

Public Awareness of Genetic Influence on Chronic Disease Risk: Are Genetic and Lifestyle Causal Beliefs Compatible?

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Key Words

Public awareness · Genetics · Open-ended questions

Abstract

Background/Aims: There is concern that raising awareness about the role of genetics in chronic disease etiology could undermine public belief that lifestyles are important, leading to adverse effects on public health. We tested the hypothesis that people who believe genetics influence chronic disease risk are less likely to believe lifestyles play a role.

Methods: Open-ended questions about cancer and heart disease risk factors were included in a population-based survey of 1,747 British adults. Responses were coded for causal beliefs about genetics and lifestyle (smoking, diet, alcohol, exercise). **Results:** One third of the respondents identified genetic factors as influencing cancer (35%) and heart disease (36%) risk. Identifying genetic risk was associated with female gender, older age and education for both diseases, as well as with family history for heart disease. Individuals identifying genetic influences on cancer risk were more likely to identify diet ($p < 0.001$) and exercise ($p < 0.05$), and mentioned more lifestyle factors overall ($p < 0.05$), independent of demographics and family history. Patterns were similar for heart disease. **Conclusion:** People who recognize that ge-

netics influence chronic disease risk appear more, not less, likely to recognize the role of lifestyles, contradicting suggestions that the public takes an 'either/or' view of the etiology of these potentially preventable diseases.

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Introduction

Information about chronic disease genetics is being increasingly disseminated to the public via the media [1]. Public reactions have tended to be broadly positive, with people expressing more optimism than concern about future medical applications arising from the Human Genome Project [2]. Establishing public acceptance of the role of genetics in chronic disease causation is important if findings from genetics research are to be translated into public health benefit. However, there is also concern about the increasing public focus on genetics of chronic diseases at a societal level because it may reduce the focus on other possibilities for improving public health, such as addressing differences in social structure and environment [3].

At an individual level, efforts to encourage people to be aware of the role of genetics in chronic disease causa-

tion may inadvertently reduce the focus on other disease prevention factors such as lifestyles. This concern is supported to some extent by models of health behavior and coping. The common sense model of self-regulation in health and illness (CSM) [4] is the most widely used model to explain how people interpret and cope with current and potential health events or threats. The CSM posits that individuals facing a health threat go through several stages, including active processing of cognitive representations of the health threat (including personal ideas about disease etiology or causal beliefs) and using the representations formed to steer the development of action plans for coping with the problem [5]. The CSM explicitly states that people's cognitive representations of disease (including causal beliefs) directly influence the coping strategies they select to reduce the disease threat [4]. Confidence in the ability of a given intervention or behavior to reduce disease risk has been labeled 'perceived response-efficacy' [6, 7] or 'outcome expectancies' [8], and is widely acknowledged to be one of the key cognitive predictors of behavior change [9]. The CSM suggests a direct relationship between causal beliefs and perceived response efficacy. For example, it suggests that if people believe that heart disease is caused by lack of exercise, then they will believe that increasing exercise levels will reduce heart disease risk; whereas, if people believe that genes cause heart disease, then they may believe there is nothing that can be done or that biologically based interventions are likely to reduce their risk of heart disease [10].

Implicit in these assertions is the idea that people hold either behavioral or genetic causal beliefs about disease. This would suggest that acceptance of genetic etiology could replace behavioral etiology, with potentially adverse effects on lifestyle choices and, therefore, health [10]. These concerns were supported to some extent by one early study which indicated that people who held stronger genetic causal beliefs about relatively common conditions, such as hypercholesterolemia, held weaker behavioral causal beliefs [11]. However, a subsequent focus group study indicated that people interpreted 'a gene for heart disease' to mean that there were both genetic and environmental causes of the disease [12]. Similar results were found in a study using network analysis, which found that people held complex mental models of heart attacks including both genetic and lifestyle factors [13]. Given these mixed findings, uncertainty remains about whether dissemination of genomic knowledge about chronic diseases, such as heart disease and cancer, will reduce public focus on the role of lifestyle causes of these diseases.

