DISEASE MANAGEMENT
Volume 10, Number 1, 2007

DOI: 10.1089/dis.2006.628

A Conceptual Framework for Targeting Prediabetes with Lifestyle, Clinical, and Behavioral Management Interventions

THOMAS J. BIUSO, M.D., M.B.A.,[1] SUSAN BUTTERWORTH, Ph.D., M.S.,[2] and ARIEL LINDEN, Dr.P.H., M.S.[3,4]

ABSTRACT

Prediabetes is a condition that does not fall squarely into the primary or secondary prevention domain, and therefore tends to be inadequately addressed by interventions in either health promotion or disease management. Prediabetes is defined as having an impaired fasting glucose (fasting glucose of 100–125 mg/dL), impaired glucose tolerance (two-hour postprandial glucose of 140–199 mg/dL), or both. There is substantial evidence to suggest that even at these blood glucose levels, significant risk exists for both micro- and macrovascular complications. This paper introduces a conceptual framework of care for prediabetes that includes both screening and the provision of up-to-date clinical therapies in conjunction with an evidence-based health coaching intervention. In combination, these modalities represent the most effective means for delaying or even preventing the onset of diabetes in a prediabetes population. This paper concludes with a brief example in which these principles are applied to a hypothetical patient. (Disease Management 2007;10:6–15)

INTRODUCTION

Disease management (DM) is principally a secondary prevention model. Individuals are usually identified as having a chronic condition via hospital claims and the intent is to prevent further costly acute exacerbations. In contrast, health promotion efforts generally operate within the primary prevention domain and mainly target populations with preventive health messages with the intent of averting the onset of disease altogether. That said, there are conditions that, while being precursors to full-blown and/or irreversible disease states, still result in their own cluster of abnormalities and impaired health. These conditions do not fall squarely into the primary or secondary prevention domain, and therefore tend to be inadequately addressed by interventions in either health promotion or disease management.

Prediabetes is defined as having an impaired fasting glucose (IFG; fasting glucose of 100–125 mg/dL), impaired glucose tolerance (IGT; two-hour postprandial glucose of 140–199 mg/dL), or both. There is substantial evidence to suggest that even at these blood glucose levels,

[1] Regional Hospitalist Program Director, Apogee Medical, Tucson, Arizona.
[2]Oregon Health & Science University, School of Nursing, Portland, Oregon.
[3]Linden Consulting Group, Portland, Oregon.
[4]Oregon Health & Science University, School of Medicine and School of Nursing, Portland, Oregon.

significant risk exists for both micro- and macrovascular complications.[1–5]

Fortunately, multiple studies have indicated that the onset of diabetes can be delayed or even prevented in prediabetes patients by following the appropriate therapeutic regimens and adopting healthy lifestyle behaviors.[6–17] Given that there are approximately 41 million people in the United States aged 40–74 who have prediabetes,[18] implementing clinical and behavioral change interventions in this population makes sense from a societal and payer perspective. However, this may be easier said than done. Individuals with prediabetes are highly likely to have entrenched habits such as sedentary lifestyles, poor eating patterns, and overall poor weight management practices.[19]

This paper introduces a conceptual framework of care for prediabetes that includes screening and the provision of up-to-date clinical therapies in conjunction with an evidence-based health coaching intervention. We believe that, in combination, these modalities represent the most effective means for delaying or even preventing the onset of diabetes in a prediabetes population. This paper concludes with a brief example in which these principles are applied to a hypothetical patient.

ETIOLOGY AND SCREENING FOR PREDIABETES

Prediabetes and insulin-resistant states

Currently, most experts agree that type 2 diabetes mellitus (T2DM) is a multiorgan disease involving defects of glucose and fat metabolism in several organs, including not only the pancreatic beta cell, liver, and skeletal muscle, but also other organs such as the gut, kidney, brain, and nervous system. Diabetes begins as a prediabetic state characterized by insulin resistance. Resistance to the actions of insulin in many tissues, including the liver, adipose tissue, and muscle, is a central metabolic abnormality in patients who have prediabetes. Considerable information is available to suggest that a cluster of metabolic abnormalities related to insulin resistance and hyperinsulinemia increases cardiovascular risk and that these risk factors are present in insulin resistant, non-diabetic individuals. Patients with prediabetes may have a dyslipidemia characterized by high triglycerides and low high-density lipoprotein (HDL) levels.

Insulin resistance and hyperinsulinemia also are associated with polycystic ovarian syndrome, nonalcoholic fatty liver disease, prostate and pancreatic cancer, congestive heart failure, HIV lipodystrophy, antipsychotic medications, and sleep disordered breathing. Insulin resistance is also common in systemic inflammatory diseases such as rheumatoid arthritis. Aging is frequently associated with insulin resistance and glucose intolerance. Smoking, gestational diabetes, and a diet high in sweet soft drinks, refined grains, and processed meats are associated with increased risk of diabetes.

