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### Patient Beliefs and Behaviors About Genomic Risk for Type 2 Diabetes: Implications for Prevention

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# Patient Beliefs and Behaviors About Genomic Risk for Type 2 Diabetes: Implications for Prevention

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Type 2 diabetes is a major health burden in the United States, and population trends suggest this burden will increase. High interest in, and increased availability of, testing for genetic risk of type 2 diabetes presents a new opportunity for reducing type 2 diabetes risk for many patients; however, to date, there is little evidence that genetic testing positively affects type 2 diabetes prevention. Genetic information may not fit patients' illness representations, which may reduce the chances of risk-reducing behavior changes. The present study aimed to examine illness representations in a clinical sample who are at risk for type 2 diabetes and interested in genetic testing. The authors used the Common Sense Model to analyze survey responses of 409 patients with type 2 diabetes risk factors. Patients were interested in genetic testing for type 2 diabetes risk and believed in its importance. Most patients believed that genetic factors are important to developing type 2 diabetes (67%), that diet and exercise are effective in preventing type 2 diabetes (95%), and that lifestyle changes are more effective than drugs (86%). Belief in genetic causality was not related to poorer self-reported health behaviors. These results suggest that patients' interest in genetic testing for type 2 diabetes might produce a teachable moment that clinicians can use to counsel behavior change.

Type 2 diabetes (T2D) is a major burden in the United States, causing acute and chronic disability, increased health services utilization, and premature death for millions (Centers for Disease Control and Prevention, 2011; Danei et al., 2010). The increasing median age and the high prevalence of obesity in the U.S. population suggest that the burden of T2D is likely to continue to increase (Engelgau et al., 2011; Sloan, Bethel, Ruiz, Shea, & Feinglos, 2008).

Several recent developments have the potential to increase the role of a new tool in T2D prevention: genetic testing for T2D risk. First, about 65 common DNA variants (single nucleotide polymorphisms) are now associated with increased risk of T2D (de Miguel-Yanes et al., 2011; McCarthy, 2011). Second, direct-to-consumer genetic testing is available and becoming less expensive. Third, the general

public has expressed high interest in and positive attitude toward genetic testing for risk of common diseases, including T2D (Etchegary et al., 2010; Grant et al., 2009; Lipkus, Iden, Terrenoire, & Feaganes, 1999). The confluence of these three developments is likely to result in a growing number of patients who are at risk for T2D asking providers for genetic testing, or obtaining genetic risk information on their own and asking providers for guidance in interpreting or responding to it. In one study that provided participants with genomic risk profiles, 92% of participants intended to share this information with their physician, mostly for the purpose of personalized recommendations for care (Gollust et al., 2012). Consumer interest in genetic risk information, then, could serve to stimulate discussion of risk reducing measures between patients and providers. Even though researchers have yet to establish the personal and clinical utility of genetic risk information (Grant et al., 2013; Marteau et al., 2010; McBride, Koehly, Sanderson, & Kaphingst, 2010; Scheuner, Sieverding, & Shekelle, 2008), providers might be

able to use these new discussion opportunities to counsel adoption of proven preventive behaviors such as diet, exercise, and medication (Knowler et al., 2002). The purpose of this paper is to examine whether patients' T2D illness representations are favorable to support such lifestyle changes.

The exploration of personal and clinical utility of genetic information can be framed by the Common Sense Model of self-regulation of health behavior (Leventhal, Brissette, & Leventhal, 2003). The Common Sense Model posits that patients' emotional and cognitive representations of a health threat play critical roles in interpretation of, and subsequent reaction to, that threat (Marteau & Weinman, 2006). Components of health threat representations, such as perceived personal risk and causality of the disease, are thought to play an important role in individuals' response to risk information and adoption of treatment or preventive behaviors in response to that threat (Marteau & Weinman, 2006; Petrie & Weinman, 1997). Personal risk information is thought to activate the threat representations associated with a health threat, which, in turn, activates a coping plan (e.g., behavior change or medication). Marteau and Weinman (2006) proposed that at least two conditions must be met for health risk information to result in adoption of coping behavior that can prevent or treat the disease: (a) a link between the health risk information and the patient's cognitive representation of the cause of the threat and (b) a link between the health risk information and possible coping strategies.

