samples drawn during the two glucose tolerance tests, before (B) and after (A) supplementation. Mean (B) and (A) values were also calculated for fasting serum cholesterol, triglyceride, and total lipid levels. The (B) and (A) means for each parameter were compared using Student's paired t tests. In addition, total area-under-the-curve means for glucose and insulin were calculated using the formula $\frac{A}{2} + B + C + D + \frac{E}{2}$ = Area Index Total (AIT), where A = fasting value, and B, C, D, and E = ½ h, 1 h, 1½ h, and 2 h values, respectively.[27] These AIT means were assessed for significant differences by paired t test analysis. Means were also calculated, and paired t tests, comparing values before and after supplementation, were made separately for all subjects, for nondiabetics, and for diabetics, for each parameter studied.

RESULTS

Glucose. Two-hour serum glucose responses to 100 g of oral glucose in each experimental (E) and control (C) subject before and after supplementation, expressed as area index totals (AIT), are shown in Figure 1. In 10 of the 12 E subjects, glucose AIT declined; it was unchanged in one subject and rose in the twelfth. In the 12 C subjects fed the chromium-poor yeast, glucose AIT declined in seven subjects (nominally in two) and increased in five.

Mean serum glucose and insulin responses to 100 g of oral glucose in the E and C groups, before and after supplementation, are presented in Table 1. After supplementation with the chromium-rich yeast, the mean AIT glucose of the 12 E subjects showed a decrement fro 632 mg·h/dl to 549 mg·h/dl, or −83 mg·h/dl, 13% ($P < 0.01$). Among the 12 C subjects fed the chromium-poor yeast, glucose AIT did not change significantly, the mean decline being from 552 mg·h/dl to 539 mg·h/dl, or 13 mg·h/dl (2%).

The baseline mean glucose and insulin AIT values of the E and C groups at the start of the study were similar. However, attrition of subjects (through noncooperation and transfers) brought the total number of volunteers who completed the 8 wk yeast supplementation period to 24, or two thirds of the intitial 36 selected. This led to somewhat higher baseline mean glucose AIT values in the E group than in the C group and somewhat higher mean insulin AIT values in the C group than in the E group, although, on t test comparison, there is no statistical difference between the groups. As can be seen in Figure 1, the glucose differences are due to the glucose AIT values of two subjects in the total E group (numbers 1 and 2), which were >2 SD above the mean of all

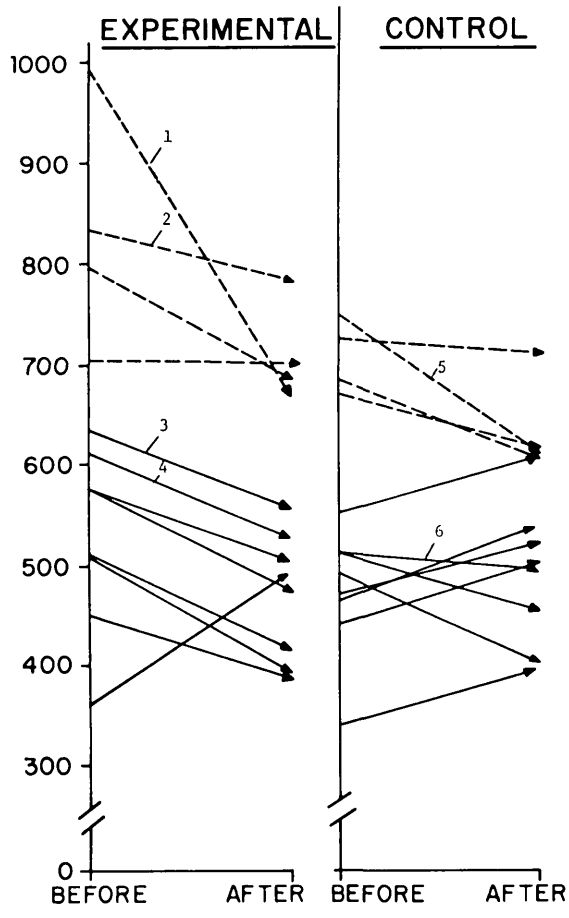

FIGURE 1. Glucose area index totals for individual subjects in response to 100 g oral glucose before and after supplementation in experimental subjects given chromium-rich yeast and in control subjects given chromium-poor yeast. ⇢ = diabetics, → = nondiabetics. N = 12 E and 12 C.

