THE *EN BALANCE* SPANISH DIABETES EDUCATION PROGRAM IMPROVES APOLIPOPROTEINS, SERUM GLUCOSE AND BODY COMPOSITION IN HISPANIC DIABETICS

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Objective: We evaluated the changes in apolipoproteins, glycemic status, and body composition after 3 months using a culturally sensitive diabetes education program, *En Balance,* in diabetic Hispanics.

Methods: Thirty-four (9 males, 25 females) Hispanic diabetics participated in the *En Balance* program over three months. Body composition was determined by dual energy X-ray absorptiometry (DXA), fasting plasma glucose (FPG), A1c, and apolipoproteins (Apo) measured after 3 months participation. Differences were analyzed using paired *t* testing and relationships between changes in Apo, A1c, total cholesterol, body mass index and body composition by Spearman correlations.

Results: Completion of *En Balance* resulted in a significant reduction in weight (80.31 ± 1.97 kg vs 81.25 ± 17.97 kg, *P*=.015), FPG (143.21 ± 57.8 mg/dL vs 166.41 ± 65.9 mg/dL *P*=.003), and A1c (7.08 ± 1.6% vs 7.87 ± 2.0%, *P*=<.001). DXA demonstrated reduction in total fat (29.54 ± 10.0 kg vs 30.24 ± 11.80 kg, *P*=<.001) and trunk fat (15.09 ± 5.6 kg vs 16.87 ± 5.4 kg, *P*=.001). High density lipoprotein significantly increased (48.85 ± 11.4 vs 44.65 ± 8.8, *P*=.002) and total serum cholesterol/high density lipoprotein ratio decreased (3.87 ± .98 vs 4.35 ± 1.0, *P*=.001). There were significant correlations at three months between changes in Apo A1 and A2 (*r*=.559, *P*<.001), Apo E and total cholesterol (*r*=.746, *P*<.001), between A1c and FPG (*r*=.563, *P*=.001) and BMI and body weight (*r*=.732, *P*<.001).

Conclusions: The *En Balance* program improved body composition, A1c, FPG, total cholesterol/HDL ratio and HDL. If these trends can be sustained, *En Balance* may serve as a unique educational paradigm for improving type 2 diabetes in Hispanics. (*Ethn Dis.* 2012;22(2):215–220)

Key Words: Hispanics, Type 2 Diabetes, Diabetes Education Programs

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INTRODUCTION

Diabetes mellitus type 2 (DM2) is one of the major causes of morbidity and mortality in the United States with a ~100% increase between 1980 and 2004.[1–5] Worldwide, 220 million people have diabetes and 1.1 million have died from complications of DM2 with projections that DM related mortality will double between 2005 and 2030.[2–3] Hispanic Americans in particular are more likely to suffer from DM and DM-related complications than Caucasians[6–9] and there is clear evidence that disparities based on ethnic differences play a significant role in diabetes-related morbidity and mortality.[9] Although many factors play a role in the increased risk of DM2 and complications in the Hispanic community, low socioeconomic status,[10–11] low educational attainment,[11–12] and limited access to health care[13] are major contributors to this health disparity. Lifestyle challenges in Hispanics are particularly important due to the effects of both immigration and acculturation on the ability to attain and achieve healthy habits.

Ideally, culturally sensitive education programs focused on the Hispanic lifestyle should be particularly important in decreasing cardiovascular risks but to date no program has reported success. One aspect of lifestyle intervention that may play a particularly key role is the effects on diabetic dyslipidemia, which is one of the most important contributors to diabetes vascular complications. Dyslipidemia in DM2 is usually manifested as decreased high density lipoprotein (HDL) and elevated triglyceride (TG) levels, which strongly correlates with coronary heart disease.[14] Additionally, apolipoproteins (Apo) A, C, and E, play significant roles in Hispanic diabetics because of the contribution of certain Apo and lipids in initiating the process of atherosclerosis and insulin resistance. Health education targeting lipids in DM2 leads to significant reductions in macrovascular and microvascular complications.[14]

En Balance was designed to have a strong nutritional component and cause beneficial changes in the diabetic dyslipidemia pattern in Hispanic diabetics. Although limited to post-hoc analysis, we sought to determine the effects of *En Balance* on lipoprotein changes within the same participants over a 3-month time period.