In the present study, our first aim was to examine and compare the proportions of people in the general population who reported, unprompted (i.e. using open-ended questions), that genetics are a causal factor for heart disease and cancer in a large-scale, population-based survey of British adults. We also examined the demographic characteristics of people who identified, versus those who did not, a genetic influence on heart disease and cancer to see whether the same patterns of associations emerged for both diseases. Our second aim was to assess the association between belief in the genetic etiology of heart disease and cancer and awareness of lifestyle causes in order to explore the hypothesis that people who hold genetic causal beliefs are less likely to hold lifestyle (diet, exercise, smoking, alcohol) causal beliefs. We have previously reported the proportions of respondents holding each of the four lifestyle causal beliefs individually, and the demographic associations with lifestyle causal beliefs, elsewhere [14].

Methods

Design and Sample

Data were collected as part of an Office of National Statistics Omnibus Survey. The Omnibus Survey is a monthly, multipurpose survey for use by government and nonprofit making organizations, which uses stratified random probability sampling to select households for a home visit. Previous Office of National Statistics Omnibus Surveys have been used to address a range of health-related issues, including obesity [15], oral health [16] and health status [17]. For the present study, we included a series of questions in the September 2002 Omnibus Survey. Of the 3,000 addresses selected in that month, 224 (7%) were ineligible on the basis that they were not occupied homes. From 2,776 eligible addresses, 586 (21%) people did not wish to take part and 230 (8%) were uncontactable, resulting in a response rate of 71%. People were also excluded if they were over 75 years, which led to the exclusion of 213 (11%) individuals and a final sample size of 1,747.

Measures

Demographic and Personal Characteristics. Measures included age, gender, race/ethnicity and educational attainment ['degree or equivalent', 'A-levels or equivalent', 'GCSE (general certificate of secondary education) or equivalent', and 'no formal qualifications']. Family history of cancer and heart disease were assessed with the question: 'Do any of your close family members have, or have had, cancer (heart disease)? Include immediate blood relatives only, not cousins or spouse/partner' (response options 'yes' and 'no'). This item was assumed to measure subjective belief about family history and not to ascertain true family history. Respondents who reported a family history were asked how many relatives with the disease they had, and were categorized according to whether 0, 1 or 2 or more relatives had the disease. Although not explicitly asked, if respondents spontaneously reported they themselves had either disease, the interviewer was briefed not to

ask any further questions about it because they were not trained to deal with any difficult issues that could arise. However, the causal beliefs questions (see below) preceded the family history questions and so were asked of all respondents. Respondents who stated that they had cancer or heart disease were not excluded from the present analyses given that it was likely that the sample included others with one or both of the diseases, but who did not choose to volunteer their personal disease status.

Awareness of Genetic and Lifestyle Risk Factors (or 'Causal Beliefs'). Awareness of genetic and lifestyle risk factors for heart disease was assessed with an open question adapted from previous research [18]: 'What do you think are the things that cause a person to develop heart disease or increase their chances of developing it?' Respondents were encouraged to list as many risk factors as they could. Interviewers were instructed to code each of the respondents' answers according to one of 20 predefined categories. Awareness of genetic and lifestyle risk factors for cancer was assessed with the open question, 'What do you think are the things that cause a person to develop cancer or increase their chances of developing it?', also adapted from Waller et al. [18]. For cancer, interviewers were instructed to code each of the respondents' answers according to one of 26 pre-defined categories. Five categories were used in the present analyses: smoking, drinking alcohol, being physically inactive, eating an unhealthy diet, and genetic (genetics/heredity/family history). See online supplemental text (www.karger.com/doi/10.1159/000294280) for further details regarding the coding procedure.

Statistical Analysis

Binary logistic regression analyses were used to examine the sociodemographic predictors of awareness of genetic risk factors for (1) cancer and (2) heart disease. Initially, univariate models were used to examine the impact of gender, age, education and family history. A multivariate analysis was then used to calculate adjusted odds ratios, controlling for the other variables in the model. This was run both unadjusted and adjusted for gender, age, education and family history. We compared the total number of lifestyle risk factors mentioned between those who did and those who did not mention genetic risk factors using analysis of variance (ANOVA). All *p* values were two-sided; *p* < 0.05 was considered statistically significant.