subjects. The insulin differences are due to the insulin AIT values of C subjects numbers 5 and 6, who had baseline insulin values >2 SD above the mean (as did E subject number 2). Therefore, the data are presented in Table 1 both with and without these four subjects. This was done to examine the possibility that the glucose tolerances of the C subjects failed to respond because they were initially lower and not amenable to improvement by factor(s) in yeast, while the poorer glucose tolerance of the E group may have favored a positive response to supplementation. As shown in Table 1, the mean glucose AIT decrement for the remaining 10 subjects in the E group, having a starting glucose AIT (571 mg·h/dl) similar to that of the controls (552 mg·h/dl), dropped to 513 mg·h/dl, or −58 mg·h/dl, 10% ($P < 0.02$). Their mean insulin AIT decrement, having a starting insulin AIT of 229 μU·h/dl compared with the C group's 223 μU·h/dl, dropped to 199 μU·h/dl, or −30 μU·h/dl (13%), but this was not significant.

Similarly, two subjects in the nondiabetic E group (numbers 3 and 4 in Figure 1) had baseline mean glucose AIT values >2 SD above the mean for all nondiabetic subjects, elevating the E group's mean, although not significantly. With subjects 3 and 4 included, the nondiabetic E group's mean glucose AIT declined from 527 mg·h/dl to 468 mg·h/dl, or −59 mg·h/dl, 11% ($P < 0.05$). Without these two cases, the mean glucose AIT declined from 495 ± 33 to 444 ± 21, or −51 mg·h/dl, 10%. In the nondiabetic C group, however, the glucose AIT rose slightly. The E group's mean insulin AIT without subjects 3 and 4 fell from 226 ± 49 μU·h/dl to 181 ± 41 μU·h/dl, or −45 μU·h/dl (20%), but this was not significant. The C group's mean insulin AIT did not change. Thus, whether comparing the total nondiabetic group, or a group more matched with the controls for glucose or insulin AIT, the E groups showed improvement of glucose tolerance, whereas this could not be found in the C group.

The glucose tolerance of both the E and C diabetic groups improved, but only in the diabetic E group was this improvement significant, with a glucose AIT decline of 131 mg·h/dl, 16% ($P < 0.05$). The diabetic C group's glucose AIT decline was 67 mg·h/dl, 10%, and was not significant.

Figure 2 presents the serum glucose tolerance values before and after supplementation. The significant decrements in serum glucose of the E group after supplementation occurred at 0 min ($P < 0.05$), 30 min ($P < 0.001$), and 60 min

TABLE 1
Serum glucose and insulin area index totals (AIT) in response to 100 g oral glucose, before (B) and after (A) supplementation, in experimental subjects given chromium-rich yeast and in control subjects given chromium-poor yeast. Results are expressed as $\bar{x}$ ± SEM. N = number of subjects. Significance is measured by paired *t* test, B/A.

Group	Experimental			Control		
	N	Glucose (mg·h/dl)	Insulin (μU·h/dl)	N	Glucose (mg·h/dl)	Insulin (μU·h/dl)
All Subjects	12			12		
Before		632 ± 53	272 ± 53		552 ± 37	308 ± 60
After		549 ± 38	225 ± 36		539 ± 27	283 ± 60
		$P < 0.01$				
All whose baseline glucose and insulin are < 2 SD above $\bar{x}$	10			10		
Before		571 ± 32			537 ± 26	
After		513 ± 35			536 ± 25	
		$P < 0.02$				
Nondiabetics	8			8		
Before		527 ± 32	222 ± 36		475 ± 24	284 ± 54
After		468 ± 22	186 ± 33		490 ± 25	274 ± 63
		$P < 0.05$				
Diabetics	4			4		
Before		843 ± 62	372 ± 140		705 ± 21	355 ± 157
After		712 ± 25	303 ± 81		638 ± 26	300 ± 109
		$P < 0.05$				