In the case of genetic risk information and T2D, the first condition would be met if patients' cognitive representations of the threat of T2D include genetic contributions to its causality. The second condition would be met if patients' representations of T2D include the belief that the threat can be addressed (i.e., the illness can be prevented, even in the face of genetic risk) through appropriate coping strategies such as dietary improvements, exercise, or medication. In contrast, genetic information might result in fatalistic beliefs that T2D cannot be prevented at all (Claassen et al., 2010), or in discounting of behavioral treatment strategies among people with high behavioral risk (O'Neill, McBride, Alford, & Kaphingst, 2010; Wang & Coups, 2010). Such fatalistic beliefs may manifest as less physical activity and poorer diet habits among people with representations that include genetic causality. Existing evidence suggests that many people believe in at least partial genetic or hereditary causality for chronic conditions (Hilbert et al., 2009; McBride et al., 2009), including T2D (Calsbeek, Morren, Bensing, & Rijken, 2007). In summary, genetic risk information might be most effective in motivating appropriate coping strategies if patients' representations of T2D include genetic causality as well as behavioral prevention. Favorable prevention beliefs and some prevention behaviors may make participants more open to the risk counseling (with or without genetic testing) and better able to accurately interpret that information and use it to actually enact positive health behaviors. In many diseases that are preventable and have well-defined risk factors and modes of reducing risk, accurate beliefs and positive beliefs about the efficacy of prevention

may reflect an activated patient, who may be the most likely population subgroup to react more appropriately and favorably to the risk information and suggestions for risk reduction. No existing studies assess beliefs about both T2D causality and T2D prevention among primary care patients who seek T2D genetic testing.

Therefore, we examined baseline illness representations and beliefs about T2D prevention in primary care patients who were participating in a T2D genetic risk testing study. Our goal was to determine whether representations of T2D and genetic testing are favorable for adoption of healthy behavior changes; if they are, patients' interest in genetic testing could serve as a springboard for productive counseling. Specifically, we intended to assess the following: (a) What are patients' causality and preventability beliefs related to T2D? (b) What are patients' beliefs regarding preventive coping behaviors for T2D prevention, given genetic risk beliefs? and (c) Are higher beliefs in genetic causality related to less self-reported diet and exercise behavior (O'Neill et al., 2010; Wang & Coups, 2010)?

## Method

This secondary analysis was conducted using baseline data from a randomized controlled trial of the clinical and behavioral effects of T2D counseling based upon traditional clinical risk factors as compared with risk counseling based upon both traditional clinical risk factors and genetic risk test results (Cho et al., 2012). The randomized controlled trial study protocol was approved by the Duke University Medical Center Institutional Review Board and took place in two primary care clinics. Full details of the trial protocol have been reported elsewhere (Cho et al., 2012).

## Participants and Procedures

Briefly, clinic patients were recruited in the clinical laboratory waiting areas of two primary care outpatient clinics in Durham, North Carolina, and through flyers posted in the two clinics. Study exclusion criteria were as follows: age <18 or >81 years; self-reported history of diabetes; self-reported history of prior genetic testing for diabetes risk; currently pregnant, current or past use of diabetes medications; baseline fasting glucose (tested at enrollment)  $\geq 7$  mmol/L (126 mg/dL); not currently fasting and unwilling to return for a fasting blood test; and unable to provide informed consent.

During the consent process, genetic testing for T2D risk was described to prospective participants; patients were informed that they could enroll in the study whether or not they wished to have testing. Participants who consented to testing were randomized to one of two study arms: (a) risk counseling visit with a provider based on clinical (phenotypic) risk factors for T2D; and (b) risk counseling visit with a provider based on clinical and genetic risk factors for T2D (four single nucleotide polymorphisms associated with T2D risk). Those who declined were assigned to a "no genetic testing" arm.

Follow-up data regarding risk perceptions and beliefs, study outcomes of behavior change (diet, exercise) and clinical markers were collected at 6 weeks, 3 and 12 months postrisk counseling in the parent study. Only baseline data are included in the present analysis.

