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Perceptions of the roles of behaviour and genetics in disease risk: Are they associated with behaviour change attempts

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Abstract

The aims of the present study were to (i) examine the prevalence of perceived behavioural and genetic causal beliefs for four chronic conditions (i.e. obesity, heart disease, diabetes and cancer); (ii) to examine the association between these causal beliefs and attempts at behaviour change (i.e. physical activity, weight management, fruit intake, vegetable intake and soda intake). The data come from the Health Information National Trends Survey, a nationally representative population-based survey of adults ($N = 3407$). Results indicated that participants held both behavioural and genetic causal beliefs for all four chronic conditions. Multivariate analyses indicated that behavioural causal beliefs were significantly associated with attempts to increase physical activity and vegetable intake and to decrease weight. Genetic causal beliefs for cancer were significantly associated with reported attempts to maintain weight. Behaviour and genetic causal beliefs were not associated with changes in either fruit or soda intake. In conclusion, while behavioural causal beliefs are associated with behavioural change, measurement must capture diseasespecific behavioural causal beliefs as they are associated with different health behaviours.

Keywords

causal beliefs; behaviour change; chronic conditions

Non-communicable, chronic diseases such as diabetes, heart disease and cancer comprise the most common and preventable health problems in the developed and developing world (Partnership to Fight Chronic Disease [PFCD], 2013). In 2010, half of the US population had at least one chronic health condition, with many suffering from multiple conditions (Anderson, 2010). By 2030, that number is projected to reach 171 million. The rising rates of chronic diseases pose a significant and growing public health problem. In the US, chronic diseases account for more than 80% of deaths annually, are the leading cause of disability and contribute to rising health care costs (Anderson, 2010; Hovort & Xu, 2012; US Burden of Disease Collaborators, 2013).

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Public perceptions of the roles of genetics and behaviours in chronic diseases

Research provides strong evidence for the association between modifiable risk factors such as tobacco use and exposure, physical inactivity, unhealthy diet and the increasing rates of chronic diseases (Danaei et al., 2009; Kim & Oh, 2013; Leventhal, Huh, & Dunton, 2013; Stein & Colditz, 2004; Yusuf et al., 2004), yet there is low public adherence to national and federal health guidelines and recommendations for physical activity and diet. Recent data from the Centers for Disease Control and Prevention (CDC) indicate that only one in five adults in the US meet recommended guidelines for both aerobic and muscle-strengthening physical activity (CDC, 2013), and 37.7 and 22.6% of US adults consume less than one daily serving of fruits and vegetables, respectively (National Center for Chronic Disease Prevention and Health Promotion, 2013). Behavioural theories such as the Health Belief Model (Rosenstock, Stretcher, & Becker, 1988), the Theory of Planned Behaviour (Ajzen, 1999) and Social Cognitive Theory (Bandura, 1986) attempt to explain behaviour change by acknowledging that (i) the link between health information and behaviour change is mediated by cognitions; (ii) knowledge about the behaviour is only one of the multiple components to produce behavioural change; and (iii) perceptions, motivations and the environment exert influence on behaviour (The National Cancer Institute [NCI], 2005).

A common theme in cognitive-behavioural theories is the tenet that behavior change is influenced by the surrounding health cognitions or beliefs that an individual has about the behaviour and disease. Behavioural causal beliefs refer to the attributions that an individual makes in identifying modifiable risk factors and lifestyle factors (e.g. diet, physical activity or smoking) as the cause of health and disease, while genetic causal beliefs refer to the attributions for genes, the role of heredity and family history of disease. On one hand, behavioural and genetic causal belief attributions may constitute diametrically opposed measures, existing on opposite ends of a polar scale, that is, the endorsement of one set of causal beliefs may diminish the value of the other, and some study findings support this polar relationship (Marteau & Weinman, 2006; Senior, Marteau, & Peters, 1999). However, individuals' causal beliefs for disease may contain both behavioural and genetic attributions as some research suggests that these are not diametrically opposed constructs as (Condit et al., 2009; Murphy et al., 2005; O'Neill, McBride, Alford, & Kaphingst, 2010; Sanderson et al., 2013; Sanderson, Waller, Humphries, & Wardle, 2011; Wang & Coups, 2010), and that in some cases, holding genetic causal beliefs increases the likelihood that the individual will also endorse behavioural causal beliefs (O'Neill et al., 2010; Sanderson et al., 2013, 2011). In the present study, we argue that the relationship between genetic and behavioural causal beliefs extends beyond a continuum scale as individuals may hold behavioural and genetic causal beliefs in equal regard.

