Disclosing Genetic Risk for Coronary Heart Disease: Effects on Perceived Personal Control and Genetic Counseling Satisfaction

Christopher L. Robinson,[a] Hayan Jouni, M.D.,[b] Teresa M. Kruisselbrink, C.G.C.,[b] Erin E. Austin, Ph.D.,[b] Kurt D. Christensen, Ph.D.,[c] Robert C. Green, M.D., MPH.,[c] Iftikhar J. Kullo, M.D.[b]

[a]School of Medicine, Saint Louis University, St. Louis, MO

[b]Division of Cardiovascular Diseases, Department of Medicine, Mayo Clinic, Rochester, MN

[c]Division of Genetics, Department of Medicine, Brigham and Women's Hospital and Harvard Medical School, Boston, MA

Corresponding author: Iftikhar J. Kullo, M.D.

Division of Cardiovascular Diseases

Mayo Clinic

200 First Street SW

Rochester, MN 55905

FAX: (507) 266-1617; TEL: (507) 266-3964

Email: Kullo.Iftikhar@mayo.edu

Conflict of interest: None of the authors have any relationships to disclose.

Acknowledgments: This study was funded as part of the National Human Genome Research Institute-supported eMERGE (Electronic Medical Records and Genomics) Network grants to the Mayo Clinic (U01HG04599 and U01HG006379). C.L.R. was supported by grant R25 HL92621 from the National Institutes of Health. K.D.C. and R.C.G were supported by HG002213, HG005092, HG006500, HG006993, and HR077671.

ClinicalTrials.gov registration number: NCT01936675

This article has been accepted for publication and undergone full peer review but has not been through the copyediting, typesetting, pagination and proofreading process, which may lead to differences between this version and the Version of Record. Please cite this article as doi: 10.1111/cge.12577

ABSTRACT

We investigated whether disclosure of coronary heart disease (CHD) genetic risk influences perceived personal control (PPC) and genetic counseling satisfaction (GCS). Participants (n=207, age: 40-65 years) were randomized to receive estimated 10-y-risk of CHD based on a conventional risk score (CRS) with or without a genetic risk score (GRS). Risk estimates were disclosed by a genetic counselor who also reviewed how GRS altered risk in those randomized to CRS+GRS. Each participant subsequently met with a physician and then completed surveys to assess PPC and GCS. Participants who received CRS+GRS had higher PPC than those who received CRS alone although the absolute difference was small (25.2±2.7 vs. 24.1±3.8, *P*=0.04). A greater proportion of CRS+GRS participants had higher GCS scores (17.3±5.3 vs. 15.9±6.3, *P*=0.06). In the CRS+GRS group, PPC and GCS scores were not correlated with GRS. Within both groups, PPC and GCS scores were similar in patients with or without family history (*P*=NS). In conclusion, patients who received their genetic risk of CHD had higher PPC and tended to have higher GCS. Our findings suggest that disclosure of genetic risk of CHD together with conventional risk estimates is appreciated by patients. Whether this results in improved outcomes needs additional investigation.

Key Words: coronary heart disease; clinical trial; clinical genetics; genetic counseling; genetic risk score; perceived personal control

INTRODUCTION

As we learn more about genetic risk for human diseases, understanding how people respond to such information will be crucial to effectively translate genetic discoveries into clinical care. There is some concern that disclosing genetic risk for complex diseases might induce feelings of fatalism (the idea that outcomes have already been decided and cannot be changed), or induce feelings of invulnerability (1, 2). Recent studies, however, have found that patients receiving genetic risk results for disparate conditions such as obesity, diabetes, depression, Alzheimer's disease, and breast cancer (1-5) did not interpret results in an overly deterministic manner that would indicate fatalism or

invulnerability. However, notable gaps in this literature are studies that focus on disclosure of genetic risk for coronary heart disease (CHD).