### Study Measures

Survey items were selected from previously validated instruments or were created based on (a) Common Sense Model principles, (b) items used by the research team in previous studies, and (c) the format and response options of the well-validated Brief Illness Perception Questionnaire (adapted for type 2 diabetes; Broadbent, Petrie, Main, & Weinman, 2006). Survey items and variables for the present analysis are defined and listed in Table 1. For example, responses to items with *strongly disagree* to *strongly agree*

scales were recoded into disagree (combining *strongly disagree* and *disagree*), agree (combining the responses *strongly agree* and *agree*), and not sure/don't know (following Morren et al., 2007).

### Statistical Analysis

Descriptive statistics were calculated for each variable. Five-point Likert-scale responses were collapsed for analysis into two to three response categories to capture the conceptual response categories of agree/positive, neutral, and disagree/negative and achieve sufficient power for these analyses. For example, responses to items with *strongly disagree* to *strongly agree* scales were recoded into disagree (combining *strongly disagree* and *disagree*), agree (combining the responses *strongly agree* and *agree*), and not sure/don't know (following

**Table 1.** Survey instruments and items

Variable	Measure
T2D clinical risk factors	Height and weight (to calculate BMI in kg/m <sup>2</sup> ) Waist circumference Fasting plasma glucose Fasting insulin Family history (number of first- and second-degree biological relatives with T2D; classified as positive or negative family history using the algorithm described in Hariri and colleagues (2006))
Interest in genetic testing	"I do not want a genetic test to tell me I am at risk to have a certain disease," reported on a 5-point scale ranging from 1 ( <i>strongly disagree</i> ) to 5 ( <i>strongly agree</i> ; Morren et al., 2007) "How important is it to know if you have a genetic risk for diabetes?" reported on a 5-point scale ranging from 1 ( <i>extremely important</i> ) to 5 ( <i>not at all important</i> ).
Belief in genetic causality of T2D	"How important are genetic factors to getting diabetes?" reported on a 5-point scale ranging from 1 ( <i>extremely important</i> ) to 5 ( <i>not at all important</i> )
Belief in behavioral preventability of T2D	"Health behaviors prescribed by my doctor will be effective in preventing diabetes," reported on a 5-point scale ranging from 1 ( <i>strongly disagree</i> ) to 5 ( <i>strongly agree</i> ) "How effective is exercising about 30 minutes every day in preventing diabetes?" "How effective is eating five servings of fruits and vegetables in preventing diabetes?" "How effective is eating a low-fat diet in preventing diabetes?" All responses were reported on a 5-point scale ranging from 1 ( <i>completely effective</i> ) to 5 ( <i>not at all effective</i> ); the mean of the latter three items was calculated as a composite score for belief in diet and exercise behaviors to prevent T2D
Belief in efficacy of medication for prevention of T2D	"How effective is taking a drug to prevent diabetes?" reported on a 5-point scale ranging from 1 ( <i>completely effective</i> ) to 5 ( <i>not at all effective</i> ) "Which is more effective in preventing diabetes, lifestyle (diet and exercise) or taking a drug?" reported on a 5-point scale ranging from 1 ( <i>completely DRUG</i> ) to 5 ( <i>completely LIFESTYLE</i> )
Perceived control over T2D	Personal control subscale of the Brief IPQ (adapted for type 2 diabetes; Broadbent et al., 2006). The personal control score was the mean of six items (e.g., "Whether or not I get diabetes depends on me") reported on a 5-point scale ranging from 1 ( <i>strongly disagree</i> ) to 5 ( <i>strongly agree</i> ).
Fatalism	"If a genetic test shows you have an above-average genetic risk for diabetes, can diabetes be prevented?" reported on a 5-point scale ranging from 1 ( <i>definitely CAN be prevented</i> ) to 5 ( <i>definitely CANNOT be prevented</i> )
Eating behavior	16-item questionnaire assessing frequency of intake of various foods over the last month (by day, week, month; Thompson et al., 2007).
Physical activity	16-item questionnaire assessing activity related to work, transit and leisure time activities as well as sedentary time (Armstrong & Bull, 2006).