Genetic information and health behaviours

With advances in commercially available genetic screening tests and with more biotechnology and genetic information disseminated to the public, there is concern that that a public focus on genetic causes of chronic diseases may reduce preventive and promotive

health practices (Condit, 2001; Sanderson et al., 2011; Ten Eyck, 2005). It is possible that beliefs for genetic causes of disease and heritability may engender beliefs in genetic determinism, invoke fatalistic beliefs and lower self-efficacy in activating behaviour change (Condit et al., 2009; Senior et al., 1999; Senior, Marteau, & Weinman, 2000; Wright et al., 2012). The assignment of genetic causes for disease has been associated with lowered perceived effectiveness of treatment (Marteau et al., 2004; Wright et al., 2012) and with lower engagement in health behaviours (Wang & Coups, 2010). A study by Wright and colleagues (2012) demonstrated that genetic causal beliefs for heart disease reduced the perceived effectiveness of non-pharmacological treatments (i.e. healthy diet and physical activity) but had no effect on perceived medication effectiveness. However, genetic causal beliefs for obesity did not appear to influence perceived effectiveness for either treatment. Thus, rather than discouraging overall behaviour change, genetic risk information may change individuals' beliefs about the most effective strategies to improve health for a particular health condition.

While previous studies have explored behavioural and genetic causal beliefs of chronic diseases, they have mainly focused on clinical samples with previously diagnosed conditions in which changes in risk perception and perceived effectiveness of treatment have been studied (Marteau et al., 2004; Wright et al., 2012). However, a recently published paper by Waters et al. (2014) utilising population-based data from the Health Information National Trends Survey (HINTS) examined the associations of behavioural and genetic causal beliefs with *current* health behaviours such as current smoking status, weekly physical activity, dietary intake, cancer screening adherence and obtaining primary care visits. While their findings indicated that endorsing both behavioural and genetic causal beliefs was associated with increased cancer screening, causal beliefs were not found to be significantly associated with the other measures of *current* health behaviours. We believe that this may be due to the way in which current engagement in preventive health behaviours (e.g. physical activity and dieting) is driven by a myriad of factors that include but also extend beyond social-cognitive factors. These factors may include socio-economic status (Carlson et al., 2014; Irala-Estevez et al., 2000), peer and societal influence (Salvy, de la Haye, Bowker, & Hermans, 2012; St George & Wilson, 2012), structural barriers and factors relating to access (McIsaac, Fuller-Thomson, & Talbot, 2001; Pahor, 2011; Riis, Grason, Strobino, Ahmed, & Minkovitz, 2012), other predisposing factors such as age and gender (Milanovic et al., 2013; Ottenbacher et al., 2014; Rolls, Dimeo, & Shide, 1995) in addition to the socialcognitive factors that include knowledge, attitudes, motivations and beliefs (Hagger & Chatzisarantis, 2009; Plotnikoff, Costigan, Karunamuni, & Lubans, 2013). As a result, causal beliefs may be unable to account for enough explanatory variance of engagement of current health behaviours.

On the other hand, while *changes* in health behaviours are susceptible to the same factors that we mention above, we believe that social-cognitive factors such as the causal beliefs surrounding chronic conditions are more relevant in determining whether or not an individual engages in healthy behavioural *changes* in attempts to mitigate risk for illness or disease. For example, one's beliefs about what causes chronic illness are likely to induce health behaviour change, particularly if the perceived cause is an under-lying health behaviour or modifiable risk factor. To our knowledge, this is the first population-based

study in the US that examines genetic and behavioural causal beliefs for the development of multiple chronic conditions and their associations with changes in health behaviour.

It is not known whether behavioural causal beliefs and genetic causal beliefs constitute constructs that exists on polar ends of a scale as some research suggests that the endorsement of one may diminish the value of the other (Marteau & Weinman, 2006; Senior et al., 1999) or if behavioural and genetic causal beliefs can exist independently as some research suggests that the endorsement of one may not affect the relative contributions of the other (Condit et al., 2009; Murphy et al., 2005; O'Neill et al., 2010; Sanderson, Waller, Humphries, & Wardle, 2011; Sanderson et al., 2013; Wang & Coups, 2010), and that in some cases, holding genetic causal beliefs increases the likelihood that the individual will also endorse behavioural causal beliefs (O'Neill et al., 2010; Sanderson et al., 2011; 2013). The present study will attempt to elucidate the roles of both genetic and behavioural causal beliefs on attempted behaviour change by employing a unique set of items assessing these constructs.

In addition, it may also be possible that individuals who highly endorse behavioural causal beliefs but not genetic causal beliefs are more likely to engage in preventive behaviours than those who highly endorse genetic causal beliefs but not behavioural causal beliefs. However, it is unclear whether individuals who endorse both behavioural and genetic causal beliefs are more or less likely to engage in preventive behaviours. It is possible that behavioural causal beliefs may act as a protective factor and buffer the negative effect of genetic causal beliefs on preventive behaviour. To test these relationships, the present study will examine the interaction between behavioural causal beliefs and genetic causal beliefs on attempted behavioural change.

Objectives

The aims of the present study are to (i) examine the prevalence of perceived behavioural and genetic causal beliefs for chronic conditions (i.e. obesity, heart disease, diabetes and cancer); (ii) to examine the association between these causal beliefs and attempts at behaviour change (i.e. physical activity, weight management, fruit and vegetable intake and soda intake).