Prediabetes begins with an excessive intake of fatty acids in the diet. This excessive intake of fatty acids leads to an accumulation of triglycerides in adipose tissue. A net spillover of fatty acids from adipose tissue to non-adipose tissues such as muscle, liver, and the pancreas occurs. There is a reciprocal relationship between intramyocellular lipid accumulation and insulin sensitivity in healthy subjects. The deposition manifests as the visceral accumulation of fat and can be measured by computed tomography (CT) scan. This visceral accumulation of fat also explains insulin resistance in the lean individual because it is the fat surrounding such organs as the liver that leads to insulin resistance, not necessarily subcutaneous fat.

Prediabetes as a vascular disease

The pathophysiology of atherosclerosis and insulin resistance is similar in that both conditions are characterized by a proinflammatory state. There is convincing evidence that low-grade inflammation is a strong independent risk factor for the development of cardiovascular disease. It has become increasingly clear that inflammation correlates with endothelial dysfunction and insulin resistance, with the best evidence coming from patients with the metabolic syndrome.[20]

Although there are many abnormal bio-

chemical and transcriptional changes in the insulin resistant cell, researchers still do not agree on the initial triggering events. That said, interactions between a sensitive genotype and dietary factors such as a high-energy fatty diet interfere with normal cellular biochemical functions and insulin sensitivity. Scientists are studying nutrient-gene interactions, particularly with fatty acids as initial events in pathogenesis. These triggering events cause a cascade of biochemical and pathophysiologic reactions characterized by the activation of proinflammatory genes and the release of adipocytokines such as tumor necrosis factor, IL-6, leptin and macrophage migration inhibition factor. Their release contributes to insulin resistance in the liver, fat cell, pancreas, and skeletal muscle. In many cases, prediabetes patients already have vascular disease before developing diabetes. Vascular disease may manifest as retinal vasculopathy, carotid artery atherosclerosis, coronary artery disease, or peripheral artery disease. It is interesting that the SHAPE Task Force strongly recommends screening at-risk asymptomatic men 45–75 years of age and women 55–75 years of age for coronary artery disease.[21] Many of these asymptomatic individuals have subclinical atherosclerosis and prediabetes.

Screening for prediabetes

Unfortunately, we still define a large portion of the population as "normal" based upon dichotomous values for blood pressure, urinary albumin, lipids, and glucose levels. There is reason to believe that our current definition of "normal" values with respect to these parameters is really abnormal. Randomized trials illustrate the benefit of treating high-risk individuals with "normal" blood pressure.[22] The same applies to glucose levels. Two recent studies of non-diabetic individuals demonstrated that higher fasting plasma glucose levels within the normoglycemic range constitute an independent risk factor for type 2 diabetes and that coronary disease is more severe in those patients with higher postload glycemia and hemoglobin A1c (HbA1c) levels.[23,24]

The best ways to screen for prediabetes are with an oral glucose tolerance test and/or a fasting glucose. One can have a normal fasting glucose but an abnormal two-hour postprandial glucose level. The overlap between subjects with IFG and IGT is incomplete and suggests that they describe different pathophysiologic aspects of dysregulated glucose and fat metabolism. Multivariate analyses show that two-hour plasma glucose is closely associated with risk factors for diabetes and with cardiovascular variables, including triglycerides and apolipoprotein B. Individuals with high normal two-hour plasma glucose are more insulin resistant than normal individuals, have reduced insulin secretion, and higher plasma triglycerides and cholesterol/HDL ratios.[21]

There are three subsets of patients that must be identified in order to personalize treatment. They may be described in the following way: obese insulin resistant, metabolically healthy but obese (MHO), and metabolically obese normal weight (MONW) individuals. Obese individuals who are metabolically normal have lower levels of visceral fat, fasting insulin, plasma triglycerides, highly sensitive CRP (hs-CRP), and higher levels of HDL.[25] They are not insulin resistant. MONW patients have higher levels of visceral fat and are insulin resistant as measured by fasting insulin, intact proinsulin, and fasting glucose. These categorical subsets apply to younger age groups as well. Obese children and adolescents may be metabolically normal or abnormal. Young, obese patients may have elevated hs-CRP, abnormal triglycerides, and early carotid atherosclerosis as manifestations of insulin resistance.

TREATMENT

Lifestyle management

Although current treatment for prediabetes includes a pharmacological and lifestyle modification approach, lifestyle interventions are the cornerstone of treatment for this condition.[26] Insulin resistance is part of the underlying pathology associated with the metabolic syndrome, and patients identified with insulin resistance may have hypertension, dyslipidemia, visceral obesity, and vascular disease. Obesity, sedentary lifestyle, and high calorie, high-fat diets correlate with the development

of insulin resistance. Lifestyle changes and therapeutic dietary intervention have been demonstrated to prevent or delay the development of diabetes. In the Diabetes Prevention Program (DPP), a 58% relative reduction in the progression to diabetes was observed in the lifestyle group versus a 31% relative reduction in progression for the metformin group after 2.8 years.[7]

Current recommended lifestyle changes include a reduction in energy intake and an increase in physical activity. Both are inversely associated with the degree of insulin resistance. Lifestyle changes can prevent the development of diabetes. A moderate decrease in caloric balance (500–1000 kcal/day) results in slow, progressive weight loss when coupled with regular moderate-intensity physical activity (150 min/week of aerobic activity).[27] Reduction in saturated and *trans* fatty acids and cholesterol intake improves lipid status and insulin sensitivity. The most recent National Cholesterol Education Program guidelines recommend total fat intake between 25% and 35% of total calories and saturated fat of $<7\%$.[28]