Multiple susceptibility variants for CHD have been identified, but the utility of genetic risk scores based on such variants is unclear. For example, it is not known whether disclosure of CHD genetic risk affects perceived personal control (PPC) which represents the belief that a person can alter his/her own situation or state by bringing about desirable change and captures three dimensions (6, 7): behavioral control, cognitive control, and decisional control. Greater PPC is associated with higher satisfaction, knowledge and self-efficacy, all of which are central to coping with health threats that may call for behavior change (8-10).

Genetic risk is often disclosed by genetic counselors and satisfaction in genetic counseling is used as an outcome measure for evaluating the quality of counseling sessions. One study found that patient satisfaction was positively associated with PPC, (9) suggesting that enhancing genetic counseling sessions may also result in higher patient satisfaction and greater patient PPC. Higher genetic counseling satisfaction scores (GCS) may be associated with greater control beliefs among patients and better clinical outcomes.

We investigated whether disclosure of genetic CHD risk influenced PPC and GCS as secondary outcomes of the Myocardial Infarction Genes (MI-GENES) study, which is exploring the clinical utility of incorporating CHD genetic risk into conventional risk prediction algorithms in adults at intermediate risk for CHD. We also investigated whether the GRS was correlated with PPC and GCS scores in the group randomized to disclosure of genetic CHD risk, and whether PPC and GCS scores were influenced by the presence of family history.

METHODS

Study Design

The MI-GENES study, approved by the Mayo Clinic IRB, is a randomized controlled trial comparing the outcomes of patients who are presented their CHD risk conventionally estimated, and patients whose CHD risk estimates include genetic risk information. The primary outcome is the change in low-density lipoprotein cholesterol (LDL-C) levels between the study arms 3 and 6 months after disclosure of 10-year CHD risk. Secondary outcome measures include changes in dietary fat

consumption and physical activity. At the first visit, height, weight, systolic BP, lipid levels, smoking status, medical history, family history of CHD, and current medications were assessed.

The 10-year CHD risk was estimated using a conventional risk score (CRS) based on the Framingham risk equation that includes conventional risk factors including age, sex, diabetes, smoking, BP, total cholesterol and HDL-C (11). A genetic risk score (GRS) was calculated based on genotypes at 28 SNPs that are associated with CHD independent of BP and lipids levels, as previously described (12). Thus a GRS of 0.8 indicates a 20% lower CHD genetic risk compared to the population average, while a GRS of 1.4 indicates a 40% higher risk compared to the population average. In those randomized to receive genetic risk information, the 10-year CHD risk was estimated by multiplying CRS by the GRS (CRS+GRS).

At the second visit, participants were randomized to receive either CRS or CRS+GRS, CHD risk being disclosed in each arm by a genetic counselor during a 30-min scripted session. This session included a discussion of recommended lifestyle modifications to decrease risk of CHD as well as the impact of family history on CHD risk. Specifically, a study participant with a family history for CHD was told that this could increase their risk 1.5-2 fold. For CRS+GRS participants, the genetic counselor also reviewed their GRS and how it was integrated into their conventional risk score. Each participant then met with a physician to engage in shared-decision making regarding the need for statin therapy. At the end of this session, study participants were asked to complete validated surveys assessing PPC and GCS.

Genetic counselor/physician scripts, slides, and template risk reports that were used during this visit are included in the supplementary material. Fidelity of the scripts was assessed by analysis of video-recorded encounters. Having one genetic counselor (T.M.K) disclose CHD risk estimates to all study participants helped ensure that risk was disclosed similarly to all study participants (in their respective randomization groups). Risk was disclosed using a decision aid that has the capability to include GRS into CRS. This decision aid can be found at: http://migenesstudy.mayoclinic.org/ (password: migenes – use is limited to research purposes) (13-15).