Note. T2D = type 2 diabetes.



Morren et al., 2007). Descriptive statistics (means, frequencies, percentages) for survey items were utilized to determine patients' causality, risk, and preventability beliefs related to T2D, as well as one item that explicitly assessed patients' beliefs regarding preventive coping behaviors for T2D prevention even in light of hypothetical genetic risk (i.e., fatalism).

To examine the relations between belief in genetic causality and lifestyle behaviors and fatalism, we used a logistic regression model with belief in genetic causality (important vs. not important/neutral) as the dichotomous outcome and self-reported diet behaviors, self-reported exercise behavior, and fatalism scores as explanatory variables (to control for all other variables when examining each variable's relation with genetic causality belief). We examined the correlation matrix of explanatory variables and checked for multicollinearity. We used chi-square analyses to examine relations between genetic causality beliefs and preventability beliefs (belief in the effectiveness of drugs, and comparative effectiveness beliefs in drugs versus health behaviors). All analyses were performed using SPSS 20 or SAS 9.2 software.

## Results

We approached 1,416 patients to participate in the study. Of these, 430 patients were fasting for laboratory tests, eligible based on initial screen criteria and provided informed consent. Of these, 12 were excluded for incomplete baseline data; nine had fasting glucose  $\geq 7$  mmol/L, resulting in total of 409 patients. Of these, 391 (95.6%) chose to undergo

genetic testing for T2D (18 enrolled in the no-testing arm and simply completed follow-up surveys). Demographic characteristics and clinical risk factors for the study sample are summarized in Table 2. All further analyses were performed using data for these 391 participants. Response distributions for all items appear in Table 3.

Participants were highly interested in genetic testing for their risk of T2D as indicated by the choice to be randomized to a study arm with genetic testing. Interest in genetic testing was further supported by the strong disagreement with the statement "I do not want a genetic test to tell me I am at risk to have a certain disease" (88% disagreed, 4% agreed and 8% were not sure). Furthermore, the importance of knowing one's genetic risk for T2D was rated as "important" by 82% of participants and "not important" by 18%.

### Threat Representation Components

The majority of participants (67%) expressed belief in at least partial genetic causality of T2D (see Table 3). Only 11% of participants expressed the fatalistic belief that T2D was unpreventable if a test indicated an increased genetic risk. Participants' average level of perceived control over development of diabetes was high ( $M = 4.01/5$ ,  $SD = 0.57$ ).

### Prevention Beliefs

A significantly larger proportion of participants expressed belief in the preventive effectiveness of behaviors (diet,

**Table 2.** Sample demographics and type 2 diabetes risk factors

	Interested in genetic testing For T2D risk ( $n = 391$ )	Declined genetic testing for T2D risk ( $n = 18$ )
Average age (years), $M$ ( $SD$ )	50 (13)	46 (16)
Sex (% female)	70	78
Race <sup>a</sup> (%)		
American Indian or Alaska Native	1	0
Asian	5	6
Black or African American	29	50
Native Hawaiian or other Pacific Islander	1	0
White	60	39
Multirace	5	6
Highest education level <sup>b</sup> (%)		
High school or less	10	39
Some college	26	22
College degree	34	22
Advanced degree	30	17
Body mass index, $M$ ( $SD$ )	30.79 (7.37)	32.36 (6.48)
Fasting glucose (mg/dL), $M$ ( $SD$ )	94.95 (10.6)	91.78 (15.6)
Family history of T2D (% positive)	58	44
Daily servings of fruit/vegetables <sup>c</sup> , $M$ ( $SD$ )	3.65 (2.38)	3.89 (2.30)
Daily servings of fatty foods <sup>d</sup> , $M$ ( $SD$ )	1.45 (1.30)	1.74 (1.67)
MVPA (days per month), $M$ ( $SD$ )	6.58 (5.41)	6.61 (5.74)
Smoking (yes)	7.9	16.7

*Note.* MVPA = moderate/vigorous physical activity. "Some college" includes trade or technical school. Percentages may not sum to 100% because of rounding. Participants with missing data are excluded from percentage calculations. Number of missing data points: <sup>a</sup>one among participants interested in genetic testing (3 did not wish to report); <sup>b</sup>three missing among participants interested in genetic testing; <sup>c</sup>eight missing among participants interested in genetic testing; one among those who declined; <sup>d</sup>eight missing among participants interesting in genetic testing; one missing among those who declined.