Design

The data come from the HINTS. The HINTS is a probability-based, nationally representative survey of the US non-institutionalised adult population, aged 18 or older, and follows trends in health-related information and communication and health-related behaviours, perceptions and knowledge (Finney Rutten et al., 2012). The data are crosssectional. The HINTS has fielded three national data collections (HINTS, 2003, 2005 and 2007) with the fourth iteration (HINTS 4) currently ongoing. HINTS 4 is comprised of four separate data collection cycles that began in 2011 and will extend into 2014, using self-administered mail questionnaires that randomly sample from a frame of non-vacant US residential addresses. All analyses in the present paper are conducted on the HINTS 4 (Cycle 2) data which were collected from July 2012 to October 2012. The overall response rate was 40%. More

detailed sampling and methodological information is available elsewhere (Finney Rutten et al., 2012).

Measures

Main outcome measures—Attempted behaviour change. Self-report measures of attempted behaviour change included whether respondents made *intentional* changes/maintenance in five domains: physical activity, weight management, vegetable intake, fruit intake and soda intake which are associated with chronic disease prevention. To assess change, participants read items that asked ‘At any time in the past year, have you intentionally tried to ...’ and chose responses relevant to the specific domain. Attempted behavioural change was assessed for *physical activity* (increase the amount of exercise you get in a typical week/maintain the amount of exercise you get in a typical week/you haven’t really paid much attention to the amount of exercise you get), *weight management* (lose weight/ maintain weight/gain weight/you haven’t really paid attention), *fruit intake* (increase the amount of fruit you eat or drink/maintain the same amount of fruit you eat or drink/you haven’t really paid attention to the amount of fruit you eat or drink each day), vegetable intake (increase the amount of vegetables you eat or drink/maintain the same amount of vegetables you eat or drink/you haven’t really paid attention to the amount of vegetables you eat or drink each day) and *soda intake* (decrease the amount of regular soda or pop you usually drink in a week/maintain the same amount of regular soda or pop you usually drink a week/you haven’t really paid attention to the amount of regular soda or pop you usually drink in a week).

Covariates—Analyses also included known covariates associated with intentions to change health behaviours, notably demographic variables and current health behaviour.

Demographic variables included age (measured in years), body mass index (BMI), gender, race/ethnicity and educational attainment. Current reported daily fruit and vegetable intake was captured by two items, one each for fruit and vegetable intake: ‘About how many cups of fruit [vegetables] (including 100% pure fruit[vegetable] juice) do you eat or drink each day?’ Weekly soda intake was assessed with one item, ‘Not counting any diet soda or pop, about how often do you drink regular soda or pop in a typical week?’ Current reported physical activity was assessed as a summary value comprised of two items, ‘In a typical week, how many days do you do any physical activity or exercise of at least moderate intensity?’ and ‘On the days that you do any physical activity or exercise of at least moderate intensity, how long do you typically do these activities?’.

Behavioural causal beliefs for chronic conditions—Participants were asked to respond to items regarding their beliefs about the role of health behaviour in developing common chronic diseases. Participants were asked ‘How much do you think health behaviors like diet, exercise and smoking determine whether or not a person will develop each of the following conditions?’ Response options included: *not at all*, *a little*, *somewhat* and *a lot*. Respondents answered these items with respect to four conditions: obesity, heart disease, diabetes and cancer.

Genetic causal beliefs for chronic conditions—Participants were asked to respond to items regarding their beliefs about the role of genetics in developing common chronic diseases. Participants were asked ‘How much do you think genetics, that is characteristics passed from one generation to the next, determine whether or not a person will develop each of the following conditions?’ Response options included: *not at all*, *a little*, *somewhat* and *a lot*. Respondents answered these items with respect to four conditions: obesity, heart disease, diabetes and cancer.

Interaction effects between genetic causal beliefs and genetic causal beliefs for chronic conditions—Higher order effects were created to examine the interaction between behavioural causal beliefs and genetic causal beliefs (behavioural causal beliefs X genetic causal beliefs) with respect to four conditions: obesity, heart disease, diabetes and cancer.

Analyses

All analyses were conducted using SAS 9.3 and SAS-callable SUDAAN 10.0 statistical software to account for the complex sampling design of HINTS. Survey weights (final survey weights and replicate weights) were used to obtain population-level point estimates and accurate variance estimates. All *p*-values reported are for two-tailed tests and a value of $< .05$ was considered statistically significant. Chi-square goodness-of-fit tests were conducted to test whether observed proportions for responses in behavioural causal belief items differed significantly from proportions for responses in genetic causal beliefs.

Weighted multinomial logistic regression models were conducted to examine the associations of behavioural *and* genetic causal beliefs across four separate medical conditions/chronic diseases (i.e. obesity, heart disease, diabetes and cancer) on self-reported behaviour change. We also examined the interaction between behavioural and genetic causal beliefs on behaviour change. The response ‘haven’t paid attention ...’ served as the reference for all outcome variables as indicated in Tables 3–6. Although we test for *all* possible contrast effects among the different categories, we chose to display the reference group that offered the most information in the Tables for ease of reading. We report *all* other significant group contrasts that are not found in the Tables in the text of the Results section. Odds ratios and the respective 95% confidence intervals are reported for each of the four levels of perceptions of the role of behaviour and genetics for each medical condition. All analyses were weighted to obtain population estimates. We only display results for behavioural changes in weight management, physical activity and vegetable intake as findings for fruit intake and soda consumption were non-significant.

