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Disclosing genetic risk for coronary heart disease: effects on perceived personal control and genetic counseling satisfaction

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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: 45–65 years) were randomized to receive estimated 10-year 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.

Keywords

clinical genetics; clinical trial; coronary heart disease; 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 (GRSs) based on such variants are 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 or 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 (GCS) scores 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 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 GRS was calculated based on

genotypes at 28 single nucleotide polymorphisms (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 with the population average, whereas a GRS of 1.4 indicates a 40% higher risk compared with 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 to 2-fold. For CRS+GRS participants, the genetic counselor also reviewed their GRS and how it was integrated into their CRS. 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 Supporting information (Appendix S1). 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 the 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). [ClinicalTrials.gov](https://clinicaltrials.gov) registration number: .

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 to 27, with higher scores indicating greater control beliefs. In addition, we analyzed cognitive, behavioral, and decisional control components as subscales of PPC. The PPC questionnaire had high internal consistency ($\alpha=0.892$).

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 to 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 ($\alpha=0.975$).

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 $\pm$ SD, and dichotomous variables were expressed as counts. The internal consistency of questionnaires was tested using Cronbach's alpha test. For analysis of the ordinal variables, we used the non-parametric 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 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 two-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 subscale 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: (i) 'I think I understand what problem brought me to genetic counseling' (2.79 ± 0.46 vs 2.64 ± 0.56 , $p=0.03$), (ii) 'I think I know what caused the problem' (2.75 ± 0.50 vs 2.57 ± 0.63 , $p=0.03$), and (iii) '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 , $p=0.047$). On the basis of 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 probably to be at ordinal levels that would indicate increased PPC compared with 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 probably 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%), $p = 0.02$].

In addition, 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 ($r = 0.15$, $p = 0.13$) nor was GCS ($r = -0.004$, $p = 0.97$). 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 PPC and satisfaction with genetic counseling compared with 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. In addition, PPC and GCS scores did not differ among patients with or 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 or 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 probably 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. As 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 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). As the cognitive control component of PPC was the subscale 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, because the CRS+GRS group had higher levels of PPC, we would expect these participants to have a higher HRQL 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 the higher PPC seen in the CRS+GRS arm produced a more satisfied cohort because we did not find PPC to be correlated with GCS. As GCS was not correlated to GRS, GRS subgroups, 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.

As PPC and GCS did not differ between GRS categories regardless of family history, it is probable 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 GRSs, regarding the correct interpretation of their GRS may help 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 estimates, 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 PPC and GCS 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. In addition, 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 higher PPC and a greater proportion of patients were 'highly satisfied' with genetic counseling compared with 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.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Table 1.Participant characteristics (mean $\pm$ SD unless other-wise noted)

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

BMI, body mass index; CHD, coronary heart disease; CRS, conventional risk score; GRS, genetic risk score; HDL-C, high-density lipoprotein cholesterol; LDL-C, low density lipoprotein cholesterol; SBP, systolic blood pressure.

Table 2.Perceived personal control questionnaire score by item (mean $\pm$ SD unless otherwise noted)

		CRS+GRS (n = 104)	Item p	OR (95% CI) ^a	Subscale p
Cognitive control	1. I think I understand what problem brought me to genetic counseling	2.64 $\pm$ 0.56	0.031 ^b	2.01 ^b (1.06, 3.81)	0.015 ^b
	2. I feel I know the meaning of the problem for me family's future and me	2.75 $\pm$ 0.54	0.660	0.86 (0.44, 1.68)	
	3. I think I know what caused the problem.	2.57 $\pm$ 0.63	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 $\pm$ 0.41	0.210	1.69 (0.75, 3.80)	0.304
	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 $\pm$ 0.40	0.047 ^b	2.53 (0.99, 6.42)	
Decisional control	6. I feel I can make decisions that will change my family's future.	2.60 $\pm$ 0.60	0.452	1.25 (0.70, 2.23)	
	7. I feel there are certain things I can do to prevent the problem from recurring	2.73 $\pm$ 0.51	0.378	1.35 (0.69, 2.64)	0.108
	8. I feel I know what to do to ease the situation	2.74 $\pm$ 0.50	0.305	1.43 (0.72, 2.85)	
	9. I think I know what should be my next step	2.84 $\pm$ 0.39	0.179	1.82 (0.76, 4.36)	
	Total PPC	24.12 $\pm$ 3.83	0.044 ^b	-	-

CI, confidence interval; CRS, conventional risk score; GRS, genetic risk score; OR, odds ratios; PPC, perceived personal control.

^aCRS is the referent group.^bDenotes statistical significance

Table 3.Genetic counseling satisfaction questionnaire score by item (mean $\pm$ 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 $\pm$ 1.35	3.52 $\pm$ 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 $\pm$ 1.30	3.20 $\pm$ 1.15	0.106	1.54 (0.92, 2.58)
3. The genetic counseling session was about the right length of time I needed	3.25 $\pm$ 1.34	3.51 $\pm$ 1.10	0.138	1.58 (0.86, 2.89)
4. The genetic counselor was truly concerned about my well-being	3.34 $\pm$ 1.33	3.53 $\pm$ 1.10	0.387	1.32 (0.70, 2.49)
5. The genetic counseling session was valuable to me	3.11 $\pm$ 1.33	3.50 $\pm$ 1.09	0.010 ^b	2.13 ^b (1.20, 3.78)
Total GCS	15.93 $\pm$ 6.34	17.27 $\pm$ 5.27	0.064	-

CI, confidence interval; CRS, conventional risk score; GRS, genetic risk score; ORs, odd ratios; GCS, genetic counseling satisfaction

^a CRS is the referent group^b Denotes statistical significance