Study Population

We screened 29,352 Mayo Clinic Biobank participants for the following eligibility criteria: ability to provide informed consent, resident of Olmsted county, MN, 45-65 years of age, no history of CHD or other atherosclerotic vascular diseases, not on statins, and at intermediate (5-20%) 10 year risk for CHD. Of the 2026 individuals who met the above criteria, a random subset of 1000 was genotyped to calculate a GRS for each individual. After quality control, genotyping results were available for 968 individuals. Subsequently, recruitment was targeted to enroll approximately 100 individuals with a high GRS (≥ 1.1), and 100 individuals with low/average GRS (<1.1). Randomization was performed in a 1:1 fashion by means of a computer-generated sequence that controlled for participant age, sex, and family history for CHD using validated methods (16).

Perceived Personal Control

For the validated 9-item PPC questionnaire, (6, 7, 9) response options were scored "1" for "do not agree", "2" for "somewhat agree", and "3" for "completely agree". The sum of all scale responses yielded a PPC score that ranged from 9-27, with higher scores indicating greater control beliefs. Additionally, we analyzed cognitive, behavioral and decisional control components as sub-scales of PPC. The PPC questionnaire had high internal consistency (α=0.892).

Genetic Counseling Satisfaction

The validated 5-item GCS questionnaire (2, 17) was scored per item with "0" for "strongly disagree", "1" for "disagree somewhat", "2" for "uncertain", "3" for "agree somewhat", and "4" for "agree strongly". The sum for all responses yielded scores that ranged from 0-20, with higher scores indicating greater satisfaction. Hierarchical cluster analysis showed two distinct groups of responders separated by a cutoff level of 15. Thus, we analyzed GCS scores both as a continuous trait and also as a dichotomous trait using a cutoff level of 15. Internal consistency of the GCS questionnaire was high (α=0.975).

Statistical Methods

All statistical analyses were performed using the program R version 2.14.1 (Foundation for Statistical Computing, Vienna, Austria). Continuous variables were expressed as mean ± SD, and dichotomous variables were expressed as percentages. The internal consistency of questionnaires was

tested using Cronbach's alpha test. For analysis of the ordinal variables, we used nonparametric Wilcoxon rank sum test with χ^2 approximation. Also, we performed ordinal logistic regression to estimate the effect of randomization and family history on the individual item responses to PPC and GCS questions using odds ratios (OR). To test for differences in the dichotomous characterization of GCS as high or lower satisfaction, we used the Fisher's exact test. We performed a Kruskal-Wallis test to compare PPC and GCS scores among the three different GRS groups. All *P*-values reported are for 2-sided tests.

RESULTS

Of 216 participants enrolled, 207 participants completed both the first and second visits. Of these 207 participants, 103 were randomized to the CRS arm, and 104 to the CRS+GRS arm. Baseline characteristics of the study participants are shown in Table 1.

Patients randomized to receive CRS+GRS had higher mean PPC scores than those randomized to CRS although the absolute difference was modest (25.24±2.65 vs. 24.12±3.83, *P*=0.04). When assessing responses to the sub-scale components of PPC (Table 2), the cognitive control component was found to be higher in patients who received CRS+GRS (*P*=0.015). However, there was no significant difference for both behavioral and decisional control components between the two arms of the study (*P*=0.304 and *P*=0.108 respectively). Assessment of responses to individual items (Table 2) in the PPC questionnaire found three of the nine items to be higher among the CRS+GRS arm than the CRS arm: (1) "I think I understand what problem brought me to genetic counseling" (2.79±0.46 vs. 2.64±0.56, *P*=0.03) (2) "I think I know what caused the problem" (2.75±0.50 vs. 2.57±0.63, *P*=0.03) (3) "I feel I can make a logical evaluation of the various options available to me in order to choose one of them" (2.92±0.30 vs. 2.83±0.40, *P*=0.047). Based on ordinal logistic regression analyses two of the nine items had significantly different responses between the two study arms. Specifically, CRS+GRS participants were approximately twice as likely to be at ordinal levels that would indicate increased PPC compared to CRS for both "I think I understand what problem brought me to genetic counseling" (OR 2.01, *P*=0.03), and "I think I know what caused the problem" (OR=2.00, *P*=0.03) (Table 2).