In the Da Qing trial, 577 subjects with IGT were randomized into diet only, exercise only, diet plus exercise groups, and control groups and followed over six years. There was a 31% ($p \leq 0.03$), 46% ($p \leq 0.0005$), and 42% ($p \leq 0.005$) reduction in the risk of developing diabetes in these groups, respectively. This benefit applied to both lean and obese individuals even after controlling for insulin resistance, body mass index (BMI), and two-hour post-glucose level.[11,29]

Clinical management

Pharmacologic intervention also may prevent the development of diabetes. The DPP concluded that metformin may prevent progression to diabetes in insulin-resistant individuals. Participants in the STOP-NIDDM trial[30] with impaired glucose tolerance randomized to acarbose had a 25% relative risk reduction in progression to diabetes after 3.3 years. Interestingly, 72% of cardiovascular events occurred prior to the subjects developing diabetes. This fact emphasizes the importance of identifying prediabetes.

In the DPP,[7] metformin was half as effective as diet and exercise in delaying the onset of diabetes and was nearly ineffective in older people (age ≥ 60 years) or in those with a BMI ≤ 30 kg/m^2. Metformin was as effective as lifestyle modification in those subjects aged 24–44 years or in those with a BMI of ≥ 35 kg/m^2.

The thiazolidinediones are oral antidiabetic agents that improve insulin resistance and decrease plasma glucose and insulin concentrations in patients with T2DM. They are selective PPAR-γ receptor agonists that have antiatherosclerotic properties. These receptors are found in target organs that are integral for insulin action including the liver, adipose tissue, and skeletal muscle. Thiazolidinediones improve the dyslipidemia of T2DM and the metabolic syndrome. Studies are currently under way to investigate the impact of using these agents to treat prediabetes.[31]

The Xenodos Trial[32] followed subjects (BMI $>$ 30 kg/m^2) treated with Xenical over 4 years. Orlistat normalized blood glucose in 72% of individuals in this group versus 49% in placebo. Three percent of patients treated with orlistat versus 7.6% in the placebo group progressed to diabetes, a greater than 50% reduction in incident diabetes. Sibutramine is a weight reducing medication that suppresses appetite by preventing the reuptake of serotonin, norepinephrine, and, to a lesser extent, dopamine. In a double-blind randomized controlled trial,[33] 359 obese subjects without hypertension or diabetes at baseline were randomized to the drug or placebo. Sibutramine was associated with significant weight loss and improvement in insulin sensitivity.

Rimonabant is another drug associated with weight loss and improvement in insulin sensitivity. Rimonabant is the first selective blocker of the cannabinoid receptor type 1 (CB_1). These receptors are present in all tissues that play an important role in the regulation of food intake. Rimonabant increases adiponectin levels, leads to significant weight loss, and has glucose lowering properties. The drug also reduces the expression of multiple proinflammatory cytokines that are upregulated in obesity.[34]

Finally, blockade of the renin angiotensin system (RAS) by angiotensin-converting enzyme inhibitors or angiotensin receptor blockers have antidiabetic effects. Several insulin sig-

naling systems are influenced by RAS, and several studies show that blockade of this system ameliorates insulin resistance. Long-term clinical trials will clarify their role in the treatment of prediabetes.[35]

The role of pharmacologic intervention in prediabetics needs further definition and ongoing studies will answer those questions. Certainly, anti-obesity drugs are appropriate for some obese patients. Surgery also has a place in the treatment of these patients. Over the last several years, bariatric surgical intervention has played an increasingly important role in the care of morbidly obese patients. This surgical technique has rapidly diffused among surgeons in the United States, and appropriate selection criteria exist in order to minimize morbidity and mortality in the perioperative period. Numerous studies have shown that in carefully selected patients there is significant weight loss (over 30% in some studies), decrease in BMI, reduction in blood pressure, and amelioration of insulin resistance.[36]

In summary, there is convincing evidence to suggest that prediabetes can be managed successfully with lifestyle and clinical interventions. However, getting patients to make and maintain behavior changes and adhere to treatment regimes requires a compelling approach. In addition, one must consider the costs. Private payers often are reluctant to cover preventive interventions that have substantial initial costs and delayed benefits.[37] Coverage decisions are often based on a strong business case that is defined as a positive return on investment (ROI). In a recent paper, Ackerman and colleagues addressed the costs for a payer to treat members with prediabetes aged 50–64.[38] Compared with placebo, the DPP intervention could prevent 37% of new cases of diabetes before age 65 at a cost of $1288 per QALY. A private payer could contribute 24% of total discounted intervention costs and achieve positive ROI after 3 years. Each year thereafter (years 4–15) results in cost savings for the health plan. In this scenario, the residual payment by the employer or member amounts to $44 per month.