Results

Sociodemographics

Respondents' ages ranged from 16 to 75 years, 53% were female and 94% were White-British. Almost half (45%) reported they had at least one close family member with heart disease, and just over half (57%) reported at least one close family member with cancer. Table 1 shows the full sociodemographic characteristics of the sample.

Awareness of Genetic Risk Factors

In response to the open-ended questions (table 2), 35% of respondents mentioned genetic factors as influencing

Table 1. Sociodemographic characteristics of the sample

	n	%
Total	1,747	
Gender		
Male	826	47.3
Female	921	52.7
Age		
16–30 years	340	19.5
31–45 years	574	32.9
46–60 years	451	25.8
61–75 years	382	21.9
Race/ethnicity ¹		
White/White-British	1,643	94.3
Black/Black-White Mixed/Black-British	51	2.9
Asian/Asian-White Mixed/Asian-British	49	2.8
Education ²		
No formal qualifications	534	30.6
GCSEs	573	32.8
A-levels	397	22.7
Degree	242	13.9
Family history of cancer ³		
0 close relatives with cancer	727	43.1
1 close relative with cancer	570	33.8
2+ close relatives with cancer	388	23.0
Family history of heart disease ⁴		
0 close relatives with heart disease	905	54.8
1 close relative with heart disease	498	30.2
2+ close relatives with heart disease	247	15.0

¹ Three respondents refused to answer the question, one respondent answered 'don't know'. ² Data missing for 1 respondent. ³ Data missing for 62 respondents. ⁴ Data missing for 97 respondents.

cancer risk, and 36% mentioned genetic factors as influencing heart disease risk (difference not significant).

Awareness of Genetic Risk Factors by Sociodemographics

People were significantly more likely to state that genetic factors influenced cancer risk if they were female, middle-aged and had higher levels of educational attainment. As table 2 shows, these differences were significant in both the unadjusted and adjusted analyses, indicating that effects of gender, age and education were independent of one another. There was no association between having a family history of cancer and being aware of genetic risk factors.

Table 2 shows that people were also significantly more likely to identify genetic factors as influencing heart disease risk if they were female, middle-aged and had higher

Table 2. Awareness of genetic risk factors for cancer and heart disease by sociodemographics

Sociodemographics	Cancer			Heart disease		
	aware of genetic risk factors (n)	unadjusted OR (95% CI)	adjusted OR (95% CI)	aware of genetic risk factors (n)	unadjusted OR (95% CI)	adjusted OR (95% CI)
Gender						
Male	29.2% (241)	1	1	33.2% (274)	1	1
Female	40.0% (368)	1.62 (1.32–1.97)**	1.83 (1.48–2.26)**	38.9% (358)	1.28 (1.05–1.56)*	1.42 (1.14–1.76)*
Age						
16–30 years	25.3% (86)	1	1	24.1% (82)	1	1
31–45 years	38.0% (218)	1.81 (1.34–2.43)**	1.85 (1.36–2.52)**	39.9% (229)	2.09 (1.55–2.82)**	2.06 (1.50–2.82)**
46–60 years	40.8% (184)	2.04 (1.50–2.77)**	2.51 (1.80–3.50)**	41.7% (188)	2.25 (1.65–3.07)**	2.62 (1.86–3.69)**
61–75 years	31.7% (121)	1.37 (0.99–1.90)	1.89 (1.31–2.74)*	34.8% (133)	1.68 (1.21–2.33)*	2.35 (1.61–3.44)**
Education						
No formal qualifications	27.0% (144)	1	1	25.8% (138)	1	1
GCSE	32.3% (185)	1.29 (0.99–1.67)	1.50 (1.11–2.03)*	31.4% (180)	1.31 (1.01–1.71)*	1.68 (1.23–2.28)*
A-levels	38.3% (152)	1.68 (1.27–2.22)**	1.93 (1.42–2.62)**	43.6% (173)	2.22 (1.68–2.92)**	2.82 (2.07–3.86)**
Degree	52.9% (128)	3.04 (2.22–4.17)**	3.67 (2.58–5.22)**	58.3% (141)	4.01 (2.91–5.52)**	5.25 (3.63–7.58)**
Family history						
0 close relatives with disease	33.4% (243)	1	1	31.2% (282)	1	1
1 close relative with disease	34.7% (198)	1.06 (0.84–1.34)	1.01 (0.79–1.28)	37.6% (187)	1.33 (1.06–1.67)*	1.30 (1.02–1.65)*
2+ close relatives with disease	37.9% (147)	1.22 (0.94–1.57)	1.16 (0.89–1.52)	51.8% (128)	2.38 (1.78–3.17)**	2.31 (1.71–3.12)**
Total	34.9% (609)			36.2% (632)		