Participants randomized to receive CRS+GRS had higher GCS scores than CRS patients but this was not statistically significant (17.27±5.27 vs. 15.93±6.34, *P*=0.064). When assessing individual item responses (Table 3), participants in the CRS+GRS arm had higher responses to "The genetic counseling session was valuable to me" (3.50±1.09 vs. 3.11±1.33, *P*=0.01). Similarly, for item 5, CRS+GRS participants were twice as likely to be at ordinal levels that would indicate greater satisfaction than participants in the CRS arm for Item 5 (OR=2.13, *P*=0.01) (Table 3). When we stratified GCS scores into "high" or "lower" satisfaction, the CRS+GRS participants were more often highly satisfied than the CRS participants [n=93 (90.29%) vs. n= 79 (78.22%), *P*=0.02].

Additionally, we tested whether there were any differences in responses across the GRS categories, or in groups defined by presence or absence of family history. PPC was not correlated to GRS (r=0.15, *P*=0.12) nor was GCS (r= -0.003, *P*=0.97). Furthermore, we found no difference in PPC scores (*P*=0.86) or GCS scores (*P*=0.95) between patients with low, average or high GRS in the CRS+GRS group. Similarly, we observed no differences within the CRS arm or the CRS+GRS arms in PPC scores (*P*=0.22 and *P*=0.47 respectively) or GCS scores (*P*=0.45 and *P*=0.77, respectively) between patients with or without family history of CHD. Among all patients in the study, PPC scores were weakly correlated with GCS scores (r=0.16, *P*=0.02).

DISCUSSION

Patients who received CHD genetic risk combined with their conventionally estimated risk had higher perceived personal control and satisfaction with genetic counseling compared to those who received conventional risk estimates alone. Within the group randomized to receive GRS, there was no correlation between GRS and either PPC or GCS. Additionally, PPC and GCS scores did not differ among patients with/without family history. Our findings suggest that disclosure of CHD risk by a genetic counselor may help patients avoid potentially harmful interpretations of genetic risk (2), by counseling patients regarding the correct interpretation thereof.

PPC measures the belief that a person can alter his/her own situation or state by bringing about desirable change. Higher levels of PPC are associated with an increased Health-Related Quality of Life (HRQL), a measure used by the CDC to assess health standards in individuals and communities (18-20). PPC is also likely an indicator of core self-evaluation (CSE), as it reflects self-

efficacy and locus of control beliefs (21). Individuals with higher levels of CSE report higher satisfaction, coping ability, and self-helping behavioral changes (22-26). Control perceptions are fundamental for behavioral changes because interventions that target control beliefs help motivate patients to be healthier thereby improving clinical outcomes. Since higher PPC suggests a greater likelihood of behavior change, it follows that disclosing risk estimates of CHD based on both genetic and conventional risk factors may be a more effective intervention than disclosing estimates based on conventional risk factors alone.

Higher PPC in CRS+GRS participants may also predict better behavioral outcomes that then translate into improved clinical outcomes, but this requires additional investigation. In several studies, (27-29) patients who received genetic risk information, in addition to conventional behavioral risk factors, had higher levels of intention to adapt positive health behaviors that reduce risk. When presented with genetic risk, patients show higher levels of self-reported adaptation of positive health behaviors that reduce their risk, (28, 30) even if, in the case of Alzheimer's disease, the effectiveness of those behavior changes are uncertain (31). It could be that presenting a clearer genetic component of risk provides just enough "cue-to-action" to move patients into subsequent stages of behavior change to induce the "tipping point" or "mini-epiphany" needed for healthy behavior change (32). Since the cognitive control component of PPC was the sub-scale that was significantly increased in the participants who received CRS+GRS, perhaps the perceptions of genetic testing as more accurate than conventional risk assessment may reduce ambiguity in risk interpretation, making patients perceive their susceptibility more accurately. Whatever the case, since the CRS+GRS group had higher levels of PPC we would expect these participants to have a higher health-related quality of life and greater rates of behavior change leading to better clinical outcomes as other studies have indicated (18-20). These outcomes, including changes in LDL-C, dietary fat consumption, and physical activity will be reported separately.