Behavioral management

As mentioned above, along with pharmacological interventions, several recent controlled trials on diabetes prevention have confirmed that lifestyle changes targeting diet, weight loss, and exercise can substantially delay or prevent the progression from impaired metabolism to type 2 diabetes.[6–8,11] However, while there are numerous examples of successful interventions to improve diet, activity patterns, and weight regulation, there is still no consensus on a standard or systematic approach that supports sustained behavior change in any of these areas.[39,40] A significant mediating factor determining successful behavior change is self-efficacy (SE), or one's belief about his or her ability to accomplish something.[41] It has been cited as a correlate with clinical outcomes, and influences whether an individual will even attempt to make behavioral changes.[42] Other mediating factors that have been cited in the literature include readiness to change[43]; ambivalence and motivation[44]; beliefs, values, and expectations[35]; and implementation intentions.[45]

In addition to the challenges of changing entrenched lifestyle habits, comorbid conditions such as depression can be a complicating factor when addressing any chronic medical condition.[46] In the case of prediabetes, the presence of depression or chronic stress has been shown to exacerbate the diabetes disease process, and has been correlated with poor participation in education programs and poor adherence to self-care behaviors such as medication and diet regimens.[47,48] Moreover, a meta-analysis confirmed that depressed patients were three times more likely than non-depressed patients to be non-compliant with physician recommendations.[49]

Traditionally, diabetes education (which is similar to prediabetes education) has emphasized increasing knowledge about diabetes, risk factors, and diabetes self-care; however, multiple studies have demonstrated that this pedagogical approach does not result in optimal clinical or behavioral outcomes.[50–52] Rather, efforts should focus on improving coping, communication, and control by enhancing SE, increasing motivation to initiate and/or change behaviors, and facilitating an individualized plan of action that takes into account personal needs, barriers, and preferences.[44,53,54] Therefore, considerable care must be taken to implement a behavioral change program that

includes these components. Likewise, recent literature supports interventions for patients/members with chronic conditions that also screen for and address depression and chronic stress.[55]

A novel intervention modality recently introduced in DM that includes these criteria is a health coaching approach that utilizes the Motivational Interviewing (MI) technique to address lifestyle-related issues known to impede the member/patient's self-management of chronic illness. Health coaching is an emerging field in which health professionals (eg, dietitians, nurses, counselors) facilitate behavior modification in clients to improve their health. MI-based health coaching embraces a set of techniques that is evidence based and involves a discrete skill set of the coach/provider that can be objectively coded and measured.[56]

MI was originally developed for addictions counseling in the 1980s and is described as a "directive, client-centered counseling style for eliciting behavior change by helping clients to explore and resolve ambivalence.[44] It has been well researched in randomized controlled trials for use in treating addictions such as illegal drugs, smoking, and alcoholism.[57–59] As the value of lifestyle management has become more fully realized, MI has expanded into health promotion and disease management settings and typically is employed in a health coaching application in the format of several telephonic sessions.

This method is different from traditional health education approaches in that it is not based on the information model, does not use scare tactics, and is not confrontational, forceful, guilt-inducing, or authoritarian[44]; rather it is shaped by an understanding of what triggers change.[60] A recent meta-analysis found that in a scientific setting MI outperforms traditional advice-giving in the treatment of a broad range of behavioral problems and diseases.[53]

Studies in this area have utilized the MI approach in the intervention for increasing fruit and vegetable intake,[61,62] promoting physical activity,[63–66] medication adherence,[67,68] managing hypertension and hypercholesterolemia,[69,70] and behavioral obesity treatment.[71,72] A recent meta-analysis by Knight of MI in the physical healthcare setting indicated that MI had high face validity across a number of domains, although more well-controlled research was recommended.[49]

MI has also been used successfully to promote self-care for both adolescents and adults with diabetes.[73–76] All studies demonstrated significant improvement in diabetes self management and/or clinical outcomes such as HbA1c scores, and in one study,[73] the adolescents reported less anxiety about their condition and more confidence that they could control it.

Promising results have also been shown in the application of MI to mental health issues (in addition to substance abuse) such as anxiety and depression.[77–79] In one study, MI-based health coaching significantly improved mental as well as physical health status scores, although the health coaches were not counselors and the presenting health concerns were typical lifestyle-related ones such as weight management, exercise, stress management, and nutrition.[80]

Supporting SE is one of the four principle objectives of MI.[44] As mentioned previously, the client's belief that change is possible is an important motivator to succeeding in making a change:

> The client can be helped to develop a belief that he or she can make a change. For example, the clinician might inquire about other healthy changes the client has made in their life, highlighting skills the client already has. Sharing brief clinical examples of other, similar clients' successes at changing the same habit or problem can sometimes be helpful. In a group setting, the power of having other people who have changed a variety of behaviors during their lifetime gives the clinician enormous assistance in showing that people can change.[81]

In supporting and increasing SE, the health coach or provider can increase motivation for change and increase the likelihood of a successful behavior change effort, which will result in a better clinical outcome.

In a successful session using MI-based health coaching, the coach emphasizes the three underlying assumptions of MI—collaboration, the evocative element, and autonomy—in order to

establish rapport, reduce resistance, improve SE, and elicit "change talk" (one's own reasons and arguments for change).[44,82] The intended outcome of these MI sessions is for clients to resolve ambivalence (a central goal), move through the stages of change,[43,83] and follow through on desirable lifestyle change, which would ideally result in improved health outcomes.