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METHODS

Research Study Design

The *En Balance* program is a 3-month trial to test the impact of a Spanish language diabetes education program on blood glucose control in diabetic Hispanics. The Loma Linda University Institutional Review Board approved the study protocol and all participants gave written informed consent to participate in Spanish. Subjects were screened by telephone and personal interviews evaluating medical histories, medications usage (oral hypoglycemic or insulin), physical activity, and diet histories by questionnaires. Our study included the following phases: recruitment, collection of baseline data, three months of diabetes education, collection of data at the end of the 3 months, and final data analysis. Spanish diabetes health education classes were offered during 3-months of follow-up. Classes were held twice a week for a period of 2 hours teaching participants how to maintain blood sugar levels and body weight checks. Registered nurses, students, physicians and registered dietitians were part of the team of instructors. All educational materials were in Spanish. A comprehensive evaluation of physical, blood, dietary and body composition assessments aimed at fully characterizing health status, dietary habits and total and regional body composition were done at baseline and at 3-months. Individuals were excluded if they: were pregnant or lactating, had a history of drug or alcohol abuse, had an impaired mental condition, were on active glucocorticoid therapy, or had unstable cardiovascular disease, hepatic, neurologic, endocrine, or other major systemic disease. Patients with pacemakers were excluded due to incompatibility with DXA measurements. A total of 34 participants (9 males and 25 females) with a known diagnosis of DM2 for >5 years and body mass index (BMI) of 21–47 kg/m^2 participated in the study.

Outcome Measures

Body composition, which assesses total and visceral fat, was determined as it plays an important role in weight-related diseases like DM2 and hypertension. Visceral fat in particular is related to insulin resistance leading to DM2,[15] and accumulation is higher in Hispanic adult and children when compared to other ethnic groups.[16] We measured body composition using DXA (Software version 12.6); total and regional body composition measurements were obtained and certain body regions of interest were isolated with participants in the supine position. For each assessment, scan time was about 3 minutes at a radiation exposure of 1.5 mrem. These scans were conducted according to standard procedures[17] and evaluated by a radiologist (ES) at baseline and three months.

Fasting blood samples were collected in the morning at baseline and 3-months. Blood samples were analyzed in the Loma Linda University clinical laboratory using established techniques for lipoproteins, glucose, insulin, cholesterol, and A1c.[18,19]

Study participants completed the validated Southwestern Food Frequency questionnaire at baseline and at three months. Dietary intake records were analyzed using the Metabolize Nutrient Analysis System, Version 2.5, at the University of Arizona, Arizona Cancer Center, Tucson, Arizona.

Weight (kg) was measured at baseline and 3 months using a balance scale (Detecto, Web City, Mo). Body weight and height were measured using a stadiometer (Holtain Ltd., Crmych, Dyfed, England).[20]

Statistical Analysis

Statistical analyses were calculated using SPSS for windows version 15.0 (SPSS, Inc, Chicago, Illinois). Log transformations were used to improve normality for the following: FPG, A1c, total cholesterol (tchol), fasting insulin, LDL cholesterol (LDLc), HDL cholesterol (HDLc), and triglycerides (TG).

Table 1. Baseline characteristic of study participants (both sexes, *N*=34)

Variables	(*n*) %
Age, years	
37–39	(4) 11.8
40–49	(9) 26.5
50–59	(14) 41.2
60–69	(7) 20.6
Sex	
Female	(25) 73.5
Male	(9) 26.5
Weight, kg	
49–59	(4) 11.8
60–69	(6) 17.6
70–79	(8) 23.5
80–89	(5) 11.8
90–99	(6) 20.6
100–122	(5) 14.7
Body mass index, kg/m^2	
21–24	(5) 14.7
25–29	(10) 29.4
30–47	(19) 55.9

Spearman's product-moment correlations were performed to relate changes in all experimental measures, which included body composition, changes in plasma lipids, apolipoproteins, glucose, A1c and insulin concentrations. The differences between the baseline and 3 month measurements of insulin, FPG, A1c, plasma lipids, Apo and body composition before and after diabetic education were assessed using a paired sample *t* test.