Adjusted models contain the other sociodemographics (gender/age/education/family history as applicable) as covariates. * $p < 0.05$; ** $p < 0.001$.

levels of educational attainment. Family history was also associated: 52% of people with at least two relatives with heart disease identified genetic factors as influencing heart disease risk, compared with only 31% of those with no family history ($p < 0.001$). This was significant in both unadjusted and adjusted analyses, indicating that the effect was independent of gender, age and education.

Association between Awareness of Lifestyle and Awareness of Genetic Risk Factors

As shown in table 3, people who mentioned genetic influences on cancer risk were significantly more likely to identify diet and exercise as influences on cancer risk than those who did not recognize the role of genetic factors. In addition, people who identified genetic risk factors identified a greater number of lifestyle risk factors overall: the mean (\pm SD) number of lifestyle risk factors identified (range 0–4) was 1.50 (0.90) among those who mentioned genetic risk, compared with 1.32 (0.83) among those who did not recognize genetic influence on cancer risk ($p < 0.001$).

Table 3 shows the same effect for heart disease: people were significantly more likely to be aware of lifestyle (diet and exercise) risk factors if they were aware of genetic risk factors for heart disease. Similarly, the mean number of

lifestyle risk factors identified (range 0–4) was 2.23 (1.09) among those who identified genetic risk, compared with 2.00 (1.10) among those who did not mention genetic influence on heart disease risk ($p < 0.001$).

Discussion

In this population-based study of British adults using open-ended questions, we found that just over a third (35% for cancer and 36% for heart disease) of the respondents spontaneously identified genetic factors (genes/heredity/family history) as influences on risk of cancer and heart disease. These findings are consistent with the results from one previous population-based study in the US in which closed questions were used to explore the extent to which the public recognized the role of genetics in four conditions known to be influenced by both genetic and environmental factors [19]. In that study, half of the respondents (49%) agreed that genes can increase the likelihood of medication side effects, 39% endorsed genetic risk for diseases caused by smoking, 27% for influenza and 23% agreed that genes can increase the likelihood of illness in response to environmental exposures such as pesticides. Although previous surveys of public aware-

Table 3. Awareness of lifestyle risk factors for cancer and heart disease by awareness of genetic risk factors

Awareness of lifestyle risk factors	Cancer			
	aware of genetic risk factors for cancer		logistic regressions/ANOVAs	
	no (n)	yes (n)	unadjusted OR (95% CI)	adjusted OR (95% CI)
Smoking	85.4% (972)	85.1% (518)	0.97 (0.74–1.28)	0.90 (0.67–1.20)
Drinking alcohol	13.7% (156)	15.1% (92)	1.12 (0.85–1.48)	0.98 (0.73–1.31)
Eating an unhealthy diet	27.6% (314)	39.9% (243)	1.74 (1.42–2.15)**	1.51 (1.21–1.89)**
Being physically inactive	5.6% (64)	9.7% (59)	1.80 (1.25–2.60)*	1.63 (1.10–2.42)*
Total number of lifestyle risk factors mentioned (range 0–4), mean (SD)	1.32 (0.83)	1.50 (0.90)	F = 16.44, p < 0.001	F = 6.22, p = 0.013

