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Research report

Community attitudes to genetic susceptibility-based mental health interventions for healthy people in a large national sample

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ABSTRACT

Background: Despite an apparent high interest in predictive genetic testing for common multifactorial disorders, few data describe anticipated health behaviour as a consequence of such testing.
Methods: A large population-based public survey with community dwelling adults (N = 1046) ascertained through random digit dialling. Attitudes were assessed via structured interviews.
Results: Intention to start therapies or courses to learn to develop better strategies to cope with stress (80%) was significantly and positively associated with self-estimation of risk for major depressive disorder as higher than average (ß = 0.12, p = 0.001); endorsement of family environment as a causal attribution (ß = 0.11, p < 0.001); and endorsement of gene–environment interaction as a causal mechanism of mental illness (ß = 0.12, p = 0.017). Intention to modify potential life stressors (84%) was significantly and positively associated with self-estimation of risk for depression as higher than average (ß = 0.07, p = 0.029); endorsement of 'abuse' as a causal attribution (ß = 0.10, p = 0.003); and endorsement of 'gene–environment interaction' as a causal mechanism (ß = 0.10, p = 0.002).
Limitations: The hypothetical nature of the genetic risk scenario may have weakened participants' sensitivity to the potential personal impact of such a genetic test result.
Conclusions: Perceptions that modifiable environmental factors strongly contribute to overall risk of major depressive disorder appeared to drive willingness to engage in risk-modifying interventions in the hypothetical scenario of a genetic predisposition. Our results suggest that screening for genetic risk in consort with environmental risk factor assessment has potential community acceptability and clinical value as an early intervention and preventive tool for high risk groups.

1. Introduction

Despite an apparent high interest in predictive genetic testing for susceptibility to common multifactorial disorders amongst individuals with an affected relative [e.g. (Austin et al., 2006; Meiser et al., 2008)] and amongst the general population unselected for disease risk (Cameron et al., 2009; Laegsgaard et al., 2009; Wilde et al., 2011), few data describe anticipated health behaviours as a consequence of such testing. How individuals respond to genetic risk is especially complex when penetrance and predictive power of genotype are uncertain. The issue is further complicated by knowledge that a genetic component only represents part of the risk for

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multifactorial disease and appropriate behavioural responses to environmental risk factors are also required to make health behavioural interventions effective.

Psychiatric genetic epidemiological studies such as those reported by Kendler and Karkowski-Shuman, 1997 have consistently reported significant gene × environmental interactions in the genesis of depression. Caspi et al., 2003 have previously reported that the s/s genotypic variant of the serotonin transporter gene was associated with increased risk for major depressive disorder in interaction with stressful life events. Although the validity of this molecular association remains controversial, with both positive and negative meta-analyses reported (Caspi et al., 2010; Karg et al., 2011; Risch et al., 2009), the overarching premise of increased genetic and environmental risks leading to major depressive disorder formed the rationale for this study. Moreover, our previous study (Wilhelm et al., 2009), found that participants who carried the higher risk (s/s) variant and were provided with this information ranked 'earlier intervention and potential to prevent the onset of depression' as the highest perceived benefit of being provided with their genetic risk status.

The marketing of an increasing range of genetic tests for psychiatric disorders direct-to-consumer (DTC) (Hudson et al., 2007) without medical supervision, raises concerns about the psychosocial impact of risk disclosure and health behavioural outcome of such genetic risk information. Several companies are currently marketing DTC genetic tests for predisposition to major depressive disorder (Genetics and Public Policy Center and Johns Hopkins University, 2010) and for the purposes of predicting individual response to selective serotonin reuptake inhibitor antidepressant (Mayo Clinic, 2006). There is strong evidence that genetic risk information impacts on perception of disease, which in turn has implications for health behaviours that aim to modify environmental risk factors (Senior et al., 2000). It has also been argued that provision of information about individual genetic risk alone may not be sufficient to change health-related behaviour (Javitt, 2006; Lemke, 2004; Marteau and Lerman, 2001).