RESULTS

Table 1 summarizes the baseline descriptive characteristics of the study participants, aged 37–69 years. The weight of the study participants ranged from 49 kg to 122 kg and the BMI ranged from 21–47 kg/m^2. Out of 44 enrolled, 34 completed the study as 10 dropped out due to lack of transportation.

Table 2 summarizes the changes in FPG, A1c, insulin, cholesterol, and Apo after three months of the intervention. There were significant decreases in FPG (−23.20 mg/dL, P=.003), and A1c (−.79, P<.001) and significant increases in HDL (4.2 mg/dL, P=.002), and

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decreases in cholesterol/HDL (chol/hdlr) ratio (−.48, *P*=.001) all compared at 3 months to baseline.

Table 3 summarizes mean changes in bodyweight and body composition by DXA. There were significant reductions in body weight (−.94 kg, *P*=.015), DXA trunk fat (−1.78 kg, *P*=.001), DXA trunk fat % (−.79, *P*=.003), DXA .total fat (−.91 kg, *P*<.001), DXA total lean mass (−.03 kg, *P*<.001), and DXA total % fat (−.6, *P*=.003) observed at 3 months when compared to baseline.

Table 4 summarizes Spearman's correlation between changes (3 months-baseline) in Apo A1 and A2 (*r*=.559, *P*<.001), Apo E and total cholesterol (*r*=.746, *P*<.001), and between A1c and FPG (*r*=.563, *P*=.001).

DISCUSSION

Our program *En Balance*, utilizing a culturally sensitive education program targeting Hispanic diabetics, previously demonstrated decreases in A1c, which would translate into a significant reduction in diabetic complications if sustained.[21]

Our current study demonstrates that, in addition to improved glycemic control, we have achieved improvements in weight, fat distribution and lipoproteins that additionally may result in reduction in cardiovascular events. Earlier studies targeting the Hispanic diabetic population have demonstrated improvements in glycemic control along with weight and diabetes-related literacy.[22–24] The importance of implementing cultural sensitive programs such as *En Balance* is demonstrated in two recent studies. Rosal et al[25] developed the *Latinos en Control* program that demonstrated improved glycemic control with an A1C difference of .53% among low-income Hispanics compared to usual care. Similarly, Brown et al[26] compared dosage effects of diabetes self-management education employing extended (24 h of education, 28 h of support groups) and compressed (16 h of education, 6 h of support groups), and concluded that culturally competent diabetes education interventions were effective in promoting improved metabolic control and diabetes knowledge in both groups.

Our current study specifically evaluated the *En Balance* program on important determinants of insulin resistance such as weight and body composition and dyslipidemia as surrogate measures of subsequent vascular risk.

Weight and Body Composition

Our study participants showed a reduction in weight after three months of diabetes education consistent with previous studies.[23–24]

Total and regional fat distribution of our study participants was measured using DXA, which provides additional information about body fat distribution than waist circumference measurement alone.[27] Our study participants had significant reduction in trunk fat and total fat after three months compared to baseline trunk fat and total fat. Nelson et al,[27] in a cross-sectional study among diabetic Hispanics in San Luis valley, compared DXA to regular waist measurement; they concluded that DXA provided additional information about trunk fat than the waist circumference measurement. Further, the significant decrease in trunk fat and total fat could be attributed to weight loss as a result of change in diet (increased consumption of fibers and vegetables, less

Table 2. Paired samples *t* tests for selected variables at baseline and three months (both sexes, *N*=34)