**Table 3.** Components of threat perceptions

Causality	Not important/important		Neutral
How important are genetic factors to getting diabetes? <sup>a</sup>	67%		33%
Interest in genetic testing			
How important is it to know if you have a genetic risk for diabetes?	82.3%		17.7%
I do not want a genetic test to tell me I am at risk for a certain disease. <sup>a</sup>	Disagree 88.0%		Agree/neutral 12.0%
Perceived control over T2D			
Mean score of IPQR—Personal Control Subscale (max. 30), <i>M</i> ( <i>SD</i> )			24.04 (3.4)
Fatalism	Yes		Neutral/no
If a genetic test shows you have an above-average genetic risk for diabetes, can diabetes be prevented?	<sup>a</sup> 61.3%		38.7%
Prevention beliefs—behavioral and medication			
Health behaviors prescribed by my doctor will be effective in preventing diabetes. <sup>b</sup>	Agree 81%	Disagree 4%	Not sure/don't know 15%
Belief in diet and exercise effectiveness	Effective 84%	Not effective/neutral 16%	
How effective is taking a drug to prevent diabetes? <sup>c</sup>	12%	88%	
Which is more effective in preventing diabetes, lifestyle (diet & exercise) or taking a drug? <sup>d</sup>	Drug <1%	Equal 13%	Lifestyle 87%

*Note.* “Belief in diet and exercise effectiveness” is a composite measure of three items assessing belief in the effectiveness of decreasing fatty food consumption, increasing fruit and vegetable consumption, and increasing exercise. Percentages may not sum to 100% because of rounding. Number of missing data points: <sup>a</sup>one, <sup>b</sup>three, <sup>c</sup>seven, <sup>d</sup>three. IPQR = Illness Perception Questionnaire (revised); T2D = type 2 diabetes.

exercise) for T2D (84%) than expressed belief in the preventive effectiveness of drugs (12%;  $\chi^2[1, N = 391] = 315.11, p < .001$ ). On the item directly comparing drug to lifestyle prevention, 87% indicated that lifestyle was more effective.

### Threat Representations and Health Behaviors

Belief in genetic causality was not related to self-reported behaviors (dietary intake or physical activity) or fatalism (all  $ps > .140$ ). Genetic causality was also not related to belief in drug treatability of T2D ( $p = .390$ ), but was related to the comparative effectiveness of drugs vs. lifestyle (Fisher's exact  $\chi^2, p = .027$ ) such that participants who believed that genetic factors are important to developing T2D were more likely to believe that behaviors are more effective in reducing disease risk (90%) than those who believed genetic factors are not important (82%; see Table 4). The logistic regression modeling belief in genetic causality (important vs. not important/neutral) as the dichotomous outcome and self-reported

diet behaviors, self-reported exercise behavior, and fatalism scores as explanatory variables did not result in any significant explanatory variables or models ( $ps = .17-.66$  for each of the variables/models).

### Discussion

In our sample, we found evidence of illness representations that would be supportive of adopting behavior changes to prevent development of T2D. Despite high interest in genetic testing, and belief that T2D risk is at least partially genetically caused, patients believed in the effectiveness of healthy eating and exercise (and to a lesser degree medication) for T2D prevention. According to the Common Sense Model (Marteau & Weinman, 2006), these beliefs create favorable conditions under which genetic risk information can help promote preventive behavior. We found little fatalism, and no relations between genetic causality beliefs and current self-reported behavior or perceived control over risk for T2D, suggesting that belief in genetic causality has not undermined perceived utility of healthy behaviors to prevent diabetes in this sample. We found a relation between belief in genetic causality and the relative preventive effectiveness of drugs versus behaviors; however, the pattern of this relation does not support the idea that belief in genetic causality leads to greater belief in biological treatment or prevention. Patients who believed genetic factors are important to developing T2D were more likely to believe that behaviors are more effective as compared with drugs than patients who believe genetic factors are not important. Although we did