Using hypothetical genetic susceptibility to major depressive disorder as an example, the present study aims to assess preparedness to modify risk for major depressive disorder at a pre-symptomatic stage through a range of preventive behaviours. This is the first national population study to examine this issue for genetic risk associated with mental health in general. The present study tested the following hypotheses: Willingness to engage in health behaviours that could ameliorate risk for major depressive disorder based on a hypothetical genetic susceptibility will be positively associated with i) a personal history of a mental illness, ii) self-estimation of risk for major depressive disorder as higher than the average person, and iii) endorsement of gene–environment interaction as a causal mechanism for mental illness.

2. Methods

Participants across Australia were recruited by a contracted market research company in May 2008 using random digit dialling of a computer-generated list of landline phone numbers that uses prefixes based on the geographic coverage of the sample's area, with the aim of producing a nationally representative sample. Respondents were selected from each household using a Computer Assisted Telephone Interviewing (CATI)-generated algorithm. Only those aged 18 years or more, and fluent in English were eligible to participate. Only one individual per household could participate. A target sample size of at least 1000 completed CATI interviews was reached. Ethical approval for the study was provided by the relevant Institutional Review Board.

This survey and sample have been previously described in a prior publication by our group (Wilde et al., 2011) which reported community interest in predictive genetic testing for susceptibility to major depressive disorder. In the current paper, we examine the willingness of these participants to engage in health behaviours that could ameliorate risk for major depressive disorder based on such hypothetical genetic susceptibility.

2.1. Demographic characteristics

Data on sex, age, highest level of education achieved and current marital status were collected using specifically designed multiple-choice items.

2.2. Self-estimation of risk for major depressive disorder

Data on self-estimation of risk of depression were collected in a three-part question early in the survey: 'Compared with the average person, would you say your risk of depression is higher than average; lower than average; the same as the average person?'

2.3. Clinical and family history data

Self-reported data on personal history of mental illness or exposure to others' experiences of mental illness through close relatives or close friends were collected on completion of the survey. Participants were asked 'have you' or 'has a close relative or friend ever been diagnosed with depression, bipolar disorder or schizophrenia?' These terms were defined to participants.

2.4. Causal attributions for mental illness

To assess the perceived importance of different factors in causing a mental illness a list of potential contributing factors were derived from Meiser et al., (2007). These were 'genetics'; 'accumulation of daily life stresses'; 'imbalance of chemicals in the brain'; 'major life changes'; 'being in a difficult relationship or marriage'; 'personality factors': 'a difficult or abusive childhood;' 'sexual abuse'; 'recreational drug abuse'; 'family environment'; 'parental behaviour'; Participants were asked: 'how important is…[insert item]… as a cause of mental illness?'

Participants responded to all items using a five-point Likert-type scale ranging from 1 'Not at all important' to 5 'Extremely important'. For statistical analysis, items were grouped according to an exploratory factor analysis which yielded a four factor solution with good internal consistency with item groupings representing (i) genetics, (ii) life stress, (iii) abuse and (iv) family environment (Meiser et al., 2007).

Three items with five-point Likert-type response options ranging from 1 'Strongly disagree', to 5 'Strongly agree' were used to assess endorsement of perceptions about causal

mechanisms for mental illness. Participants were asked "How strongly do you agree or disagree with the following statements?"1 'Mental illnesses are caused by an interplay of genetic risk and stressful life experiences' (gene–environment interactions); 2 'It is possible to have a genetic risk for a mental illness but never actually get the disorder' (incomplete penetrance); and 3 'It is possible to have a mental illness without a genetic risk' (no causal genetic factors).

2.5. Outcome variables

2.5.1. Anticipated health behaviours after learning of having an increased risk for major depressive disorder

Based on the results of the qualitative study reported in Wilde et al. (2009) a range of perceived health behaviours were explored using five-point Likert-type response options ranging from 'Strongly disagree' to 'Strongly agree'. Participants were told, "If you were found, through genetic testing, to have an increased risk for major depressive disorder in the event of stress, how much do you agree or disagree with the following possible changes you might make to your lifestyle?" Five potential health behaviours triggered by being hypothetically identified as having increased risk for major depressive disorder were: 'You would start therapies or courses that would help you learn to develop better strategies to cope with stress'; 'You would modify potential stressors in your life such as stressful job, relationship or domestic situation'; 'You would reduce excessive drug or alcohol use'; 'You would help your children learn how to be more resilient to stress in case they were also at increased risk for major depressive disorder'; and, 'You would decide to not to have children.'