GCS scores were also higher in the CRS+GRS arm although this was only of borderline statistical significance. Furthermore, participants who received their GRS in addition to CRS responded that the disclosure session was more valuable to them. It could be that patients who received genetic risk information were more satisfied simply for receiving more information. (33).

Moreover, it is unlikely that that the higher PPC seen in the CRS+GRS arm produced a more satisfied cohort since we did not find PPC to be correlated with GCS. Since GCS was not correlated to GRS, GRS sub-groups, or to PPC, it suggests that these increases in GCS may be due to receiving more information in the risk disclosure session. These results suggest that a care provider disclosing risk should be equipped with genetic, behavioral and clinical risk information to increase counseling satisfaction and control beliefs.

Since PPC and GCS did not differ between GRS categories regardless of family history, it is likely that the differences we found in the two study groups simply reflect the effect of disclosing GRS for CHD to patients. Duffy et al. (34) argue that how patients are counseled influences internal motivation and self-efficacy; specifically noting that motivational counseling improves satisfaction of patients and creates meaningful relationships whereby physicians can more effectively advocate for positive behavioral changes. Moreover, it is important to note that physicians can be trained to use these motivational counseling strategies (35). Some have expressed concern that disclosing genetic risk for complex diseases might induce feelings of fatalism (the idea that outcomes have already been decided and cannot be changed), or on the other hand induce feelings of invulnerability (1, 2). However, other studies in the context of obesity, diabetes, depression and breast cancer have found that patients did not interpret results in an overly deterministic manner that would indicate fatalism or invulnerability (1-3). Likewise we speculate that counseling patients, especially those at extremes for genetic risk scores, regarding the correct interpretation of their GRS helps avoid feelings of fatalism and invulnerability. Additional analysis will be needed to explore the effects of these more extreme GRS scores on changes of primary and secondary outcomes compared to intermediate genetic risk patients.

Moreover, other studies of risk disclosure (36, 37) suggest that by targeting knowledge deficits, relaying the correct interpretation of risk tools, and comparative judgments of risk may help patients avoid harmful perceptions of their risk that could lead to decreased usage of available preventative resources. Specifically, by addressing gaps in patient knowledge of CHD risk, conveying the correct context of the GRS, and using a shared decision-making process that focuses on lifestyle modifications and statin therapy we hope to avoid these incorrect interpretations of risk. Thus, the

incorporation of genetic risk into preventive cardiology and clinical genetics practices could be a logical tool to increase patient satisfaction, and inspire changes in health-related behaviors. Bloss et al. (38) found that when patients received direct-to-consumer genetic risk profiles, they were unlikely to report making any significant changes in their dietary fat or exercise unless they discussed their results with a physician. Given the current shortage of genetic counselors, it is important to explore whether genetic risk information for complex diseases can be effectively disclosed by physicians untrained in genetics or by other care providers.

Study strengths and limitations

Strengths of our study include the assessment of perceived personal control and genetic counseling satisfaction following disclosure of genetic risk for CHD in a randomized clinical trial using a community-based sample with inclusion of participants with varying categories of GRS (high, average and low). A limitation is that the findings are generalizable to only individuals of European ancestry. Also, we did not assess baseline PPC before CHD risk disclosure. Additionally, our study participants may represent early adopters who are well-educated and from a higher socioeconomic status. This study is still ongoing, and the primary and secondary outcomes of changes in LDL-C, dietary fat consumption, and physical activity following CHD risk disclosure, will be reported in the near future.

CONCLUSION

Disclosing CHD genetic risk alongside conventional risk was associated with significantly higher perceived personal control and a greater proportion of patients were "highly satisfied" with genetic counseling compared to conventional risk disclosure alone. PPC and GCS scores did not differ based on GRS category or the presence or absence of family history. These findings suggest that disclosure of CHD genetic risk is appreciated by patients. Whether higher PPC and GCS lead to favorable changes in health-related measures will require further investigation; however, these results provide promising early data about the potential of genetic risk disclosure to empower CHD prevention.