Other characteristics of this technique that make it particularly suitable for use in disease management to address prediabetes are as follows: (1) it is most effective when implemented with clients who are considered difficult (ie, reluctant to change, stuck, or ambivalent about changing their behavior); (2) it has been found to be efficacious in small doses (2–3 sessions); (3) it has been found to work across gender, age, cultural, and socioeconomic boundaries; and (4) it has been found to be an effective pretreatment adjunct to traditional disease management programs.[84,85]

It is becoming more widely acknowledged that most lifestyle changes are infused with psychosocial dynamics such as ambivalence, SE, self-image, motivation, self-doubt, and core identity.[43,44,86–88] As described by Prescott:

> MI views people as complex, driven by competing motives and in conflict with themselves. This complexity is noticeable in motivational conflict (ambivalence) and fluctuating levels of self efficacy (both optimism and doubts about being able to change grow and fade).[89]

Thus it appears that MI is also particularly well suited for impacting the psychosocial aspects of desired behavior change in prediabetes.

AN EXAMPLE OF MOTIVATIONAL INTERVIEWING–BASED HEALTH COACHING FOR PREDIABETES

Once an individual is identified as having prediabetes via laboratory values, a health coach is assigned to the case. Over several telephone sessions, the health coach uses the following MI-based coaching techniques: rapport-setting/building; agenda-setting (identification of critical health behavior); exploration of ambivalence; assessment of importance/confidence/readiness; development of discrepancy (acknowledging the gap between current and ideal behaviors); support of SE; identification of action plan; appropriate referrals, resources, or information; and a follow-up plan.

In this example, Ruth, a 52-year-old woman with a family history of diabetes has an IFG of 119 and an IGT of 165. She has high cholesterol (257) and mild hypertension (142/92), and is obese (BMI = 35). Her provider has prescribed enalapril and lovastatin, and recommended lifestyle change.

During the initial rapport-building segment of health coaching, the health coach (Maria) explores Ruth's current health habits, and overall knowledge of and attitude about her condition. Maria establishes that Ruth is sedentary, lives alone, does not like to cook, and is taking her medication on a regular basis. Ruth is fairly well informed about prediabetes and is very concerned about it developing into diabetes. Maria ascertains that Ruth has low SE about her ability to lose weight because she has failed at several previous attempts.

During the agenda-setting portion of health coaching, Maria validates Ruth's medication adherence and directs her toward exercise and appropriate dietary choices as her primary goals. Over the course of the first three sessions, Maria has explored Ruth's ambivalence, barriers, and available resources. They jointly develop a feasible and detailed plan of action that includes walking five days a week, cutting back on fast food, including more fruits and vegetables in her food preparations, and eating smaller portion sizes. Maria continues to work with Ruth on improving her confidence levels. By the fourth coaching session, success indicators include increased SE for weight management, healthier lifestyle habits, and, most importantly, improved blood glucose values.

CONCLUSION

Although there are currently no consensus guidelines on the screening and treatment of prediabetes, the recent literature underscores the importance of screening, introducing the appropriate therapeutic regimens, and adopt-

ing healthy lifestyle behaviors in order to delay or even prevent the onset of diabetes in prediabetes patients. The best way to screen for these individuals is with either a fasting glucose and/or an oral glucose tolerance test. These individuals cannot be reliably identified from claims data unless the clinician codes for glucose intolerance, or possibly, metabolic syndrome. A DM program targeting this population will require the cooperation of its physician network in the identification process. We believe that there will be additional compelling evidence that warrants further scrutiny of prediabetes as a condition to be considered for DM using a MI-based health coaching approach.