We had originally stratified all analyses for previous diagnosis of chronic conditions (e.g. cancer, diabetes, heart disease and obesity). Due to a limited sample of those with a diagnosis, the analyses were underpowered. As a result, we collapsed across previous diagnosis. We then controlled for previous diagnosis of chronic conditions though all failed to be significantly associated with behavioural change with the exception of obesity status. As a result, we control for BMI in all analyses.

Results

The HINTS 4 adult sample¹ was comprised of 1384 (50%) males and 2023 (50%) females. With regard to race/ethnicity, there were 2184 (68%) non-Hispanic Whites, 614 (13%) non-Hispanic Blacks, 132 (5%) Asians, 27 (1%) American Indians or Alaskan Natives and 456 (12%) Hispanic respondents. Refer to Table 1 for other respondent characteristics.

Prevalence of behavioural and genetic causal beliefs for chronic disease

Results indicated that participants held both behavioural and genetic causal beliefs for all four chronic conditions (refer to Figure 1). Overall, participants rated behaviours as being *more* important than genetics in determining chronic conditions (refer to Table 2).

Multinomial logistic regression

Neither behaviour nor genetic causal beliefs were significantly associated with changes in soda or fruit consumption. As a result, we do not provide further detail on those models in Tables 3–6. In addition, higher order effects that examined the interaction between behavioural and genetic causal beliefs did not significantly improve overall model variance, as a result, we omit interaction effects and only present results of the models with lower order effects.

Behavioural and genetic causal beliefs for obesity—Behavioural causal beliefs for obesity were significantly associated with reported attempts to increase exercise *and* to increase vegetable intake. Those who answered ‘a little’ were less likely than those who answered ‘somewhat’ to have intentionally increased exercise. Those who answered ‘a little’ and ‘somewhat’ were less likely to have intentionally increased exercise and vegetable intake in comparison to those who answered ‘a lot’. Overall, genetic causal beliefs for obesity were not significantly associated with attempted behaviour change (refer to Table 3).

Behavioural and genetic causal beliefs for heart disease—Behavioural causal beliefs for heart disease was significantly associated with reported attempts to increase exercise. Those who answered ‘a little’ were less likely to have intentionally increased exercise in comparison to those who answered ‘a lot’. Behavioural causal belief for heart disease was significantly associated with reported attempts to increase vegetable intake. Those who answered ‘somewhat’ were less likely to have intentionally increase vegetable intake in comparison to those who answered ‘a lot’. Overall, genetic causal belief for heart disease was not significantly associated with attempted behaviour change (refer to Table 4).

Behavioural and genetic causal beliefs for diabetes—Behavioural causal beliefs for diabetes were significantly associated with reported attempts to lose weight. Specifically, those who answered that behaviour ‘somewhat’ contributed to diabetes were less likely to report having intentionally tried to lose weight in the past year compared to those who answered that behaviour contributes ‘a lot’ to diabetes. Behavioural causal beliefs for diabetes were significantly associated with reported attempts to increase exercise *and* to

Note

1.Counts are unweighted, while proportions are weighted.

increase vegetable intake. Those who answered ‘a little’ and ‘somewhat’ were less likely to have intentionally increased exercise *and* vegetable intake in comparison to those who answered ‘a lot’. Overall, genetic causal beliefs for diabetes were not significantly associated with attempted behaviour change (refer to Table 5).

Behavioural and genetic causal beliefs for cancer—Genetic causal beliefs for cancer were significantly associated with reported attempts to maintain weight. Those who answered genetics contribute ‘a little’ and ‘a lot’ to cancer were more likely to have attempted to intentionally maintain weight in the past year compared to those who answered that genetics contributes ‘somewhat’ to cancer. Overall, behavioural causal beliefs for cancer were not significantly associated with attempted behaviour change (refer to Table 6).

Discussion

The aims of the present study were twofold. We were interested in examining the prevalence of perceived behavioural and genetic causal beliefs for chronic conditions across different chronic conditions (i.e. obesity, heart disease, diabetes and cancer). In addition, we were interested in studying the association between these causal beliefs and attempts at behaviour change in across multiple domains (i.e. physical activity, weight management, fruit and vegetable intake and soda intake). In addressing the first aim, our findings indicated that participants held both behavioural and genetic causal beliefs for chronic diseases. Our findings are in agreement with other research that suggests that the public has a nuanced understanding of health and illness and possesses multifactorial theories of the etiology of chronic illness (Condit et al., 2009; Murphy et al., 2005; O’Neill et al., 2010; Waters et al., 2014). Previous studies indicate that individuals who endorse genetic causal beliefs for chronic diseases were likely to also endorse behavioural causal beliefs for cancer (Sanderson et al., 2011) and for obesity (Sanderson et al., 2013). With rapid development in genomics research, there is concern that belief in genetic causes of disease may lead to diminished perceived importance of lifestyle and behaviours (Marteau & Weinman, 2006). Our findings provide evidence that behavioural and genetic causal beliefs do not constitute opposite ends of a continuum, suggesting that the public has a more complex and nuanced understanding of the etiology of chronic diseases examined in this study than previously assumed.