2.6. Statistical analyses

Data were explored initially with descriptive statistics. Bivariate associations between possible predictor variables and outcome variables were first examined using Spearman's rank correlations (r_s) and Mann–Whitney U tests for ordinal predictor variables and Pearson's chi-square cross tabulations for categorical predictors. All variables with a bivariate association with $p<0.1$ were entered into a backward stepwise removal regression model until the only remaining variables were those with $p<0.05$.

The following variables were assessed as possible predictor variables in the analyses of anticipated health behaviours in response to receiving genetic test result that suggests a higher than average hypothetical risk for major depressive disorder: personal history of a mental illness, experience of a mental illness though a close relative or close friend, self-estimation of risk for major depressive disorder, causal attributions for mental illness, gene–environment interaction as a causal mechanism, incomplete penetrance as a hereditary mechanism and no causal genetic factors. All regression analyses were adjusted for age, sex and educational level.

3. Results

Of the 1544 eligible individuals contacted, 498 declined, resulting in 1046 completed surveys and a participation rate of 68%. Detailed sociodemographic characteristics of the 637 (61%) female and 409 (39%) male participants – mean age 50.7 years (range 18–88) years – have been reported in Wilde et al. (2011).

Fig. 1 shows the frequency of endorsement of perceived importance of different causal attributions for mental illness.

Fig. 2 details the proportions of participants who agreed or strongly agreed with a range of anticipated health behaviours in response to receiving a genetic test result that indicates an increased risk for major depressive disorder.

Results from bivariate analyses of factors associated with anticipated health behaviours in the event of receiving a major depressive disorder risk genetic test result are shown in Tables 1a and 1b.

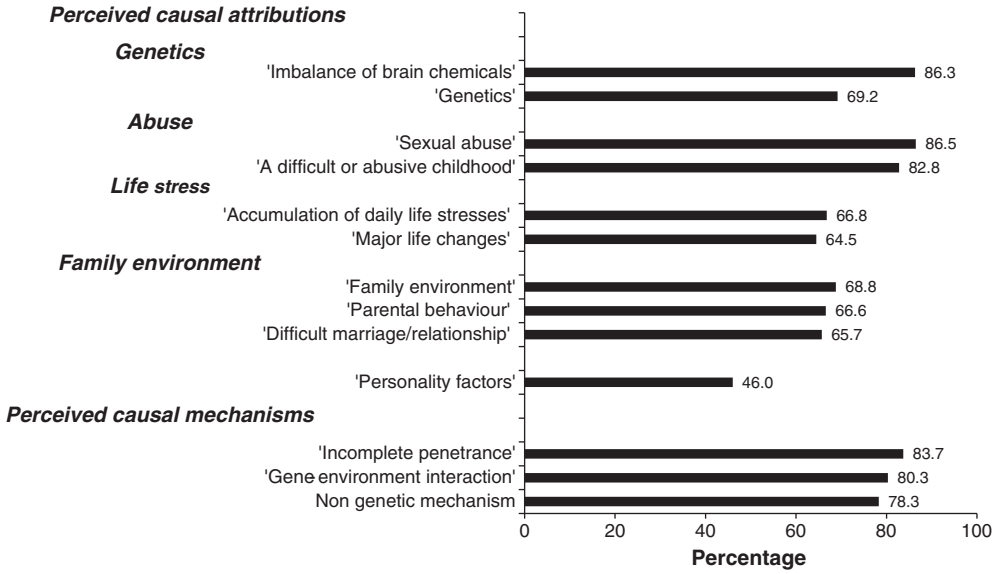

Fig. 1. Frequency of causal attributions perceived as important or very important to the development of mental illness (N = 1046).