REFERENCES

1. Collins RE, Wright AJ, Marteau TM. Impact of communicating personalized genetic risk information on perceived control over the risk: a systematic review. Genetics in medicine: official journal of the American College of Medical Genetics 2011: 13: 273-277.
2. Waxler JL, O'Brien KE, Delahanty LM et al. Genetic counseling as a tool for type 2 diabetes prevention: a genetic counseling framework for common polygenetic disorders. Journal of genetic counseling 2012: 21: 684-691.
3. Kaphingst KA, McBride CM, Wade C et al. Patients' understanding of and responses to multiplex genetic susceptibility test results. Genetics in medicine: official journal of the American College of Medical Genetics 2012: 14: 681-687.
4. Green RC, Roberts JS, Cupples LA et al. Disclosure of APOE genotype for risk of Alzheimer's disease. N Engl J Med 2009: 361: 245-254.
5. Grant RW, O'Brien KE, Waxler JL et al. Personalized genetic risk counseling to motivate diabetes prevention: a randomized trial. Diabetes Care 2013: 36: 13-19.
6. McAllister M, Wood AM, Dunn G et al. The perceived personal control (PPC) questionnaire: reliability and validity in a sample from the United Kingdom. American journal of medical genetics Part A 2012: 158A: 367-372.
7. Smets EM, Pieterse AH, Aalfs CM et al. The perceived personal control (PPC) questionnaire as an outcome of genetic counseling: reliability and validity of the instrument. American journal of medical genetics Part A 2006: 140: 843-850.
8. Lipinski SE, Lipinski MJ, Biesecker LG et al. Uncertainty and perceived personal control among parents of children with rare chromosome conditions: the role of genetic counseling. American journal of medical genetics Part C, Seminars in medical genetics 2006: 142C: 232-240.
9. Berkenstadt M, Shiloh S, Barkai G et al. Perceived personal control (PPC): a new concept in measuring outcome of genetic counseling. American journal of medical genetics 1999: 82: 53-59.
10. Aalfs CM, Oort FJ, de Haes JC et al. A comparison of counselee and counselor satisfaction in reproductive genetic counseling. Clinical genetics 2007: 72: 74-82.
11. Wilson PW, D'Agostino RB, Levy D et al. Prediction of coronary heart disease using risk factor categories. Circulation 1998: 97: 1837-1847.
12. Ding K, Bailey KR, Kullo IJ. Genotype-informed estimation of risk of coronary heart disease based on genome-wide association data linked to the electronic medical record. BMC cardiovascular disorders 2011: 11: 66.
13. Jouni H, Haddad RA, Marroush TS et al. Shared Decision Making Following Disclosure of Coronary Heart Disease Genetic Risk: A Randomized Clinical Trial. JAHA 2015: (under review).
14. Shameer K, Jouni H, Chaudhry R et al. A Genomic Decision Aid Linked to the Electronic Health Record to Disclose Coronary Heart Disease Risk and Enable Shared Decision-Making. 2014: Presented at the American Society of Human Genetics 64th Annual Meeting. .
15. Kruisselbrink TM, Jouni H, Haddad RA et al. The Effect of Disclosing Coronary Heart Disease Genetic Risk on Shared-Decision Making. . 2014: Presented at the American Society of Human Genetics 64th Annual Meeting.
16. Pocock SJ, Simon R. Sequential treatment assignment with balancing for prognostic factors in the controlled clinical trial. Biometrics 1975: 31: 103-115.
17. DeMarco TA, Peshkin BN, Mars BD et al. Patient satisfaction with cancer genetic counseling: a psychometric analysis of the Genetic Counseling Satisfaction Scale. Journal of genetic counseling 2004: 13: 293-304.
18. Huang T, Yu H, Tsai M et al. Superior perceived control comes with improved health related quality of life in younger heart failure patients (Abstract). Journal of Cardiac Failure 2012: 18: S5.
19. Banerjee T, Lee KS, Browning SR et al. Limited association between perceived control and health-related quality of life in patients with heart failure. The Journal of cardiovascular nursing 2014: 29: 227-231.