REFERENCES

1. The DECODE Study Group, the European Diabetes Epidemiology Group. Glucose tolerance and cardiovascular mortality: comparison of fasting and 2-hour diagnostic criteria. Arch Intern Med 2001;161:397–405.
2. Saydah SH, Loria CM, Eberhardt MS, Brancati FL. Subclinical states of glucose intolerance and risk of death in the U.S. Diabetes Care 2001;24:447–453.
3. Coutinho M, Gerstein HC, Wang Y, Yusuf S. The relationship between glucose and incident cardiovascular events: a metaregression analysis of published data from 20 studies of 95,783 individuals followed for 124 years. Diabetes Care 1999;22:233–240.
4. Balkau B, Shipley M, Jarrett RJ, et al. High blood glucose concentration is a risk factor for mortality in middle-aged nondiabetic men: 20-year follow-up in the Whitehall Study, the Paris Prospective Study, and the Helsinki Policemen Study. Diabetes Care 1998;21:360–367.
5. Bjornholt JV, Erikssen G, Aaser E, et al. Fasting blood glucose: an underestimated risk factor for cardiovascular death: results from a 22-year follow-up of healthy nondiabetic men. Diabetes Care 1999;22:45–49.
6. Tuomilehto J, Lindstrom J, Eriksson JG, et al. Prevention of type 2 diabetes mellitus by changes in lifestyle among subjects with impaired glucose tolerance. N Engl J Med 2001;344:1343–1350.
7. Knowler WC, Barrett-Connor E, Fowler SE, et al. Reduction in the evidence of type 2 diabetes with lifestyle intervention or metformin. N Engl J Med 2002;346:393–403.
8. The Diabetes Prevention Program: design and methods for a clinical trial in the prevention in type 2 diabetes. Diabetes Care 1999;22:623–634.
9. The Diabetes Prevention Program Research Group. The Diabetes Prevention Program: baseline characteristics of the randomized cohort. Diabetes Care 2000;23:1619–1629.
10. Buchanan TA, Xiang AH, Peters RK, et al. Preservation of pancreatic beta-cell function and prevention of type 2 diabetes by pharmacological treatment of insulin resistance in high-risk hispanic women. Diabetes 2002;51:2796–2803.
11. Pan XR, Li GW, Hu YH, et al. Effects of diet and exercise in preventing NIDDM in people with impaired glucose tolerance: the Da Qing IGT and Diabetes Study. Diabetes Care 1997;20:537–544.
12. Tuomilehto J, Lindstorm J, Eriksson JG, et al. Finnish Diabetes Prevention Study Group: Prevention of type 2 diabetes mellitus by changes in lifestyle among subjects with impaired glucose tolerance. N Engl J Med 2001;344:1343–1350.
14. Diabetes Prevention Program Research Group: The Diabetes Prevention Program: reduction in the incidence of type 2 diabetes with lifestyle intervention or metformin. N Engl J Med 2002;346:393–403.
15. Chiasson J, Josse RG, Gornis R, et al. Acarbose for prevention of type 2 diabetes mellitus: the STOP-NIDDIM randomized trial. Lancet 2002;359:2072–2077.
16. Ramachandran A, Snehalatha C, Mary S, et al. The Indian Diabetes Prevention Programme shows that lifestyle modification and metformin prevent type 2 diabetes in Asian Indian subjects with impaired glucose tolerance (IDPP-1). Diabetologia 2006;49:289–297.
17. Ratner RE, The Diabetes Prevention Program Research. An update on the Diabetes Prevention Program. Endocr Pract 2006;12:20–24.
18. American Diabetes Association. Prediabetes. Available at: www.diabetes.org/diabetes-prevention/prediabetes.jsp. Accessed July 24, 2006.
19. American Diabetes Association. The prevention or delay of type 2 diabetes. Diabetes Care 2002;25:742–749.
20. Giugliano D, Ceriello A, Esposito K. The effects of diet on inflammation. J Am Coll Cardiol 2006;48:677–685.
21. Naghavi M, Falk E, Hecht H, et al. From vulnerable plaque to vulnerable patient—Part III: executive summary of the Screening for Heart Attack Prevention and Education (SHAPE) Task Force report. Am J Cardiol 2006;98(suppl):2H–15H.
22. Forman JP, Brenner BM. "Hypertension" and "microalbuminuria": the bell tolls for thee. Kidney Int 2006;69:22–28.
23. Tirosh A, Shai I, Likes-Manova D, et al. Normal fasting blood glucose levels and type 2 diabetes in young men. N Engl J Med 2005;353:1454–1462.
24. Sasso F, Carbonara O, Nasti R, et. al. Glucose metabolism and coronary heart disease in patients with normal glucose tolerance. JAMA 2004;291:1857–1863.
25. Karelis AD, Faraj M, Bastard JP, et. al. The metabolically healthy but obese individual presents a favorable inflammation profile. J Clin Endocrinol Metab 2005;90:4145–4150.
26. Chiasson JL, Brindisi MC, Rabasa-Lhoret R. The prevention of type 2 diabetes: what is the evidence? Minerva Endocrinol 2005;30:179–191.
27. Phillips C, Lopez-Miranda J, Perez-Jimenez F, et al. Genetic and nutrient determinants of the metabolic syndrome. Curr Opin Cardiol 2006;21:185–193.