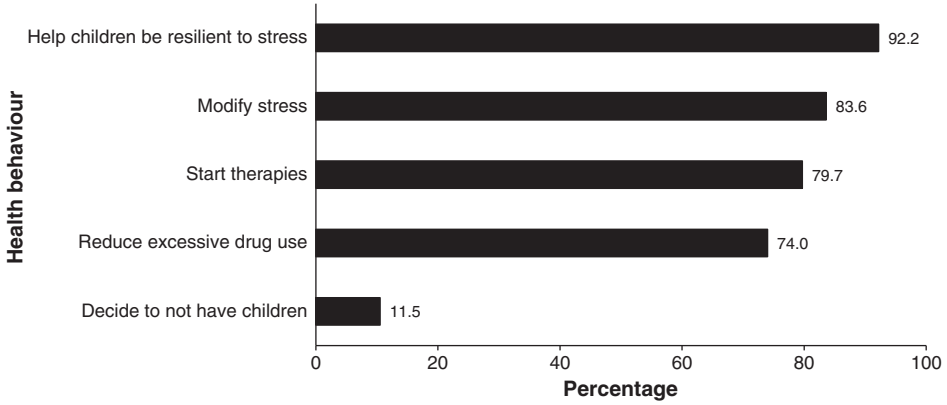

Fig. 2. Anticipated health behaviours in response to receiving a hypothetical genetic test result indicating higher than average risk for depression (N = 1046).

3.1. Start therapies or courses

As detailed in the final linear regression model shown in Table 2, participants willing to start therapies or courses that would facilitate learning of coping strategies in response to receiving a genetic test result indicating increased risk for major depressive disorder were significantly more likely to have estimated their risk for depression to be higher than average (ß = 0.12, $p<0.001$); endorse family environment as a causal attribution (ß = 0.11, $p<0.001$) and endorse 'gene–environment interaction' as a causal mechanism (ß = 0.12, $p<0.001$).

3.2. Behaviours to modify life stressors

Participants willing to engage in behaviours that modify life stressors after receiving a genetic test result indicating increased risk for major depressive disorder were significantly more likely to have estimated their risk for depression to be higher than average (ß = 0.07, $p = 0.029$); endorse 'abuse' as a causal attribution (ß = 0.10, $p = 0.003$); and endorse 'gene–environment interaction' as a causal mechanism (ß = 0.10, $p = 0.002$).

3.3. Reduce excessive drug and alcohol use

Participants willing to reduce excessive drug and alcohol use were significantly more likely to be female (ß = 0.09, $p = 0.009$).

3.4. Help one's own children learn to be more resilient to stress

Participants willing to help one's own children learn to be more resilient to stress were significantly more likely to be female (ß = 0.07, $p = 0.027$) and endorse gene–environment interaction as a causal mechanism for mental illness (ß = 0.16, $P<0.001$).

3.5. Decide to not have children

Participants who said they would decide to not have children in response to receiving a genetic test result indicating increased risk for major depressive disorder were significantly more likely to be older (ß = 0.18, $p<0.001$), and have a lower level of education (ß = −0.11, $p = 0.003$).

4. Discussion

This large population-based study found high acceptance for a range of behavioural interventions to ameliorate risk for major depressive disorder in the hypothetical scenario of receiving a high-risk estimate based on genetic testing. Participants who stated an intention to engage in protective health behaviours to reduce risk for major depressive disorder were more likely to perceive a higher than average population risk for major depressive disorder. This finding confirms the value of targeting preventive interventions at groups with an elevated risk of future depressive episodes who are more likely to be interested in preventive behavioural strategies.

The most frequently rated anticipated health behaviours in response to a hypothetical increased genetic risk for major depressive disorder risk were: helping one's own children learn how to be more resilient to stress (92.2%), modify potential life stressors (82.6%), and start therapies or courses to learn better coping strategies (79.7%). These findings are consistent with previous reports about preferred protective behaviours in response to genetic risk (Wilde et al., 2009; Wilhelm et al., 2009). The findings are also consistent with beliefs reported in a previous study that such testing could facilitate prevention and earlier intervention of major depressive disorder (Wilhelm et al., 2009). Having a personal history of mental illness was not a predictor of willingness to engage in anticipated health behaviours in the final model, in contrast to our first hypothesis. However, self-perception of having a higher than the average population risk for major depressive disorder was significantly and positively associated with willingness to start therapies and modify stress, consistent with our second hypothesis. Individuals intending to engage in health behaviours were more likely to endorse a gene–environment model for major depressive disorder, with endorsement of 'family environment' and 'abuse' as causal attributions significantly and positively associated with the intention to take up mental health protective behaviours (hypothesis three). This finding suggests that such individuals may view risk for major depressive disorder as modifiable.