**Table 4.** Relation between belief in genetic causality and belief in comparative effectiveness of drugs versus lifestyle (%)

Genetic causality belief	Prevention belief		
	Drug	Equal	Lifestyle
Important	<1	10	90
Neutral/not important	0	18	82

*Note.* The distribution of prevention beliefs differed by genetic causality belief, Fisher's exact  $\chi^2, p = .027$ .

not find specific evidence to support or refute this finding in the literature, we hypothesize that patients who believed genetic factors are important or at least somewhat influential in T2D have 'accurate' genetic causality beliefs in regard to this chronic disease and may also be more informed as to what preventive measures are most effective based on the scientific evidence such as the Diabetes Prevention Program (Florez et al., 2006). In other words, these patients may simply represent more activated and informed patients in regard to T2D and associated preventive measures.

In sum, our results suggest that patients' interest in genetic testing for T2D might prove fruitful by spurring consultation with providers about prevention, and in turn, actual behavior change. In further investigation of this possibility, it will likely be crucial to consider emotional processes that work alongside cognitive processes, and could act to promote or undermine positive coping. Prospective trials that comprehensively assess threat representations are needed to learn more about how both cognitive and emotional components of representations affect reaction to genetic risk information and subsequent coping behaviors.

### *Potentially Negative Correlates of Threat Representations*

Some have posited that representations that include genetic causality might also lead to a fatalistic belief that a disease with a genetic component cannot be prevented or treated through behavior change. For example, belief in genetic causality for cardiovascular disease, obesity, or cancer has been reported to be associated with less preventive behaviors, more risky behaviors, greater belief in biological treatment effectiveness, or less perceived control (Claassen et al., 2010; Kaphingst, Lachance, & Condit, 2009; Senior & Marteau, 2007; Senior, Marteau, & Peters, 1999; Wang & Coups, 2010). A recent meta-analysis, however, concluded that the evidence for such fatalism in response to genetic testing is limited (Collins, Wright, & Marteau, 2011). An example of this lack of fatalism is the findings of Pijl and colleagues' (2006) randomized controlled trial, which showed that providing personal familial risk information for T2D risk resulted in higher perceived control and subsequent health behavior changes as compared with standard clinical T2D risk information alone. The present findings are commensurate with this latter view; we found little evidence for fatalism despite prevalent belief in genetic causality. It could be simply that relations between causality beliefs and other constructs are specific to diseases and populations, and could change on the basis of personal risk, new diagnosis, or general risk assessment. To our knowledge, ours is the first investigation of causality and preventability beliefs and their correlates specifically related to T2D and considering genetic risk.

### *Implications for Utility of Genetic Testing*

The present findings suggest that patients who express interest in, or already possess, genetic information about their risk of developing T2D do not necessarily hold beliefs that will hamper effective prevention. Patients believe that genetic factors

do play a role in developing T2D, but this belief does not indicate (a) a lack of belief in the effectiveness of behavior change, (b) an inappropriate level of belief in biological (drug) prevention strategies, or (c) current poor diet or exercise behaviors. Provider and patient discussion of risk for T2D, then, can focus on explaining personal risk factors (genetic and/or clinical), education and counseling about behavior change, and consideration of medication if warranted. Discussion of T2D risk does not, for example, need to focus on disabusing patients of fatalistic beliefs or persuading them that behavior change is important. Instead, providers could focus on personalized approaches to address motivation, barriers to behavior change, and supportive resources.