Variables	Baseline Mean (SD)	3 Months Mean (SD)	Mean Difference[a]	95% CI	*P*
A1c, %	7.87 (2.0)	7.08 (1.6)	−.79	.43, 1.16	<.001[b]
FPG, mg/dL	166.41 (65.9)	143.21 (57.8)	−23.2	8.43, 37.99	.003[b]
Insulin, μg/L	12.89 (9.6)	12.55 (12.4)	−.34	−1.84, 2.53	.753
Total cholesterol, mg/dL	190.00 (44.18)	185.00 (49.57)	5.0	−6.81, 17.99	.366
HDL cholesterol, mg/dL	44.65 (8.8)	48.85 (11.4)	4.2	−6.80, −1.62	.002[b]
LDL cholesterol, mg/dL	123.38 (38.27)	118.41 (37.64)	−4.97	−4.62, 14.57	.300
Cholesterol/HDL ratio	4.35 (1.0)	3.87 (.98)	−.48	.20, .77	.001[b]
Triglycerides, mg/dL	217.41 (131.02)	193.97 (128.59)	−23.44	−9.56, 56.44	.158
Apo A1, mg/L	817.25 (144.48)	831.11 (120.23)	13.86	−60.66, 32.94	.551
Apo A2, mg/L	190.18 (48.92)	194.60 (56.22)	4.42	−25.41, 16.56	.671
Apo C2, mg/L	437.95 (20.22)	509.74 (55.62)	71.79	−25.72, 11.36	.437
Apo C3, mg/L	129.72 (60.57)	128.87 (61.06)	−.85	−13.74, 15.45	.906
Apo E, mg/L,	376.47 (13.72)	412.55 (21.38)	36.08	−10.60, 3.39	.302

[a] Baseline-3 months.
[b] *P*<.005.

Table 3. Changes in body weight/composition after 3-months of Spanish diabetes education. (both sexes, *N*=34)

Variables	Baseline Mean (SD)	3 Months Mean (SD)	Mean Difference[a]	95% CI	*P*
Body weight, kg	81.25 (17.90)	80.31 (17.97)	.94	.20, 1.68	.015[b]
BMI, kg/m^2	32.17 (7.0)	31.72 (6.7)	−.45	−.14, 1.04	.132
DXA trunk fat, kg	16.87 (5.4)	15.09 (5.6)	−1.78	.27, .95	.001[b]
DXA trunk fat, %	37.57 (7.1)	36.78 (7.1)	−.79	.28, 1.31	.003[b]
DXA total fat, kg	30.41 (10.68)	29.50 (10.52)	−.91	.43, 1.38	.000[b]
DXA total lean mass, kg	4.98 (10.65)	4.95 (10.71)	−.03	−.21, .66	.000[b]
DXA total fat, %	36.37 (7.49)	35.77 (7.52)	−.6	.22, .97	.003[b]

[a] Baseline-3 months.
[b] $P<.01$.

consumption of foods with high glycemic index) and increase in physical activities. This is in agreement with a study by Goodpaster et al[28] in the study of effects of weight loss on regional fat distribution and insulin sensitivity in obesity in 32 obese sedentary women and men over a 4-month weight loss program.

Further, Hairston et al,[29,30] in a study of Hispanic and African Americans over a five year period, reported that lifestyle interventions such as physical activities and high consumption of fiber were associated with a decrease in abdominal visceral fat. The study, though spanning a five year period, (compared to our study of three months) does highlight the importance of physical activities and consumption of healthy diet in mitigating the effects of insulin resistance due to abdominal visceral and consequently improving insulin sensitivity.

Dyslipidemia

Our study is unique in that it evaluated the effects of the *En Balance* program on changes in lipids and lipoproteins.

There was an overall improvement in HDL and Apo A1 at the three month

Table 4. Spearman's correlation between changes (three months-baseline) in apolipoproteins and body compositions (both sexes, *N*=34), *P* value is in parentheses