Table 1a
Items explored for bivariate association with anticipated health behaviours in response to receiving a hypothetical genetic test result indicating higher than average risk for depression. (Maximum N = 1046).

Variable	Start therapies				Modify stress				Reduce excessive drug, alcohol use			
	N	Mean (S.D.) agreement score	r_s^e/z	p	N	Mean (S.D.) agreement score	r_s^e/z	p	N	Mean (S.D.) agreement score	r_s^e/z	p
Endorsement of causal attributions												
Genetics	1033	–	0.96	0.156	1011	–	0.15	0.641	870	–	0.01	0.771
Abuse	1033	–	0.11	<0.001[e]	1011	–	0.10	<0.002[e]	870	–	0.07	0.047[e]
Life stress	1033	–	0.13	<0.001[e]	1011	–	0.09	0.006[e]	870	–	0.02	0.578
Family environment	1033	–	0.10	0.002[e]	1011	–	0.05	0.103	870	–	0.05	0.111
Endorsement of gene–environment interaction	990	–	0.10	<0.001[e]	969	–	0.12	<0.001[e]	834	–	0.09	0.007[e]
History of mental illness self[a]												
Yes	237	4.1 (0.9)	−3.43	0.001[e]	233	4.1 (0.8)	−3.55	<0.001[e]	204	4.1 (0.8)	0.98	0.329
No	794	3.8 (0.9)			775	3.9 (0.8)			665	4.1 (0.8)		
Close relative/friend[b]												
Yes	653	3.9 (0.9)	−0.35	0.728	646	4.0 (0.8)	−0.63	0.53	562	4.1 (0.8)	1.98	0.048[e]
No	372	3.9 (0.9)			358	4.0 (0.8)			301	4.0 (0.8)		
Self-estimation of risk for major depressive disorder[c]												
Higher than average	239	4.1 (0.8)	0.11	0.001[e]	235	4.1 (0.7)	0.11	<0.001[e]	212	4.1 (0.8)		
Same as average	492	3.8 (0.9)			488	3.9 (0.8)			425	4.1 (0.8)		
Lower than average	291	3.8 (1.0)			278	3.9 (0.9)			222	4.2 (0.8)	0.03	0.329
Sex												
Male	401	3.8 (0.9)			392	3.9 (0.8)			354	4.0 (0.9)		
Female	632	3.9 (0.9)	−2.12	0.034[e]	619	4.0 (0.8)	−0.72	0.469	516	4.2 (0.8)	2.62	0.009[e]
Age	1031		0.02[d]	0.525	1009		−0.01[d]	0.77	868		0.03[d]	0.429
Education level												
No post-school education	468	3.9 (0.9)			457	4.0 (0.8)	−0.04	0.7	391	4.0 (0.9)		
Tertiary education	562	3.9 (0.9)	−0.03	0.98	551	4.0 (0.8)			476	4.0 (0.8)	1.63	0.104

[a] Refers to personal history of a mental illness (depression, bipolar disorder or schizophrenia).
[b] Refers to experience of depression, bipolar or schizophrenia through a close relative or close friend.
[c] Refers to personal estimation of risk for major depressive disorder compared to average population risk. z values are absolute values from Mann Whitney-U tests.
[d] r_s values are Spearman's rank correlations.
[e] p values <0.1 entered into linear regression.

Few quality RCTs are available to assess the broader impact of disease risk estimates, clinical or hypothetical, on behavioural change. A recent Cochrane review of 17 'poor quality' studies found little evidence that communicating DNA-based disease risk estimates had an effect on smoking and physical activity although there was a possible small effect on self-reported diet and on intentions to change behaviour (Marteau et al., 2010).