28. Manco M, Calvani M, Mingrone G. Effects of dietary fatty acids on insulin sensitivity and secretion. Diabetes Obes Metab 2004;6:402–413.
29. Li G, Hu Y, Yang W, et al. Effects of insulin resistance and insulin secretion on the efficacy of interventions to retard development of type 2 diabetes mellitus: the Da Qing IGT and Diabetes Study. Diabetes Res Clin Pract 2002;58:193–200.
30. Chiasson JL, Josse RG, Gomis R, et.al. Acarbose for the prevention of type 2 diabetes mellitus: the STOP-NIDDM randomized trial. Lancet 2002;359:2072–2077.
31. Zinman B, Harris SB, Gerstein HC, et al. Preventing type 2 diabetes using combination therapy: design and methods of the Canadian Normoglycaemia Outcomes Evaluation (CANOE) trial. Diabetes Obes Metab 2006;8:531–537.
32. Heymsfield SB, Segal KR, Hauptman J, et al. Effects of weight loss with orlistat on glucose tolerance and progression to type 2 diabetes in obese adults. Arch Intern Med 2000;160:1321–1326.
33. Faria AN, Ribeiro FF, Kohlmann NE, et al. Effects of sibutramine on abdominal fat mass, insulin resistance and blood pressure in obese hypertensive patients. Diabetes Obes Metab 2005;7:246–253.
34. Gelfand EV, Cannon CP. Rimonabant: a cannabinoid receptor type blocker for management of multiple cardiometabolic risk factors. J Am Coll Cardiol 2006;47: 1919–1926.
35. Ando K, Fujita T. Antidiabetic effect of blockade of the renin angiotensin system. Diabetes Obes Metab 2006;8:396–403.
36. Ferchak CV, Meneghini LF. Obesity, bariatric surgery and type 2 diabetes—a systematic review. Diabetes Metab Res Rev 2004;20:438–445.
37. Leatherman S, Berwick D, Iles D, et al. The business case for quality: case studies and an analysis. Health Aff (Millwood) 2003;22:17–30.
38. Ackermann RT, Marrero DG, Hicks KA, et al. An evaluation of cost sharing to finance a diet and physical activity intervention to prevent diabetes. Diabetes Care 2006;29:1237–1241.
39. Brug J, Oenema A, Ferreira I. Theory, evidence and Intervention Mapping to improve behavior nutrition and physical activity interventions. Int J Behav Nutr Phys Act 2005;2:2. Available at: www.ijbnpa.org/content/2/1/2. Accessed December 1, 2006.
40. Dzewaltowski DA, Estabrooks PA, Klesges LM, Bull S, Glasgow RE. Behavior change intervention research in community settings: how generalizable are the results? Health Promot Int 2004;19:235–245.
41. Bandura A. Health promotion by social cognitive means. Health Ed Behav 2004;31:143–164.
42. Marks R, Allegrante JP, Lorig K. A review and synthesis of research evidence for self-efficacy-enhancing interventions for reducing chronic disability: implications for health education practice (part I). Health Promot Pract 2005;6:37–43.
43. Prochaska JO. Systems of psychotherapy: a transtheoretical analysis. Homewood, IL: Dorsey Press, 1979.
44. Miller WR, Rollnick S. Motivational interviewing: preparing people to change addictive behavior. New York: Guildford Press; 2002.
45. Gollwitzer PM. Implementation intentions: strong effects of simple plans. American Psychologist 1999;54: 493–503.
46. Patten SB. An analysis of data from two general health surveys found that increased incidence and duration contributed to elevated prevalence of major depression in person with chronic medical conditions. J Clin Epidemiol 2005;58:184–189.
47. Lustman PJ, Clouse RE. Depression in diabetic patients: the relationship between mood and glycemic control. J Diabetes Complications 2005;19:113–122.
48. Park H, Hong Y, Lee H, Ha E, Sung Y. Individuals with type 2 diabetes and depressive symptoms exhibited lower adherence with self-care. J Clin Epidemiol 2004;57:978–984.
49. Knight KM, McGowan L, Dickens C, Bundy C. A systematic review of motivational interviewing in physical health care settings. Br J Health Psychol 2006;11: 319–332.
50. Bradley C. Health beliefs and knowledge of patients and doctors in clinical practice and research. Patient Educ Couns 1995;26:99–106.
51. Brown SA. Studies of educational intervention and outcomes in diabetic adults: a meta-analysis revisited. Patient Educ Couns 1990;16:189–215.
52. Grumbach K. Patient self-management of chronic disease in primary care. JAMA 2002;288:2469–2475.
53. Krichbaum K, Aarestad V, Buethe M. Exploring the connection between self-efficacy and effective diabetes self-management. Diabetes Educ 2003;29:653–662.
54. Marks R, Allegrante JP, Lorig K. A review and synthesis of research evidence for self-efficacy-enhancing interventions for reducing chronic disability: implications for health education practice (part II). Health Promot Pract 2005;6:148–156.
55. NIOSH. Worker health chartbook 2004. NIOSH publication no. 2004-146. Chapter 2: fatal and nonfatal injuries, and selected illnesses and conditions. Available at: www.cdc.gov/niosh/docs/ chartbook/. Accessed March 6, 2006.
56. Madson MB, Campbell TC. Measures of fidelity in motivational enhancement: a systematic review. J Subst Abuse Treat 2006;31:67–73.
57. Ershoff DH, Quinn VP, Boyd NR, Stern J, Gregory M, Wirtshafter D. The Kaiser Permanente prenatal smoking-cessation trial: when more isn't better, what is enough? Am J Prev Med 1999;17:161–68.
58. Aubrey LL. A motivational intervention for adolescent smokers. Prev Med 1998;27:A39–A46.
59. Masterman PW, Kelly AB. Reaching adolescents who drink harmfully: fitting intervention to developmental reality. J Subst Abuse Treat 2003;24:347–355.
60. Rubak S, Sandbæk A, Lauritzen T, Christensen B. Motivational interviewing: a systematic review and meta-analysis. Br J Gen Pract 2005;55:305–312.
61. Resnicow K, Jackson A, Wang T, et al. A motivational