	Apo A2	Apo C2	Apo C3	Apo E	Insulin	A1c %	FPG mg/dL	tchol	HDLc	LDLc	Chol/hdlr	BMI	BWT
Apo A1	.559[b]	.216	.506[b]	.392[a]	.105	.138	−.113	.091	.289	.004	−.009	.016	−.042
	(.001)	(.219)	(.022)	(.022)	(.553)	(.438)	(.524)	(.610)	(.098)	(.981)	(.958)	(.929)	(.811)
Apo A2	1.000	.449[b]	.523[b]	.523[b]	−.130	.055	.022	.105	.236	.113	.020	−.039	−.123
		(.008)	(.001)	(.002)	(.462)	(.757)	(.902)	(.555)	(.180)	(.524)	(.911)	(.829)	(.489)
Apo C2		1.000	.683[b]	.632[b]	.268	.068	−.319	.530[b]	.086	.344[a]	.563[b]	.045	−.073
			(<.001)	(<.001)	(.125)	(.701)	(.066)	(.001)	(.630)	(.046)	(.001)	(.801)	(.683)
Apo C3			1.000	.760[b]	−.177	.405[a]	.023	.635[b]	.233	.366[a]	.487[b]	.057	−.009
				(<.001)	(.318)	(.017)	(.896)	(<.001)	(.185)	(.033)	(.004)	(.749)	(.960)
Apo E				1.000	.103	.313	.054	.746[b]	.151	.608[b]	.638[b]	.079	.046
					(.561)	(.072)	(.763)	(<.001)	(.395)	(<.001)	(<.001)	(.658)	(.796)
Insulin					1.000	−.010	.041	−.198	−.126	−.285	−.067	−.026	.055
						(.956)	(.817)	(.262)	(.479)	(.103)	(.707)	(.883)	(.756)
A1c %						1.000	.563[b]	.391[a]	.241	.370[a]	.196	.253	.334
							(.001)	(.022)	(.170)	(.031)	(.266)	(.149)	.054
FPG							1.000	.054	.174	.244	−.124	.248	.217
								(.760)	(.324)	(.165)	(.484)	(.158)	(.218)
tchol								1.000	.189	.847[b]	.833[b]	.043	.079
									(.284)	(<.001)	(<.001)	(.811)	.659
HDLc									1.000	.249	−.257	.039	−.088
										(.155)	(.143)	(.827)	(.619)
LDLc										1.000	.674[b]	.103	.089
											(<.001)	(.562)	(.619)
Chol/hdlr											1.000	.054	.077
												(.762)	(.663)
BMI												1.000	.732[b]
													(<.001)

[a] $P<.05$.
[b] $P<.005$.

point of the study; HDL is rich in Apo A2[31] and there is evidence to suggest that Apo A2 is a significant predictor of HDL levels.[32] We found a nonsignificant increase in the mean of Apo A2 at three months compared to baseline. This may have also contributed to the increase in HDL observed in the study participants. Weng et al[33] reported that in an experimental study on Apo A2 knockout mice, a significant reduction in HDLc was observed, further supporting that Apo A2 is abundant in HDL.

A nonsignificant reduction in the mean of Apo C3 was observed in all study participants, which is associated with reductions in triglycerides and may contribute to mortality and morbidity seen in diabetics and is in agreement with Florez et al.[34]

Levels of Apo C2 increased at three months when compared to baseline; Apo C2 is responsible for the activation of lipoprotein lipase in peripheral tissues, particularly adipocytes and muscles. The presence of high TG levels diminishes the activity of Apo C2.[35] Our study participants had high TG levels at the beginning of the study that decreased after three of months of study, thus the increase in Apo C2 observed after three months of study was possibly the result of reduction in TG.

Apolipoprotein E is responsible for clearance of triglyceride-rich containing particles; Apo E increased after three months of study which may be explained by the decrease in cholesterol levels.[36] Also, Apo E is a component of very low density lipoprotein, HDL and intermediate low density lipoprotein, which affects cholesterol level.[37] It is also possible that the increase in Apo E levels could be attributed to the increase in its HDL portion. We are not aware of any studies that have evaluated the impact of diabetes education in reducing Apo E levels in Hispanic diabetics.

Limitations

There are limitations to our study, the most important of which is the short term nature of our current design. Despite this major limitation, the surrogate measures that we determined are directly related to important clinical outcomes and if sustained would yield improved quality of life, reduction in overall health costs and possibly lowered mortality in diabetic patients. The lack of a contemporary control group also limits the conclusions. However, historical evidence suggests *En Balance* would result in improved outcomes compared to usual care. The experimental participants lacked most of the basic structured lifestyle that supports consistent health care. Hence, success within this group suggests that *En Balance* would translate into even greater benefits in patients with more structured and consistent resources.

Lastly, the specific benefits seen in improvement of the lipoproteins are not identified and may not directly relate to the interventions in *En Balance*. Further studies will need to identity elements within the program that specifically result in improvement in diabetic dyslipidemia.