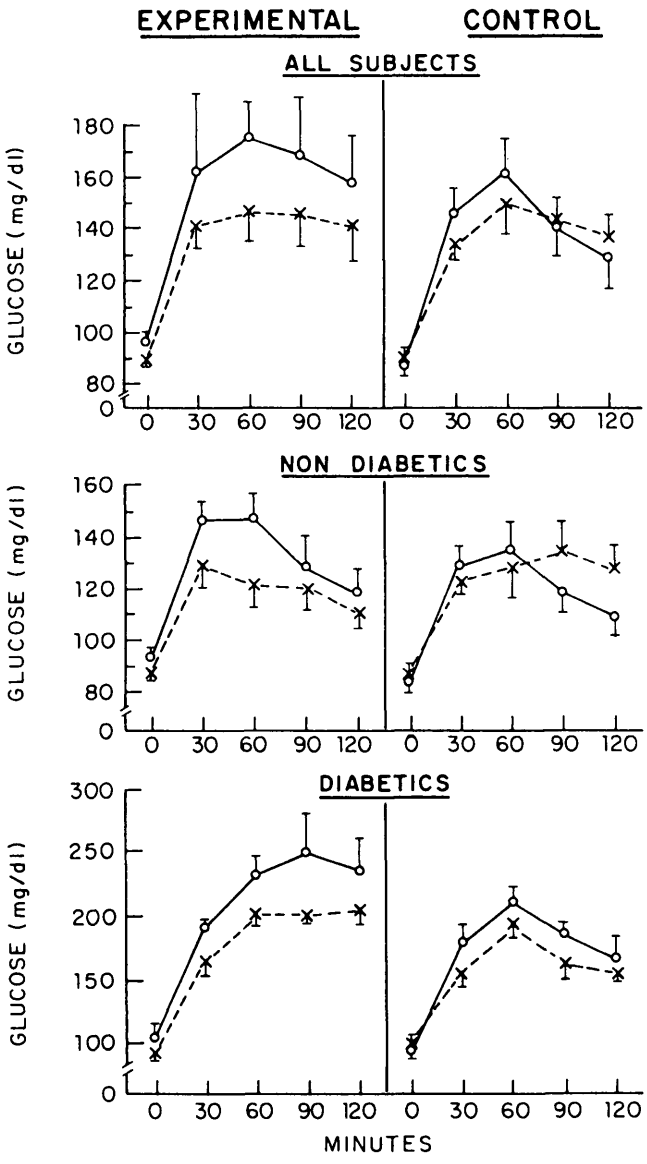

FIGURE 2. Serum glucose values in response to 100 g oral glucose before (O——O) and after (×-----×) supplementation in experimental subjects given chromium-rich yeast and in control subjects given chromium-poor yeast. Results are expressed as $\bar{x}$ ± SEM. Number of subjects for experimental and control groups is: all subjects, 12 and 12; nondiabetics, 8 and 8; diabetics, 4 and 4.