In addressing the second aim of the study, our findings provide support that behavioural causal beliefs are associated with behavioural change, confirming a study by Wang and Coups (2010) that found that within the 2007 adult HINTS sample, endorsement of behavioural causal beliefs were associated with reported levels of physical activity. However, our findings extend current understanding of the role of health cognitions by shedding light on how disease-specific behavioural causal beliefs are associated with different health behaviours. For example, behavioural causal beliefs for obesity, heart disease and diabetes were related to behavioural changes in exercise and vegetable intake; however, we failed to reproduce the same association for behavioural causal beliefs for cancer. These findings highlight the importance of specificity in the measurement of health cognitions and risk perception as our findings suggest that respondents were more likely to associate vegetable intake (but not soda intake) with heart disease (but not cancer).

Our study findings indicated that behavioural causal beliefs for obesity, heart disease and diabetes were related to some behavioural outcomes (i.e. exercise and vegetable intake) but not others (i.e. fruit or soda consumption). In addition, only genetic causal beliefs for cancer were associated with health behaviour. These findings suggest that causal beliefs are important to consider when communicating messages about disease prevention and risk factors as causal beliefs appear to vary across chronic diseases. While less than a quarter of respondents in one study believed that genetics were important in determining obesity (Wang & Coups, 2010), more than 75% of women in another study believed that genetics were important in determining breast and colorectal cancer (Wang, Miller, Egleston, Hay, & Weinberg, 2010). Causal attributions about behavioural and genetic factors are disease-specific and may have important implications for how genetic risk information affects how individuals process and react to the information (Wang & Coups, 2010). Those who endorse genetic causal beliefs for cancer may be less likely to engage in smoking cessation behaviours as demonstrated by one study found that smokers who believed that genes are the main cause of lung cancer were significantly more likely to be current smokers (Kaphingst, Lachance, & Condit, 2009).

Unlike our models for diabetes, obesity and heart disease, behavioural causal beliefs for cancer were not significantly associated with behaviour change. We offer caution in over-interpreting these results. In HINTS 4 (Cycle 2), causal beliefs were assessed for general cancer than for specific cancer sites. Because cancer is a heterogeneous disease with many different subtypes, it is possible that this particular item activated thoughts about different subtypes of cancer for different respondents. Findings from the HINTS 2005 showed that beliefs about the role of lifestyle and behaviour varied across cancer sites as respondents were less likely to endorse behavioural causal beliefs for colorectal cancer as to other cancers sites that included skin or lung, suggesting that some modifiable risk factors (e.g. sun protection or smoking) are perceived to be more influential for cancer etiology. Also, significant differences in causal attributions between cancer sites were noted by Wang et al. (2010) as respondents were more likely to endorse heredity and pollution as a cause of breast cancer while endorsing diet, aging, alcohol and lack of exercise as a cause of colorectal cancer. It is possible that the existence of various cancer subtypes may be masking differences among these relationships and there may be too much noise to detect meaningful effects. Future research should follow up disaggregated analyses for specific cancer subtypes.

Our study failed to find significant interactions between behavioural causal beliefs and genetic causal beliefs. We believe this may have been likely due to issues relating to power. In examining cross-tabulations of behavioural and genetic causal beliefs, the majority of respondents highly endorsed both behavioural and genetic causal beliefs, while fewer endorsed either the role of behaviour or the role genetics, and even fewer endorsed neither behavioural or genetic causal beliefs. In addition, our study focused on outcomes of primary prevention (e.g. diet and exercise). Future research should also examine the role of causal beliefs on other types of health behaviours such as cancer screening, where focus may shed light on the public's understanding of secondary prevention and disease risk. Additional research is also needed to explore potential additive effects endorsing multiple behavioural

causal beliefs. It is possible that possessing behavioural causal beliefs across multiple chronic conditions would lead to greater behavioural changes.

Lastly, future studies should also examine the role of other health cognitions such as the perceived risk or fatalism as effect modifiers on the relationship between causal beliefs and health behaviours. It is possible that for individuals who perceive themselves to be at high risk for a chronic illness such as cancer, high endorsement of behavioural causal beliefs for cancer may be associated more strongly with preventive health behaviours than in comparison to individuals who perceive themselves to be at low risk.