CONCLUSIONS

Overall, the findings from our study demonstrate the effectiveness and the importance of lifestyle intervention in improving glycemic control in diabetics and increasing the level of healthy Apo A1, A2 and C2 while decreasing Apo E and C3. These improvements in health outcomes could be attributed to culturally competent and sensitive Spanish health educators. It could also be attributed to the effectiveness and frequency at which the health education classes were conducted and the proximity of the homes of the study participants to the venue. There are clearly social and health service delivery issues that are important; study participants complained about the cost of healthy foods that were necessary for improving dietary habits and glycemic control. In addition, most of the study participants had no health insurance thus limiting access to health care providers. As a result, most of the study participants sought medical attention only when symptoms were very serious. Some study participants were semiliterate with long standing diabetes and this may have affected the slow response to change from old dietary and lifestyle habits to better lifestyle interventions. Finally, it is important that socioeconomic factors existing in a community be taken into consideration when designing effective interventions to improve health outcomes in resource poor populations.

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REFERENCES

1. LeRoith D, Taylor S, Olefsky J. *Diabetes Mellitus. A Fundamental and Clinical Text* Lippincott Williams & Wilkins; 2003.
2. World Health Organization. *Diabetes*; 2009. http://www.who.int/mediacentre/factsheets/fs312/en/index.html. Accessed January 2011.
3. Engelgau MM, Geiss LS, Saaddine JB, et al. The evolving diabetes burden in the United States. *Ann Intern Med*. 2004;140:945–950.
4. Fowler MJ. Diabetes: Magnitude and Mechanisms. *Clin Diabetes*. 2007;25:25–28.
5. U.S. Department of Health and Human Services. *National Diabetes Fact Sheet: General Information and National Estimates on Diabetes in the United States, 2005*. 2005:1–10.
6. Center for Disease and Prevention Control. *General information and National Estimates on Diabetes in the United States. Centers for Disease Control and Prevention*; 2008. http://www.cdc.gov/diabetes/pubs/pdf/ndfs_2007.pdf. Accessed January 2011.
7. He G, Black K, Lopez-Payan S, Omark K, Schillinger D. Diabetes in California Counties. 2009. Available at: http://www.caldiabetes.org. Accessed December 2010.
8. Mc Bean A, Li S, Gilbertson D, Collins A. Differences in diabetes prevalence, incidence, and mortality among the elderly of four racial/ethnic groups: Whites, Blacks, Hispanics, and Asians. *Diabetes Care*. 2004;27:2317–2324.
9. American Association of Clinical Endocrinologist. Medical guidelines for clinical practice for management of diabetes mellitus. Medical guidelines for clinical practice for the management