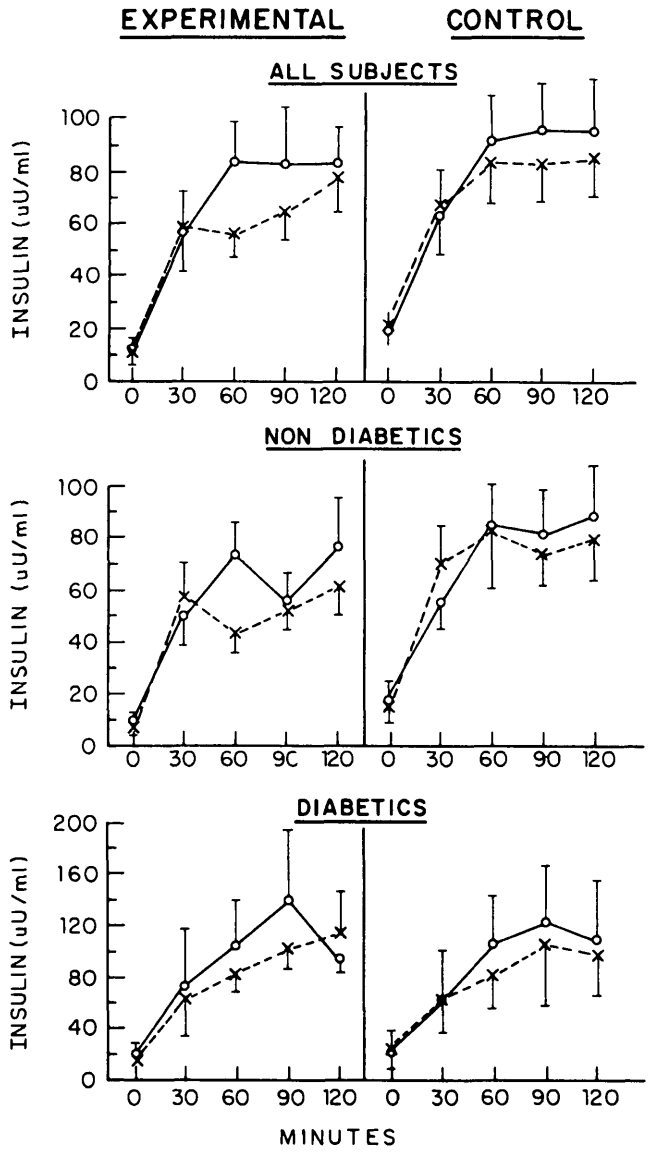

Figure 3. Serum insulin values in response to 100 g oral glucose before (O——O) and after (×-----×) supplementation in experimental subjects given chromium-rich yeast and in control subjects given chromium-poor yeast. Results are expressed $\bar{x}$ ± SEM. Number of subjects for experimental and control groups is: all subjects, 12 and 12; nondiabetics, 8 and 8; diabetics, 4 and 4.

($P < 0.001$). Both the diabetic E and the nondiabetic E groups followed this pattern. In the nondiabetic E group, the decrements were significant at 0 ($P < 0.05$), 30 ($P < 0.002$), and 60 ($P < 0.01$) min; in the diabetic E group, significance was found at 30 ($P < 0.02$) and 60 min ($P < 0.05$). The largest mean decrement in the diabetic E group was at 90 min ($P < 0.1$). While the diabetic C group's glucose values also declined somewhat after supplementation at each time interval, except 0 min, none differed significantly from those before supplementation.

Insulin. The insulin-secretory response to glucose of all subjects in the E group was reduced after supplementation, as seen in Table 1, notwithstanding the improved glucose disposal. The AIT declined 47 μU·h/dl, 17%. The insulin decrement of 28 μU·h/dl, 33%, at 60 min was significant ($P < 0.02$); this is shown in Figure 3. No C group subject's insulin value was significantly different from the presupplementation level.

The nondiabetic E group's insulin AIT improved, declining 36 μU·h/dl, 16%. This insulin decrement was significant at 60 min, with a drop of 31 μU·h/dl, 42% ($P < 0.02$). The nondiabetic C group's insulin AIT declined only 10 μU·h/dl, 4%, and its 60 min decrement was 4 μU·h/dl, 5%, as shown in Figure 2. No significant change in the insulin AIT of the diabetics in either the E or the C groups was found. As seen in Table 1, even though the mean insulin AIT fell 69 μU·h/dl, 19%, in the E group diabetics and −55 μU·h/dl, 15%, in the C group diabetics, this was not a significant change for either group.

Lipids. The lipid responses to yeast supplementation are presented in Table 2. For all subjects in the E group, a significant decline in cholesterol of 30 mg/dl, 12% ($P < 0.001$),

TABLE 2
Fasting serum lipid values, before (B) and after (A) supplementation, in experimental subjects given chromium-rich yeast and in control subjects given chromium-poor yeast. Results are expressed as $\bar{x} \pm$ SEM. N = number of subjects. Significance is measured by paired *t* test, B/A.

Group	Experimental				Control			
	N	Cholesterol (mg/dl)	Triglycerides (mg/dl)	Total lipids (g/dl)	N	Cholesterol (mg/dl)	Triglycerides (mg/dl)	Total lipids (g/dl)
All Subjects	12				12			
Before		257 ± 18	157 ± 19	0.71 ± 0.05		258 ± 12	135 ± 13	0.65 ± 0.03
After		227 ± 14	151 ± 16	0.58 ± 0.04		247 ± 14	140 ± 11	0.58 ± 0.03
		$P < 0.001$		$P < 0.05$				
Nondiabetics	8				8			
Before		252 ± 21	169 ± 23	0.71 ± 0.04		256 ± 13	136 ± 17	0.64 ± 0.03
After		231 ± 17	165 ± 21	0.57 ± 0.03		238 ± 14	137 ± 1	0.56 ± 0.04
		$P < 0.005$		$P < 0.02$		$P < 0.02$		
Diabetics	4				4			
Before		267 ± 37	132 ± 32	0.72 ± 0.10		261 ± 16	132 ± 22	0.66 ± 0.06
After		220 ± 20	122 ± 15	0.59 ± 0.09		267 ± 20	145 ± 28	0.62 ± 0.05
Cholesterol >300	4				1			
Before		335 ± 16	198 ± 38	0.96 ± 0.08		355	124	0.61
After		274 ± 21	159 ± 28	0.71 ± 0.09		339	126	0.61
		$P < 0.01$	$P < 0.05$	$P < 0.01$				
Cholesterol <300	8				11			
Before		218 ± 8	136 ± 18	0.59 ± 0.05		249 ± 9	136 ± 14	0.65 ± 0.03
After		204 ± 10	146 ± 20	0.51 ± 0.04		239 ± 11	141 ± 12	0.57 ± 0.03
		$P < 0.05$		$P < 0.05$				

was found. There was an insignificant decline in the C group's cholesterol of 11 mg/dl, 4%. For the nondiabetics, while the improvement was greater in the E group (−21 mg/dl, 8%) than in the C group (−18 mg/dl, 7%), there was a significant fall in both groups (E group, $P < 0.005$; and C group, $P < 0.02$).