This study had several limitations. The HINTS is cross-sectional in design and restricts our ability to make causal inferences. Also, single-item measures were used to assess both behaviour and genetic causal beliefs for chronic conditions, limiting the reliability for assessing causal beliefs domains. In addition, all behavioural change measures relied on self-report measures which may not be accurate estimates of actual behaviour due to error in recall and biased reporting. However, using a self-administered survey – where answering in socially desirable ways will be less salient– suggests that this issue may not be as important here. Future research should incorporate the use of longitudinal data in order to clarify the causal pathways between behavioural and genetic attributions and behavioural change. In addition, the population-based data are weighted to reflect the US adult population. As a result, we draw from a large population which may capture statistically significant but small effect sizes.

Conclusion

The public possess a multifactorial understanding of the etiology of chronic illness. Behavioural causal beliefs are associated with behavioural change; however, measurement must capture disease-specific behavioural causal beliefs as they are associated with different health behaviours. Additional research is needed to examine the potential interaction between behavioural and genetic causal beliefs and how they may impact preventive behaviours.

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Figure 1.
Prevalence of casual beliefs for chronic conditions.

Table 1.

Demographic traits of the HINTS 4 adult sample.

Trait	N (%)[*]
<i>Gender</i>	
Male	1384 (50.16%)
Female	2023 (49.83%)
<i>Race/ethnicity</i>	
Non-Hispanic White	2183 (68.42%)
Black	614 (12.75%)
Asian	132 (5.40%)
American Indian or Alaskan Native	27 (1.02%)
Hispanics	456 (12.42%)
<i>Educational attainment</i>	
Less than high school	329 (13.51%)
High school diploma/GED equivalent	775 (20.29%)
Some college	1057 (37.57%)
College degree +	1380 (28.64%)
<i>Age (years)</i>	
18–24	105 (12.91%)
25–34	424 (17.63%)
35–49	845 (26.44%)
50+	2138 (43.01%)
<i>Marital status</i>	
Married/living with partner	1857 (57.2%)
Divorced/separated/widowed	1043 (15.7%)
Never married/single	628 (27.2%)
<i>Annual household income</i>	
<\$15,000	536 (16.38%)
\$15,000–34,999	705 (20.39%)
\$35,000–74,999	983 (32.25%)
\$75,000–99,999	376 (12.13%)
\$100,000–199,999	427 (15.00%)
\$200,000+	123 (3.84%)
<i>Health insurance</i>	
Yes	2999 (82.18%)
No	584 (17.82%)

^{*} Counts are unweighted while proportions are weighted.

Table 2.

Causal beliefs across chronic conditions.

	Behaviours (%)	Genetics (%)	<i>p</i> -value*
<i>Causal beliefs for diabetes</i>			
Not at all	2.29	2.27	<.001
A little	6.32	11.78	
Somewhat	22.18	43.25	
A lot	69.22	42.70	
<i>Causal beliefs for obesity</i>			
Not at all	2.70	6.78	<.001
A little	4.33	22.91	
Somewhat	15.59	41.59	
A lot	77.38	28.72	
<i>Causal beliefs for heart disease</i>			
Not at all	2.21	1.98	<.001
A little	7.05	10.93	
Somewhat	26.51	44.16	
A lot	64.23	42.93	
<i>Causal beliefs for cancer</i>			
Not at all	4.47	3.93	<.001
A little	16.99	14.91	
Somewhat	34.22	40.89	
A lot	44.33	40.27	

*
Tests for differences in observed proportions.

Table 3.

Causal beliefs for obesity and attempted behavioural change.

Weight management ^a						
REF = Haven't paid much attention (n = 1638)						
	Decrease (n = 1773)		Maintain (n = 816)		p-value	F
	OR	95% CI OR	OR	95% CI OR		
						68.37**
<i>Behaviour beliefs</i>					.41	
Not at all	.82	.25, 2.70	.74	.14, 3.84		
A little	.58	.22, 1.56	.73	.23, 2.29		
Somewhat	.53	.29, .97	.56	.32, .99		
A lot (REF)	1.00	-	1.00	-		
<i>Genetic beliefs</i>					.33	
Not at all (REF)	2.22	.90, 5.43	1.39	.57, 3.33		
A little	1.24	.76, 2.04	1.43	.81, 2.52		
Somewhat	1.29	.84, 1.99	1.06	.67, 1.67		
A lot (REF)	1.00	-	1.00	-		
Physical activity ^a						
REF=Haven't paid much attention (n=963)						
	Increase (n=1773)		Maintain (n=763)		p-value	F
	OR	95% CI OR	OR	95% CI OR		
						65.49**
<i>Behaviour beliefs</i>					.001	
Not at all	1.33	.38, 4.68	2.44	.44, 13.51		
A little	.18	.08, .41	.49	.12, 2.02		
Somewhat	.47	.31, .72	.72	.45, 1.14		
A lot (REF)	1.00	-	1.00	-		
<i>Genetic beliefs</i>					.77	
Not at all	1.36	.72, 2.59	1.29	.56, 2.99		
A little	1.41	.93, 2.13	1.09	.60, 2.00		
Somewhat	1.25	.79, 1.96	1.08	.63, 1.84		
A lot (REF)	1.00	-	1.00	-		
Vegetable intake ^a						
REF=Haven't paid much attention (n=1081)						
	Increase (n=1402)		Maintain (n=989)		p-value	F
	OR	95% CI OR	OR	95% CI OR		
						60.44**
<i>Behaviour beliefs</i>					.02	
Not at all	.51	.15, 1.79	4.16	.64, 27.03		
A little	.33	.11, .97	.95	.48, 1.89		
Somewhat	.60	.60, .41	1.04	.64, 1.70		
A lot (REF)	1.00	-	1.00	-		