In this way, genetic testing for T2D may not have clinical utility by its restrictive definition—genetic testing results may not lead to behavior change, to more accurate diagnosis, or to better treatment or prevention (Foster, Mulvihill, & Sharp, 2009). A review of the clinical utility in terms of behavioral outcomes of genetic risk counseling for disease in which behavioral strategies were effective in preventing disease revealed that smoking and physical activity did not improve, whereas self-reported diet improved slightly in two studies (Marteau et al., 2010). However, patients' interest in genetic testing might have personal utility completely separate from any actual results of the test. Patients who would otherwise not seek information about their risk could be exposed to such information in the course of pursuing or asking providers about genetic test results. That is, a patient who is at risk, but does not yet meet clinical diagnosis of diabetes, might never discuss medication or behavior change with a provider until developing T2D, when prevention is no longer a possibility. Some research indicates that this poor awareness and discussion of T2D risk is the norm; for example, in the National Health and Nutrition Survey, 34.6% of respondents had prediabetes, but only 4.8% of these reported having received this diagnosis from their provider, and only around 32% of these received recommendations for exercise or diet (Karve & Hayward, 2010).

We must acknowledge the selection bias that is created in this study and other proposed risk counseling scenarios, in which patients who may already be information seekers discuss diabetes risk with their providers regardless of the availability and consideration of genetic risk testing. Yet, genetic testing could be a spur that facilitates information seeking and subsequent greater exposure to T2D risk and prevention counseling among at-risk patients. Several findings support the plausibility of this type of utility of T2D genetic risk information. First, many at-risk patients never discuss T2D risk with a health professional (Karve & Hayward, 2010). Second, the present study adds to the many studies to date that have found very high levels of interest in genetic testing among many populations (Calsbeek et al., 2007; Grant et al., 2009; Hivert et al., 2009; Jallinoia & Aro, 2000). Third, at least one study found that interest in genetic testing was related to uptake of testing (Sanderson, O'Neill, Bastian, Bepler, & McBride, 2010), demonstrating that interest does lead to actual pursuit of information. And fourth, at least two studies have found that most of the people who obtain



genetic risk information intend to share it with their provider (Gollust et al., 2012; O'Daniel, Haga, & Willard, 2010). With greater availability of genetic testing, these processes could converge to result in more patients discussing T2D risk with providers, and more patients initiating preventive behaviors. Continuing research is needed into best practices for communicating genetic risk results and integrating genetic risk with other risk factors.

### Limitations

The present findings should be interpreted with consideration of study limitations. First, our sample was comprised exclusively of people who were interested in genetic testing. Because our research questions focused on representations in people with interest in genetic testing, this was an appropriate sample; however, our results do not speak to illness representations in those who are not interested in genetic testing. Most studies have found widespread interest in genetic risk testing in many samples and for several diseases, but for those not interested, illness representations might be different, and the prospect of a genetic test might have entirely different consequences. A related limitation is that our sample was generally highly educated, and representations and behaviors in this sample might not be representative of the full population of people interested in genetic testing. Although interest in genetic testing appears to be widespread, differences in illness representations between patients might complicate use of genetic testing to counsel behavior change.

A third limitation is related to the measures. We assessed several study constructs with single or few items. Although brief measures of psychological constructs have proven to be valid (Wood, Nye, & Saucier, 2010), future research might benefit from more thorough or detailed measurement of threat representation concepts. These constructs were also assessed using self-report measures, which have potential for social desirability bias and other limitations. However, this study may also provide validity evidence for self-report measures of threat representations, upon which the present study and other research rely. A fourth limitation is that the cross-sectional nature of our study does not allow for investigating the impact of patients' representations on clinical outcomes. Future prospective research is necessary to establish connections between representations, behaviors, and downstream clinical outcomes after seeking or receiving T2D risk information.

### Conclusion

One proposed framework for exploring the potential personal and clinical utility of genetic risk information is to explore whether the cognitive representations of illness and illness threats support adoption of effective treatment or prevention behaviors. The present results suggest that among patients interested in genetic testing, illness representations for T2D might have a good fit with genetic risk information. Although it does not fit the formal definition of clinical utility, this supports the idea of using interest in genetic testing as

a teachable moment for providers and patients to discuss T2D risk and modifiable risk factors. These results also underscore the need for continued investigation of the personal and clinical utility of including genetic information in comprehensive risk counseling for chronic diseases such as T2D, to determine whether favorable conditions created by illness representations can be leveraged to change behavior and prevent disease.

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