In the E group, four subjects (two of whom were diabetic) had cholesterol levels >300 mg/dl. These hypercholesterolemic individuals showed significant improvement in their cholesterol values, since there was a mean decline of 61 mg/dl, 18% ($P < 0.01$). Only one C group subject was hypercholesterolemic, and her cholesterol declined 16 mg/dl, 5%. The response of subjects with cholesterol values <300 mg/dl in both the E and the C groups was more modest. The E group decrement was 14 mg/dl, 6%, and the C group decrement was 10 mg/dl, 4%. Neither was significant by paired *t* tests, comparing the cholesterol level before supplementation with that after supplementation. Comparison of the magnitude of change in cholesterol levels of the E and the C groups after supplementation showed a significant difference ($P < 0.02$), with E having the greater change.

Triglycerides. No significant change in serum triglycerides occurred in either the E or the C groups. Triglyceride levels did improve significantly, however, in the group of four E subjects who were hypercholesterolemic. The mean decrement for this group was 39 mg/dl, 20% ($P < 0.05$). The triglyceride level of the single hypercholestrolemic subject who received the chromium-poor yeast remained unchanged.

Total lipids. Total lipid changes paralleled those of cholesterol. The E group total mean lipid fell 0.13 g/dl, 18% ($P < 0.05$), while the C group total mean fell 0.07 g/dl, 11%. As with cholesterol and triglycerides, the greatest effect in total lipid response was noted among the hypercholesterolemic E group subjects. Their lipid values declined an average of 0.25 g/dl, 26% ($P < 0.01$). The single hypercholesterolemic C subject's total lipids remained unchanged.

DISCUSSION

The purpose of this study was to evaluate whether a chromium-rich supplement would be more effective than a chromium-poor supplement in improving insulin sensitivity and, consequently, glucose tolerance in a population thought to be chromium deficient.[11,16,17] In addition, the effect of this chromium-rich supplement on serum cholesterol, triglycerides, and total lipids was investigated. The results of the present study, utilizing a control and an experimental group in a single-blind study design, lend support to the hypothesis that chromium is important for optimal glucose tolerance in older Americans. Taking our putative chromium-deficient population, we found that both the diabetic and the nondiabetic experimental groups responded to the 8 wk of chromium-rich yeast supplementation with significantly improved glucose tolerance. This finding, and the finding of no change in the control group fed the chromium-poor yeast, suggests a beneficial effect of the chromium-rich yeast on glucose disposal.

We suggest one or a combination of hypotheses to account for this effect: (1) The subjects were chromium deficient, and a chromium supplement alleviated this deficiency. (2) A factor in brewers' yeast (other than chromium) contributed to the improvement. (3) The amount of chromium fed had a pharmacologic, rather than physiologic, effect.

Since the initial mean glucose AIT values were lower in control than in experimental subjects (in the total group, 552 ± 32 compared with 632 ± 53), we thought it important to determine whether the difference in response to treatment was due to there being significantly more control subjects

having better initial glucose tolerance values not capable of further improvement by some factor in yeast. It is clear from Table 1 that, if the data are recalculated with the top two subjects in the experimental group being left out (patients 1 and 2 in Figure 1), the starting values in the experimental group for glucose AIT approximate quite closely those for the control group, and a significant change with treatment is still found in the experimental group while none is found in the controls.

Though the changes in the E group's insulin AIT are not significant, they do show a fall of 17% compared with no change in the C group. Reduction in blood glucose, despite lower or unchanged insulin levels, suggests enhanced insulin sensitivity in chromium-supplemented subjects. The significant insulin decrement, after supplementation, of 33% in the E group at 60 min strengthens the evidence that the improvement in glucose tolerance may be related to enhanced insulin sensitivity.