Weight management ^a						
REF = Haven't paid much attention (n = 1638)						
	Decrease (n = 1773)		Maintain (n = 816)		p-value	F
	OR	95% CI OR	OR	95% CI OR		
<i>Genetic beliefs</i>					.60	
Not at all	1.00	.45, 2.23	.62	.25, 1.56		
A little	0.97	.65, 1.45	.81	.54, 1.23		
Somewhat	1.00	.70, 1.42	.72	.48, 1.10		
A lot (REF)	1.00	-	1.00	-		

^a controlling for age, BMI, gender, education, race and weekly soda intake.

^{**} Significant at p .01.

^{*} Significant at p .05.

Table 4.

Causal beliefs for heart disease and attempted behavioural change.

Weight management ^a						
REF = Haven't paid much attention (n = 1638)						
	Decrease (n = 1773)		Maintain (n = 816)		p-value	F
	OR	95% CI OR	OR	95% CI OR		
						64.24 **
<i>Behaviour beliefs</i>					.23	
Not at all (REF)	1.00	-	1.00	-		
A little	.79	0.6,10.59	1.26	0.6,26.40		
Somewhat	0.94	.07, 11.74	2.32	.10, 51.93		
A lot	1.52	.12, 19.29	2.99	.14, 64.46		
<i>Genetic beliefs</i>					.54	
Not at all (REF)	1.00	-	1.00	-		
A little	.36	.04,3.15	.023	.03,1.70		
Somewhat	.39	.04,3.41	.18	.02,1.60		
A lot	.32	.04,2.84	.15	.02,1.37		
Physical activity ^a						
REF=Haven't paid much attention (n=963)						
	Increase (n=1773)		Maintain (n=763)		p-value	F
	OR	95% CI OR	OR	95% CI OR		
						99.08 **
<i>Behaviour beliefs</i>					.02	
Not at all	.70	.11,.4.40	3.23	.46,22.95		
A little	.36	.16,.80	.78	.33,1.83		
Somewhat	.72	.49,1.06	.64	.42, .96		
A lot (REF)	1.00	-	1.00	-		
<i>Genetic beliefs</i>					.95	
Not at all	1.61	.57,4.56	.90	.20, 3.97		
A little	.91	.56,1.49	1.05	.60, 1.84		
Somewhat	1.08	.74,1.57	1.12	.73, 1.71		
A lot (REF)	1.00	-	1.00	-		
Vegetable intake ^a						
REF=Haven't paid much attention (n=1081)						
	Increase (n=1402)		Maintain (n=989)		p-value	F
	OR	95% CI OR	OR	95% CI OR		
						11.84 **
<i>Behaviour beliefs</i>					.02	
Not at all	.45	.09, 2.32	3.06	.07, 133.52		
A little	.54	.25,1.14	.85	.42, 1.71		
Somewhat	.56	.42, .76	.80	.59,1.08		
A lot (REF)	1.00	-	1.00	-		

Weight management ^a						
REF = Haven't paid much attention (n = 1638)						
	Decrease (n = 1773)		Maintain (n = 816)		p-value	F
	OR	95% CI OR	OR	95% CI OR		
<i>Genetic beliefs</i>					.95	
Not at all	.99	.15, 6.54	1.13	.11, 11.95		
A little	1.03	.61, 1.73	1.00	.48, 2.08		
Somewhat	1.13	.84, 1.53	.92	.64, 1.31		
A lot (REF)	1.00	-	1.00	-		

^a controlling for age, BMI, gender, education, race and weekly soda intake.

** Significant at $p = .01$.

* Significant at $p = .05$.

Table 5.

Causal beliefs for diabetes and attempted behavioural change.