A study involving predictive genetic testing for the familial hypercholesterolaemia mutation showed that participants with the mutation believed more strongly that a biological-based intervention such as cholesterol-lowering medication would be most effective in reducing cholesterol level and believed less strongly that behavioural change, such as altering diet, would be useful (Marteau et al., 2004). Similar results were seen in a small qualitative study on the impact of neonatal genetic screening for familial hypercholesterolaemia, in which parents who perceived the condition as dietary rather than genetic in origin, viewed the condition controllable by altering neonatal diet (Senior et al., 1999). Furthermore, provision of a hypothetical genetic test result linked to increased risk of nicotine dependence found that smokers provided with such a genetic test result were more likely to select a pharmacological agent to assist stopping smoking and less likely to select their own willpower, than smokers who were not given such information about genetic risk (Wright et al., 2003). These studies show that perceived origin of a health problem may influence what intervention is preferred and selected. That is, individuals may seek biological-based interventions when a health risk is perceived as genetic in origin and less preventable, or behavioural-based interventions when risk is perceived as environmental in origin and more controllable.

By contrast, the present study suggests that genetic risk information is unlikely to demotivate individuals to consider reducing risk through behavioural change, nor induce a sense of genetic fatalism as shown previously (Senior et al., 1999); (Marteau et al., 2004). Rather, it shows that motivation to modify risk of a hypothetical genetic predisposition appeared to be driven by perceptions that environmental factors contribute to overall risk of major depressive disorder and that these could be controlled by adopting preventive behaviours. The findings may differ from Senior et al. (1999) and Marteau et al. (2004) because the previous studies did not attempt to evaluate endorsement of both genetic and environmental causes of disease or gene–environment interactions as a causal mechanism.

The present study found little support for the contention that a hypothetically increased genetic risk for major depressive disorder would lead to the decision to not have children in the event of receiving an unfavourable genetic test result. The minority of participants (10.5%) who said that an increased risk of major depressive disorder would deter them from having

Table 1b
Items explored for bivariate association with anticipated health behaviours in response to receiving a hypothetical genetic test result indicating higher than average risk for depression (Maximum N = 1046).

Variable	Help children be resilient				Decide to not have children			
	N	Mean (S.D.) agreement score	r_s^e/z	p	N	Mean (S.D.) agreement score	r_s^e/z	p
Endorsement of causal attributions								
Genetics	1007	–	0.01	0.716	816	–	0.05	0.191
Abuse	1007	–	0.10	0.001[e]	816	–	0.26	0.454
Life stress	1007	–	0.03	0.263	816	–	0.10	0.003[e]
Family environment	1007	–	0.03	0.172	816	–	0.05	0.203
Endorsement of gene–environment interaction	965	–	0.19	<0.001[e]	785	–	−0.01	0.832
History of mental illness personal[a]								
Yes	226	4.4 (0.6)	−3.24	0.001[e]	192	2.0 (1.1)		0.002[e]
No	780	4.3 (0.7)			623	2.2 (1.0)	−0.31	
Close relative/friend[b]								
Yes	645	4.3 (0.7)	−1.24	0.217	529	2.0 (1.0)		
No	354	4.3 (0.6)			280	2.3 (1.1)	−2.84	0.004[e]
Self-estimation of risk for major depressive disorder[c]								
Higher than average	229	4.4 (0.6)	0.10	0.001[e]	200	2.1 (1.1)		
Same as average	483	4.3 (0.7)			387	2.1 (1.0)		
Lower than average	286	4.3 (0.7)			223	2.3 (1.0)	−0.01	0.002[e]
Sex								
Male	392	4.3 (0.7)	−0.71	0.007[e]	317	2.2 (1.0)	−1.21	0.227
Female	615	4.4 (0.6)			499	2.1 (1.0)		
Age	1005		−0.04[d]	0.225	814		0.20[d]	<0.001[e]
Education level								
No post-school education	452	4.3 (0.7)	−1.48	0.14	345	2.3 (1.1)	−3.62	<0.001[e]
Tertiary education	553	4.4 (0.7)			469	2.3 (0.9)		

[a] Refers to personal history of a mental illness (depression, bipolar disorder or schizophrenia).
[b] Refers to experience of depression, bipolar or schizophrenia through a close relative or close friend.
[c] Refers to personal estimation of risk for major depressive disorder compared to average population risk. z values are absolute values from Mann Whitney-U tests.
[d] r_s values are Spearman's rank correlations.
[e] p values <0.1 entered into linear regression.

children were older and had no post-school education. Previous studies have reported reluctance to have children in the event of having an increased genetic risk of major depressive disorder (Iles et al., 2003), bipolar disorder (Trippitelli et al., 1998), or schizophrenia (Iles et al., 2003) amongst individuals unselected for family history and amongst individuals with a strong family

Table 2
Final linear regression models predicting factors influencing intention to take up various behaviours in response to receiving a hypothetical genetic test result indicating higher than average risk for depression, after adjusting for age, sex and education level.