These results, showing improvement of glucose tolerance with chromium-rich yeast supplementation, are in agreement with several previous studies[4,6,7] in animals. In experimental chromium deficiency, tissue sensitivity to insulin decreased.[14] Mertz et al. demonstrated in rats[2,28] that chromium enhances the movement of glucose into tissues and chromium deficiency causes impaired glucose uptake and insulin resistance. These can be restored to normal by repletion of chromium.[2,28] Others found that plasma glucose of genetically diabetic mice improves after GTF supplementation.[29,30]

Recently, several investigators reported a marked improvement in glucose disposal and/or insulin output in man after chromium supplementation. Jeejeehboy et al.[31] described a patient who developed diabetes after receiving total parenteral nutrition for over 3 yr. Adding 250 μg of inorganic chromium to her infusate for 2 wk reversed the diabetes. A second, similar case has been reported since.[32] Possible additional evidence for a chromium-diabetes link was provided by a study showing that high iron saturation of transferrin in hemochromatosis patients decreased the chromium-binding capacity of the transferrin and, therefore, the retention of an injected ^{51}Cr dose.[33] The authors consider the low chromium status in hemochromatosis to be a likely contributing cause for the high incidence of diabetes in this disorder. Liu et al.[20] reported a correlation between increased serum chromium levels and improved glucose tolerance in 27 normal and hyperglycemic women, aged 40 to 75, who were supplemented with a chromium-rich brewers' yeast extract. Unfortunately, no placebo group was included. The present study also suggests that chromium has an ameliorative effect on glucose tolerance.

Conclusive evidence of a causal relationship awaits the development of truly accurate and reproducible biochemical methods to assess human chromium status.

Not only a diabetes-like state, but also an atherosclerotic-like syndrome was reported to be caused by chronic deficiency in rats.[6] Chromium supplements protected against hypercholesterolemia and decreased the incidence of plaques in the animals.[6,7] Rats fed a hypercholesterolemic diet for 35 days were protected from hypercholesterolemia when supplemented with chromium.[34]

The present study documents significantly improved serum cholesterol and total lipids in both diabetic and nondiabetic E subjects receiving the chromium-rich supplement, suggesting a role for chromium in human cholesterol metabolism. It is particularly significant that all individuals who were hypercholesterolemic (>300 mg/dl) showed a fall in their cholesterol levels, and the mean fall was −61 mg/dl. In those subjects receiving the chromium-poor supplement, no significant change in serum cholesterol and total lipids was found in the overall group, although a modest and significant fall in cholesterol was found in the nondiabetic group after supplementation. This improvement in C group nondiabetic cholesterol also raises the possibility of the presence of another beneficial factor in yeast in addition to chromium. Further studies are necessary to investigate this possibility.

Other workers reported improvement in serum triglyceride levels as a result of chromium supplementation.[11,29,30] The present study could document triglyceride reduction only in subjects having cholesterol levels >300 mg/dl.

Further studies, with larger numbers of subjects, especially diabetics, are needed to quantify the chromium effect and to relate glucose tolerance and serum lipid status to chromium levels in body tissues. At present, accurate methods for the clinical determination of levels of chromium in body tissues are still in the developmental state.[35,36] Blood, urine, and hair samples were investigated in several laboratories by a variety of techniques.[8–10,20,31–33,36,37] Recently, concurrent results were reported with normal values of <2 ppb in blood and of <1 ppb in urine.[20,36,37] However, these values are close to the sensitivity limits of present instrument detection, and any sample contamination during collection and preparation procedures introduces significant and unacceptable error.

We encountered some of these problems in this study. Urinary chromium levels were assayed before and after the period of yeast supplementation in samples collected from the subjects in both the E and C groups.† The increment in urinary chromium in several E subjects after a glucose challenge was twofold or threefold greater after the supplementary period than before. But low levels in some samples, coupled with known or suspected contamination in others, precluded collection of sufficiently reliable data to allow us to report it.

Newer, more sensitive, spectrophotometric methods and equipment may be expected to resolve these measurement difficulties in the near future. Once reliable measures of human chromium status become available, it may be possible to document accurately chromium deficiency and the effects of supplementation. If chromium deficiency is found to be prevalent in subgroups of the population and is also confirmed as a diabetes risk factor, then some cases of diabetes may be amenable to treatment by chromium supplementation.

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† We chose to measure chromium in urine samples to utilize the chromium-free dry ashing procedure developed at the Human Nutrition Center, Nutrition Institute, U.S.D.A., Beltsville, Md.[36] This method does not require the use of HNO_3, which is likely to contain not less than 5 ppb chromium and was used to prepare blood samples by the wet digestion method, thereby adding to contamination and to uncertainty of the result.

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