of diabetes mellitus. *Endocr Pract*. 2007;13: 7–8.
10. Agency for Healthcare Research and Quality National Healthcare Disparities Report: Summary, February 2004. http://www.ahrq.gov/qual/nhdr03/nhdrsum03.htm. Accessed February 2011.
11. Williams DR, Neighbors HW, Jackson JS. Racial/ethnic discrimination and health: findings from community studies. *Am J Public Health*. 2003;93:200–208.
12. Hajat A, Lucas JB, Kington R. Health outcomes among Hispanic subgroups: data from the national health interview survey, 1992–1995. *Adv Data Vital Health Stat*. 2000;310:1–14.
13. Carter-Pokras O, Zambrana RE. Latino health status. In: Aguirre-Molina M, Molina CW, Zambrana RE, eds. *Health Issues in the Latino Community*. San Francisco: Jossey-Bass; 2001: 23–54.
14. American Diabetes Association. Management of dyslipidemia in adults with diabetes. *Diabetes Care*. 2003;26:s83–86.
15. Despres JP, Lemieux I. Abdominal obesity and metabolic syndrome. *Nature*. 2006;444(7121): 881–887.
16. Cruz ML, Bergman RN, Goran MI. Unique effect of visceral fat on insulin sensitivity in obese Hispanic children with a family history of type 2 diabetes. *Diabetes Care*. 2002;25(9): 1631–1636.
17. Going SB, Massett MP, Hall MC, et al. Detection of small changes in body-composition by dual-energy x-ray absorptiometry. *Am J Clin Nutr*. 1993;57:845–850.
18. Relijic R, Ries M, Anic N, Ries B. New chromogen for assay of glucose in serum. *Clin Chem*. 1992;38:522–25.
19. Rifai N, Warnick GR, Dominiczak MH. *Handbook of Lipoprotein Testing*. 2nd ed. Washington, DC: AACC Press; 2000:630.
20. Lohman TG, Roche AF, Martorell R. *Anthropometric Standardization Reference Manual*. Champaign, Ill: Human Kinetics; 1991.
21. American Diabetes Association. Implications of the Diabetes Control and Complications Trial. *Diabetes Care*. 2003;26:25–27.
22. Corkery E, Schechter CB, Palmer C, Frisher L, Foley ME, Roman SH. Effect of a bicultural community health worker on completion of diabetes education in a Hispanic population. *Diabetes Care*. 1997;20:254–257.
23. Vero L, Wien M, Byham-Gray L, Touger-Decker R, Maillet J. Effect of individual versus group diabetes outpatient education on weight change, hemoglobin a1c and lipid values in adults with type 2 diabetes mellitus. *J Am Diet. Assoc*. 2007;107(40).
24. Jimenez-Cruz A, Bacardi-Gascon M, Rosales-Garay P, Herrera-Espinoza J, Willis O. A culturally sensitive tool for Mexican people with diabetes: ''La Manzana de la Salud''. *Rev Biomed*. 2003;14:51–59.
25. Rosal MC, Ockene IS, Restrepo A, et al. Randomized trial of a literacy-sensitive, culturally tailored diabetes self-management intervention for low-income Latinos: Latinos en control. *Diabetes Care*. 2011;34(4):838–844.
26. Brown SA, Blozis SA, Kouzekanani K, Garcia AA, Winchell M, Hanis CL. Dosage effects of diabetes self-management education for Mexican Americans: the Starr County Border Health Initiative. *Diabetes Care*. 2005;28(3): 527–532.
27. Nelson TL, Bessesen DH, Marshall JA. Relationship of abdominal obesity measured by DXA and waist circumference with insulin sensitivity in Hispanic and non-Hispanic White individuals: The San Luis Valley Diabetes Study. *Diabetes-Metabolism Research and Reviews*. 2008;24:33–40.
28. Goodpaster BH, Kelley DE, Wing RR, Meier A, Thaete FL. Effects of weight loss on regional fat distribution and insulin sensitivity in obesity. *Diabetes*. 1999;48(4):839–847.
29. Hairston KG, Scherzinger A, Foy C, et al. Five-year change in visceral adipose tissue quantity in a minority cohort: the Insulin Resistance Atherosclerosis Study (IRAS) family study. *Diabetes Care*. 2009;32(8):1553–1555.
30. Hairston KG, Vitolins MZ, Norris JM, Anderson AM, Hanley AJ, Wagenknecht LE. Lifestyle Factors and 5-Year Abdominal Fat Accumulation in a Minority Cohort: The IRAS Family Study. Obesity (Silver Spring). 2011;6.
31. Rader DJ. Molecular regulation of HDL metabolism and function: implications for novel therapies. *J Clin Invest*. 2006;116: 3090–3100.
32. Allayee H, Castellani LW, Cantor RM, de Bruin TWA, Lusis AJ. Biochemical and genetic association of plasma apolipoprotein A-II levels with familial combined hyperlipidemia. *Circ Research*. 2003;92:1262–1267.
33. Weng W, Brandenburg NA, Zhong SB, et al. ApoA-II maintains HDL levels in part by inhibition of hepatic lipase: studies in apoA-II and hepatic lipase double knockout mice. *J Lipid Research*. 1999;40:1064–1070.
34. Florez H, Mendez A, Casanova-Romero P, et al. Increased apolipoprotein C-III levels associated with insulin resistance contribute to dyslipidemia in normoglycemic and diabetic subjects from a triethnic population. *Atheroscl*. 2006;188:134–141.
35. Gerber Y, Goldbourt U, Cohen H, Harats D. Association between serum apolipoprotein C-II concentration and coronary heart disease. *Prev Med*. 2002;35:42–47.
36. Relly M RD. Apolipoprotein E and coronary heart disease: a puzzling paradox. *Plos Medicine*. 2006;3:0736–0738.
37. Zimmerman J, Kaufmann N, Fainaru M, et al. Effect of weight loss in moderate obesity on plasma lipoprotein and apolipoprotein levels and on high density lipoprotein composition. *Arterioscler Thromb Vasc Biol*. 1984;4: 115–123.

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