Adjusted models contain the covariates gender, age, education and family history. * p < 0.05; ** p < 0.001.

ness of risk factors for cancer have been conducted, many of these have focused more on modifiable risk factors and have tended not to focus on respondents' awareness of genetic risk factors [20, 21]. When genetics has been included, endorsement has tended to be very high. For example, Ackermann et al. [22] found that the majority (93%) of healthy women attending outpatient clinics in Germany rated genetic factors and familial disposition as the most important general risk factor for cervical cancer. Similarly, Peacey et al. [23] found high endorsement of genetics as a risk factor for breast cancer in an international survey of female students: 57% of women were aware of genetic causes overall, and awareness was particularly high in some countries such as the US (94%) and the UK (73%). However, these studies asked people to endorse risk factors from a list, a method which has been shown to produce higher estimates of knowledge than open-ended questions and is likely to overestimate awareness [18]. One previous study used open-ended questions to assess awareness of risk factors for cervical cancer, and found that up to 20% mentioned genetic or hereditary factors [24]. Our study is the first, to our knowledge, to use open-ended questions to assess awareness of genetic risk factors for cancer more generally, and the first to assess awareness of genetic risk factors for heart disease per se in a general population sample. Our finding that 1 in 3 people, without prompting, identified genetic factors as playing a role in cancer and heart disease, indicates that a significant proportion of the public already hold genetic causal beliefs about these serious chronic diseases. Given the expectation that genetics will be increasingly incorporated into general medical care and prevention of chronic diseases, it is arguably encouraging that a third

of the population already readily acknowledges that genetics play a role in these diseases and may therefore be receptive to preventive interventions and treatments that incorporate genetic information.

We found that people who reported a family history of heart disease were significantly more likely to identify genetic risk for heart disease than those without a family history. This is perhaps not surprising given that their personal experience may lead them to be more likely to state that the disease runs in families or to state that the disease is inherited to some extent. Our findings are consistent with clinical studies which have shown that individuals at increased personal or family history-based risk of heart disease have tended to hold stronger genetic causal beliefs about the disease [25–27]. A family history is strongly associated with individual risk and, therefore, people are correct to infer that genes contribute to their heart disease risk. It remains true, however, that risk can still be reduced by behavior change [28–32].

In light of this finding, it was perhaps surprising that there was no association between family history of cancer and awareness of genetic risk factors for cancer. This may be because heart disease is a relatively homogeneous condition, whereas cancer comprises many different conditions (e.g. colon cancer, breast cancer, lung cancer, leukemia). The somewhat broad and basic question assessing family history of cancer in this study (i.e. 0, 1 or 2 or more family members with cancer) may therefore not have been sensitive enough to pick up differences according to familial cancer status. Future studies are needed to see whether this finding is replicated when more rigorous assessments of personal and family history of cancer are used.

Heart disease			
aware of genetic risk factors for heart disease		logistic regressions/ANOVAs	
no (n)	yes (n)	unadjusted OR (95% CI)	adjusted OR (95% CI)
62.7% (699)	65.3% (413)	1.12 (0.92–1.38)	1.01 (0.81–1.26)
24.2% (270)	25.0% (158)	1.04 (0.83–1.31)	1.02 (0.80–1.30)
65.1% (726)	72.3% (457)	1.40 (1.13–1.73)*	1.25 (0.98–1.58)
48.0% (535)	60.4% (382)	1.66 (1.36–2.02)**	1.39 (1.12–1.73)*
2.00 (1.10)	2.23 (1.09)	F = 17.87, p < 0.001	F = 4.79, p = 0.029

We also found that awareness of genetic risk factors for both diseases was lowest in the youngest age group, the 16- to 30-year-olds, which might suggest a need to improve current educational curriculums on genetics. It may be worth targeting genetic educational efforts at young adults, particularly given they are the ones who will be exposed to future genomic developments. The greater awareness of genetic risk factors amongst older adults may also be due to their greater exposure to death and disease in people close to them, and their consequent greater concern about being healthy [33] and seeking out medical information [34]. It will also be important that social disparities in genetics awareness (indicated by the educational attainment association) are addressed to ensure that potential future benefits from genomics technologies do not exacerbate the existing disparities in health between social groups.