Weight management ^a						
REF = Haven't paid much attention (n = 1638)						
	Decrease (n = 1773)		Maintain (n = 816)		p-value	F
	OR	95% CI OR	OR	95% CI OR		
						59.54**
<i>Behaviour beliefs</i>					.05	
Not at all (REF)	.44	.07, 2.91	.61	.08, 4.47		
A little	.42	.17, 1.00	.59	.23, 1.48		
Somewhat	.50	.33, .77	.56	.29, 1.06		
A lot	1.00	-	1.00	-		
<i>Genetic beliefs</i>					.50	
Not at all (REF)	2.11	.55, 8.03	2.46	.67, 9.09		
A little	1.56	.83, 2.94	1.64	.85, 3.15		
Somewhat	1.16	.86, 1.59	.97	.68, 1.37		
A lot	1.00	-	1.00	-		
Physical activity ^a						
REF=Haven't paid much attention (n=963)						
	Increase (n=1773)		Maintain (n=763)		p-value	F
	OR	95% CI OR	OR	95% CI OR		
						74.33**
<i>Behaviour beliefs</i>					.002	
Not at all	1.22	.31, 4.83	4.54	.90, 22.81		
A little	.38	.19, .77	.70	.30, 1.65		
Somewhat	.50	.34, .74	.58	.38, .91		
A lot (REF)	1.00	-	1.00	-		
<i>Genetic beliefs</i>					.57	
Not at all	1.05	.39, 2.83	.68	.24, 1.98		
A little	1.21	.73, 2.00	1.12	.60, 2.11		
Somewhat	1.24	.92, 1.67	1.21	.84, 1.76		
A lot (REF)	1.00	-	1.00	-		
Vegetable intake ^a						
REF=Haven't paid much attention (n=1081)						
	Increase (n=1402)		Maintain (n=989)		p-value	F
	OR	95% CI OR	OR	95% CI OR		
						34.10**
<i>Behaviour beliefs</i>					.002	
Not at all	.81	.19, 3.52	3.53	.19, 64.01		
A little	.29	.13, .63	.80	.37, 1.71		
Somewhat	.49	.34, .72	.68	.48, .96		
A lot (REF)	1.00	-	1.00	-		

Weight management ^a						
REF = Haven't paid much attention (n = 1638)						
Decrease (n = 1773)		Maintain (n = 816)		p-value	F	
OR	95% CI OR	OR	95% CI OR			
<i>Genetic beliefs</i>						.15
Not at all	.89	.33, 2.39	1.15	.29, 4.54		
A little	2.08	1.10, 3.94	1.52	.85, 2.72		
Somewhat	1.25	.95, 1.64	1.01	.72, 1.43		
A lot (REF)	1.00	-	1.00	-		

^acontrolling for age, BMI, gender, education, race and weekly soda intake.

** Significant at $p = .01$.

* Significant at $p = .05$.

Table 6.

Causal beliefs for cancer and attempted behavioural change.

Weight management ^a						
REF = Haven't paid much attention (n = 1638)						
	Decrease (n = 1773)		Maintain (n = 816)		p-value	F
	OR	95% CI OR	OR	95% CI OR		
						82.22 **
<i>Behaviour beliefs</i>					.56	
Not at all (REF)	1.00	-	1.00	-		
A little	.75	.20, 2.76	1.12	.19, 6.64		
Somewhat	.65	.21, 2.07	1.10	.19, 6.29		
A lot	.93	.28, 3.08	1.41	.26, 7.58		
<i>Genetic beliefs</i>					.05	
Not at all	1.05	.36, 3.05	1.26	.38, 4.17		
A little	1.20	.76, 1.89	2.25	1.25, 4.07		
Somewhat (REF)	1.00	-	1.00	-		
A lot	1.16	.78, 1.71	1.56	1.05, 2.32		
Physical activity ^a						
REF=Haven't paid much attention (n=963)						
	Increase (n=1773)		Maintain (n=763)		p-value	F
	OR	95% CI OR	OR	95% CI OR		
						83.30 **
<i>Behaviour beliefs</i>					.13	
Not at all (REF)	1.00	-	1.00	-		
A little	.61	.24, 1.54	.30	.09, .94		
Somewhat	.66	.28, 1.56	.26	.09, .79		
A lot	.91	.39, 2.16	.32	.11, .96		
<i>Genetic beliefs</i>					.84	
Not at all (REF)	1.00	-	1.00	-		
A little	.77	.33, 1.78	1.21	.40, 3.64		
Somewhat	.80	.39, 1.62	1.43	.57, 3.59		
A lot	.91	.47, 1.74	1.55	.63, 3.84		
Vegetable intake ^a						
REF=Haven't paid much attention (n=1081)						
	Increase (n=1402)		Maintain (n=989)		p-value	F
	OR	95% CI OR	OR	95% CI OR		
						23.39 **
<i>Behaviour beliefs</i>					.43	
Not at all (REF)	1.00	-	1.00	-		
A little	1.27	.52, 3.11	.60	.12, 3.06		
Somewhat	1.44	.60, 3.45	.74	.16, 3.34		
A lot	1.73	.75, 3.98	.64	.14, 2.97		

Weight management ^a						
REF = Haven't paid much attention (<i>n</i> = 1638)						
	Decrease (<i>n</i> = 1773)		Maintain (<i>n</i> = 816)		<i>p</i> -value	<i>F</i>
	OR	95% CI OR	OR	95% CI OR		
<i>Genetic beliefs</i>					.18	
Not at all (REF)	1.00	-	1.00	-		
A little	1.80	.61, 5.36	.77	.26, 2.29		
Somewhat	1.45	.60, 3.46	.68	.27, 1.75		
A lot	1.90	.79, 4.58	.96	.35, 2.61		

^a controlling for age, BMI, gender, education, race and weekly soda intake.

** Significant at *p* .01.

* Significant at *p* .05.