20. Calfee CS, Katz PP, Yelin EH et al. The influence of perceived control of asthma on health outcomes. Chest 2006: 130: 1312-1318.
21. Judge TA, Locke EA, Durham CC. The dispositional causes of job satisfaction: A core evaluations approach. Research in Organizational Behavior 1997: 19: 151-188.
22. Zhang Y, Kwekkeboom K, Petrini M. Uncertainty, self-efficacy, and self-care behavior in patients with breast cancer undergoing chemotherapy in China. Cancer nursing 2014: Jun 18. [Epub ahead of print].
23. Kullo IJ, Haddad R, Prows CA et al. Return of results in the genomic medicine projects of the eMERGE network. Frontiers in genetics 2014: 5: 50.
24. Maddison R, Pfaeffli L, Stewart R et al. The HEART Mobile Phone Trial: The partial mediating effects of self-efficacy on physical activity among cardiac patients. Frontiers in public health 2014: 2: 56.
25. Jiang W, Li F, Jiang H et al. Core self-evaluations mediate the associations of dispositional optimism and life satisfaction. PloS one 2014: 9: e97752.
26. Srivastava A, Locke EA, Judge TA et al. Core self-evaluations as causes of satisfaction: The mediating role of seeking task complexity. Journal of Vocational Behavior 2010: 77: 255-265.
27. Sanderson SC, Persky S, Michie S. Psychological and behavioral responses to genetic test results indicating increased risk of obesity: does the causal pathway from gene to obesity matter? Public health genomics 2010: 13: 34-47.
28. Marteau TM, French DP, Griffin SJ et al. Effects of communicating DNA-based disease risk estimates on risk-reducing behaviours. The Cochrane database of systematic reviews 2010: CD007275.
29. Grant RW, Hivert M, Pandiscio JC et al. The clinical application of genetic testing in type 2 diabetes: a patient and physician survey. Diabetologia 2009: 52: 2299-2305.
30. Qureshi N, Kai J. Informing patients of familial diabetes mellitus risk: How do they respond? A cross-sectional survey. BMC health services research 2008: 8: 37.
31. Chao S, Roberts JS, Marteau TM et al. Health behavior changes after genetic risk assessment for Alzheimer disease: The REVEAL Study. Alzheimer disease and associated disorders 2008: 22: 94-97.
32. Resnicow K, Page SE. Embracing chaos and complexity: a quantum change for public health. American journal of public health 2008: 98: 1382-1389.
33. Wijers D, Wieske L, Vergouwen MD et al. Patient satisfaction in neurological second opinions and tertiary referrals. Journal of neurology 2010: 257: 1869-1874.
34. Duffy FD. Counseling for Behavior Change. In: Goldman L, Schafer AI, eds. Goldman's Cecil Medicine. Philadelphia, PA: Saunders, 2012.
35. Madson MB, Loignon AC, Lane C. Training in motivational interviewing: a systematic review. Journal of substance abuse treatment 2009: 36: 101-109.
36. Katapodi MC, Dodd MJ, Facione NC et al. Why some women have an optimistic or a pessimistic bias about their breast cancer risk: experiences, heuristics, and knowledge of risk factors. Cancer nursing 2010: 33: 64-73.
37. Austin JC. Re-conceptualizing risk in genetic counseling: implications for clinical practice. Journal of genetic counseling 2010: 19: 228-234.
38. Bloss CS, Schork NJ, Topol EJ. Effect of direct-to-consumer genome-wide profiling to assess disease risk. N Engl J Med 2011: 364: 524-534.