interviewing intervention to increase fruit and vegetable intake through black churches: results of the Eat For Life trial. Am J Public Health 2001;91:1686–1693.
62. Berg-Smith SM, Stevens VJ, Brown KM, et al. A brief motivational intervention to improve dietary adherence in adolescents. Health Educ Res 1999;3:399–410.
63. Brodie DA, Inoue Al. Motivational interviewing to promote physical activity for people with chronic heart failure. JAN J Adv Nurs 2005;50:518–527.
64. Hudec JC. Individual counseling to promote physical activity. Dissertation Abstracts Int 2000;61:931.
65. Harland J, White M, Drinkwater C, Chinn D, Farr L, Howel D. The Newcastle exercise project: a randomized controlled trial of methods to promote physical activity in primary care. BMJ 1999;319:828–832.
66. Jones KD, Burckhardt CS, Bennett JA. Motivational interviewing may encourage exercise in persons with fibromyalgia by enhancing self efficacy. Arthritis Rheum 2004;51:864–867.
67. Aliotta SL, Vlasnik JJ, Delor B. Enhancing adherence to long-term medical therapy: a new approach to assessing and treating patients. Adv Ther 2004;21: 214–231.
68. Kemp R, Kirov G, Everitt B, et al. Randomised controlled trial of compliance therapy. 18-month follow-up. Br J Psychiatry 1998;172:413–419.
69. Woollard J, Beilin L, Lord T, et al. A controlled trial of nurse counselling on lifestyle change for hypertensives treated in general practice: preliminary results. Clin Exp Pharmacol Physiol 1995;22:466–468.
70. Mhurchu CN, Margetts BM, Speller V. Randomised clinical trial comparing the effectiveness of two dietary interventions for patients with hyperlipidaemia. Clin Sci 1998;95:479–487.
71. DiLillo V, Siegfried NJ, West DS. Incorporating motivational interviewing into behavioral obesity treatment. Cognit Behav Pract 2003;10:120–130.
72. Smith DE, Heckemeyer CM, Kratt PP, Mason DA. Motivational interviewing to improve adherence to a behavioral weight-control program for older obese women with NIDDM: a pilot study. Diabetes Care 1997;20:52–54.
73. Channon S, Smith VJ, Gregory JW. A pilot study of motivational interviewing in adolescents with diabetes. Arch Dis Child 2003;88:680–683.
74. Clark M, Hampson SE. Implementing a psychological intervention to improve lifestyle self-management in patients with Type 2 diabetes. Patient Educ Couns 2001;42:247–256.
75. Knight KM, Bundy C, Morris R, et al. The effects of group motivational interviewing and externalizing conversations for adolescents with type-1 diabetes. Psychol Health Med 2003;8:149–157.
76. Pill R, Stott NC, Rollnick SR, Rees M. A randomised controlled trial of an intervention designed to improve the care given in general practice to type II diabetic patients: patient outcomes and professional ability to change behaviour. Fam Pract 1998;15: 229–235.
77. Arkowitz H, Westra HA. Integrating motivational interviewing and cognitive behavioural therapy in the treatment of depression and anxiety. J Cognit Psychother 2004;18:337–350.
78. Westra HA. Managing resistance in cognitive behavioural therapy: application of motivational interviewing in mixed anxiety depression. Cognit Behav Ther 2004;33:161–175.
79. Kelly AB, Halford WK, Young RM. Maritally distressed women with alcohol problems: the impact of a short-term alcohol-focused intervention on drinking behaviour and marital satisfaction. Addiction 2000;95:537–549.
80. Butterworth S, Linden A. The effect of motivational interviewing-based health coaching on employees' physical and mental health status. J Occup Health Psych 2006;11:358–365.
81. ATTC. Motivational interviewing principals 2003. Available at: www.motivationalinterview.org/clinical/principles.html Accessed August 17, 2006.
82. Miller W. Motivational interviewing in service to health promotion. Art Health Promot 2004;18: A1–A10.
83. Linden A, Roberts N. Disease management interventions: what's in the black box? Dis Manage 2004;7:275–291.
84. Burke BL, Arkowitz H, Menchola M. The efficacy of motivational interviewing: a meta-analysis of controlled clinical trials. J Consult Clin Psychol 2003;71: 843–861.
85. Hettema J, Steele J, Miller WR. Motivational interviewing. Annu Rev Clin Psychol 2005;1:91–111.
86. Bodenheimer T, Lorig K, Holman H, Grumback K. Patient self-management of chronic disease in primary care. JAMA 2005;288:2469–2475.
87. Holahan CK, Suzuki R. Adulthood predictors of health promoting behavior in later aging. Int J Aging Hum Dev 2004;58:289–313.
88. Loeb SJ. Older men's health: motivation, self-ratings, and behaviors. Nurs Res 2004;53:198–206.
89. Prescott P. Similarities and differences between cognitive therapy and MI. Presented at the MINT Forum 2005: Highlights of the Annual Meeting of the Motivational Interviewing Network of Trainers, Amsterdam, The Netherlands, 2006.

Address reprint requests to:
Ariel Linden, DrPH, MS
Linden Consulting Group
6208 NE Chestnut St.
Hillsboro, OR 97124

E-mail: alinden@lindenconsulting.org