A key novel finding in this study was that people who were aware that genetic factors influence cancer and heart disease risk were more, not less, likely than those who were unaware of genetics to recognize that lifestyle factors play a role in these chronic diseases. These findings suggest that people are capable of holding genetic and lifestyle causal beliefs at the same time and that the two sets of beliefs are not incompatible. This may be important because it has previously been hypothesized that if people believe a disease is genetic, then they will behave one way (e.g. take medication), whereas if they believe it is behavioral, then they will behave another way (e.g. increase physical activity) [10]. However, our findings beg the question, what behavioral choices will people make if they hold both genetic and lifestyle causal beliefs about chronic diseases simultaneously? We could not address

this critical question in the present study, but our findings suggest this may be a fruitful direction for future research. It is timely to explore ways to enhance the complexity of lay models of disease to incorporate both genetic and lifestyle risk, and to examine whether and how this influences health behavior downstream.

One related issue is that there has been a tendency to simplify public health messages that are sent out to the population and focus exclusively on lifestyle causes of disease, e.g. ‘cigarette smoke causes oral cancer, gum diseases and tooth loss’ [35]. Yet this of course is not always borne out in an individual’s personal experience, who may, for example, have a close relative who has smoked all their life and never developed cancer [36]. While the concern is that the increasing dissemination of genomic information will undermine the effectiveness of messages which attempt to drum home the lifestyle-disease association, it is also possible that by excluding other risk factors from the health messages, people are in fact more able to reject them. This perspective is inherent in the CSM [4], which posits that persuasive health communications are more likely to be accepted if there is a ‘fit’ between the content of the message and the individual’s pre-existing mental model of the disease. Individuals who hold genetic as well as behavioral beliefs about chronic disease may therefore more readily accept, respond to and act on persuasive health communications which acknowledge the role of genetics in disease causation as well as the role of lifestyle factors. Future research exploring whether some subgroups more readily accept public health messages that incorporate both lifestyle and genetic factors than those that focus on lifestyle factors only would be useful.

Limitations of this study include the wording of the question addressing family history of cancer and heart disease, which may have led to more frequent endorsement than if the question had more explicitly asked for first-degree relatives (siblings, children, parents) only. Also, although some people volunteered the information that they themselves had one of these diseases when responding to the family history questions, a specific question addressing whether they themselves had cancer or heart disease was not included in the survey; as a result, we were unable to assess the associations between personal disease history and awareness of genetic risk factors. Because of limitations on the number of questions feasible to include in the survey, we were not able to answer this or many other interesting research questions (e.g. associations between awareness and perceived disease risk). The measure of causal beliefs was limited because interviewers coded most of the responses at the time of the interview using the coding frame that we supplied. That meant that verbatim responses could not be coded by an additional independent rater to assess interrater reliability. Also, the cross-sectional nature of this study meant that while we could address whether people are more or less likely to hold lifestyle causal beliefs if they hold genetic causal beliefs, we could not address longitudinally whether providing people with genetic information about chronic diseases increases their genetic causal beliefs and reduces their lifestyle causal beliefs. These cross-sectional findings need to be extended with intervention studies to confirm that the association is not due to residual confounding. Finally, this study used a sample that was representative of the UK population in terms of

race and ethnicity, which meant that the majority of respondents were White. The small numbers of people in the non-White categories limits the generalizability of the findings, and meant that we were unable to examine differences according to race and ethnicity. Further research is needed to explore genetic and nongenetic causal beliefs about chronic diseases comparing between different racial and ethnic groups.

In conclusion, our finding that people who hold genetic causal beliefs are more, not less, likely than others to hold lifestyle causal beliefs, is important in public health genomics as we try to understand the impact of dissemination of genomic information in the public domain. Prospective research is now needed to determine whether introducing genomic information to individuals who were previously unaware of it is equally benign, and to find ways to communicate genomic information that maximize positive outcomes and minimize negative outcomes.

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