Table 1. Participant Characteristics (mean ± SD unless otherwise noted)

	CRS **n=103**	**CRS+GRS** **n=104**	***P***
Age, years	58.9±5.2	58.9±4.8	0.98
Male sex, n (%)	50 (48.5%)	48 (46.1%)	0.78
College education or higher, n (%)	68 (66.0%)	53 (56.7%)	0.25
Ever smoked, n (%)	41 (39.8%)	32 (30.7%)	0.20
Family history of CHD, n (%)	30 (29.1%)	25 (24.0%)	0.43
BMI, kg/m^2	30.3±6.9	30.2±6.1	0.90
SBP, mmHg	130±14	131±17	0.48
Waist circumference, cm	101±16	100±14	0.59
Total cholesterol, mg/dL	201±30	202±28	0.70
LDL-C, mg/dL	119±23	120±25	0.72
HDL-C, mg/dL	55±16	56±16	0.68
Triglycerides, mg/dL	134±69	132±78	0.89

CHD, coronary heart disease; BMI, body mass index; SBP, systolic blood pressure; LDL-C, low-density lipoprotein cholesterol; HDL-C, high-density lipoprotein cholesterol

Table 2. Perceived Personal Control Questionnaire Score by Item (mean ± SD unless otherwise noted)

		CRS n=103	CRS+GRS n=104	Item *P*	OR (95% CI)[a]	Sub-scale *P*
Cognitive Control	1. I think I understand what problem brought me to genetic counseling	2.64±0.56	2.79±0.46	0.031[b]	2.01[b] (1.06,3.81)	
	2. I feel I know the meaning of the problem for my family's future and me	2.75±0.54	2.75±0.48	0.66	0.86 (0.44,1.68)	0.015[b]
	3. I think I know what caused the problem	2.57±0.63	2.75±0.50	0.027[b]	2.00[b] (1.08,3.70)	
Behavioral Control	4. I feel I have the tools to make decisions that will influence my future	2.83±0.41	2.89±0.31	0.21	1.69 (0.75,3.80)	
	5. I feel I can make a logical evaluation of the various options available to me in order to choose one of them	2.83±0.40	2.92±0.30	0.047[b]	2.53 (0.99,6.42)	0.304
	6. I feel I can make decisions that will change my family's future	2.60±0.60	2.67±0.53	0.452	1.25 (0.70,2.23)	
Decisional Control	7. I feel there are certain things I can do to prevent the problem from recurring	2.73±0.51	2.79±0.46	0.378	1.35 (0.69,2.64)	
	8. I feel I know what to do to ease the situation	2.74±0.50	2.82±0.41	0.305	1.43 (0.72,2.85)	0.108
	9. I think I know what should be my next step	2.84±0.39	2.91±0.28	0.179	1.82 (0.76,4.36)	
	Total PPC	24.12±3.83	25.24±2.65	0.04[b]	-	-

[a] CRS is the referent group; [b] Denotes statistical significance

Table 3. Genetic Counseling Satisfaction Questionnaire Score by Item (mean ± SD unless otherwise noted)

	CRS n=101	**CRS+GRS n=103**	***P***	**OR (95% CI)[a]**
1. The genetic counselor helped me identify what I needed to know to make decisions about what would happen to me	3.31±1.35	3.52±1.09	0.366	1.33 (0.72,2.46)
2. I felt better about my health after meeting with the genetic counselor	2.93±1.30	3.20±1.15	0.105	1.54 (0.92,2.58)
3. The genetic counseling session was about the right length of time I needed	3.25±1.34	3.51±1.10	0.138	1.58 (0.86,2.89)
4. The genetic counselor was truly concerned about my wellbeing	3.34±1.33	3.53±1.10	0.387	1.32 (0.70,2.49)
5. The genetic counseling session was valuable to me	3.11±1.33	3.50±1.09	0.010[b]	2.13[b] (1.20,3.78)
Total GCS	15.93±6.34	17.27±5.27	0.064	-

[a] CRS is the referent group; [b] Denotes statistical significance