Variable	Raw coefficient	ß	95% CI raw coefficient	t	p
Start therapies or courses[a]					
Self-estimation of risk for major depressive disorder higher than average	0.15	0.12	0.07 to 0.23	3.67	<0.001
Endorse 'family environment' as a causal attribution	0.11	0.11	0.05 to 0.17	3.56	<0.001
Endorse 'gene–environment interaction' as a causal mechanism	0.14	0.12	0.07 to 0.21	3.68	<0.001
Modify stress[b]					
Self-estimation of risk for major depressive disorder higher than average	0.08	0.07	0.01 to 0.15	2.18	0.029
Endorse 'abuse' as causal attribution	0.11	0.10	0.04 to 0.19	2.94	0.003
Endorse 'gene–environment interaction' as a causal mechanism	0.10	0.10	0.04 to 0.17	3.04	0.002
Reduce excessive drug and alcohol use[c]					
Sex	0.15	0.09	0.04 to 0.26	2.63	0.009
Help one's own children learn to be more resilient to stress[d]					
Endorse 'gene–environment interaction' as a causal mechanism	0.14	0.16	0.08 to 0.19	5.15	<0.001
Sex	0.01	0.07	0.01 to 0.18	2.21	0.027
Decide to not have children[e]					
Age	0.01	0.18	0.01 to 0.02	5.15	<0.001
Education level	−0.21	−0.11	−0.35 to −0.07	−3.01	0.003

[a] Final model: $R^2 = 0.049$, $F = 8.405$, $p < 0.001$. Adjusted $R^2 = 0.044$, $R = 0.222$. $N = 976$.
[b] Final model: $R^2 = 0.029$, $F = 4{,}655$, $p < 0.001$. Adjusted $R^2 = 0.022$, $R = 0.169$. $N = 956$.
[c] Final model: $R^2 = 0.014$, $F = 4.074$, $p < 0.007$. Adjusted $R^2 = 0.011$, $R = 0.118$. $N = 862$.
[d] Final model: $R^2 = 0.034$ $F = 8.498$, $p < 0.001$. Adjusted $R^2 = 0.030$, $R = 0.185$. $N = 950$.
[e] Final model: $R^2 = 0.057$, $F = 12.146$, $p < 0.001$. Adjusted $R^2 = 0.052$, $R = 0.238$. $N = 812$.

history of bipolar disorder (Meiser et al., 2007). Furthermore, overestimation of risk amongst unaffected relatives of individuals with psychosis favoured fewer children (Austin et al., 2006). Given the low predictive power and incomplete penetrance of psychiatric genotypes, decisions to not have children based on genetic risk for these disorders may be unjustified. Since genetic risk information also has potential to influence reproductive decisions, further research is required to assess the influence of actual genetic risk information on reproductive decisions amongst individuals with a family history of major depressive disorder.

Sex differences were detected in the present study, with females more likely than males to choose to reduce excessive drug and alcohol use and to help children learn resilience as protective behavioural options. The latter finding could be explained by females being more likely to be caregivers to children. Both findings could reflect the greater likelihood of females to engage in medical interventions generally (Moynihan, 1998).

Genetic testing or the provision of risk estimates in psychiatry may provide information that can lead to behaviours that promote mental health and reduce risk for disease. It should be borne in mind that provision of genetic risk information and intention to take up protective health behaviours may not translate into actual change of behaviour (Leventhal et al., 1997). The findings do not suggest that provision of genetic risk information directly promotes protective health behaviours, but shows that individuals may be receptive to undertaking protective health behaviours as part of a genetic risk assessment for major depressive disorder.

There is a possibility the hypothetical nature of the genetic risk scenario in the present study weakened participants' sensitivity to the potential personal impact of such a genetic test result. It should be noted that evidence thus far for the impact of clinical or hypothetical risk estimates on promoting behavioural change is based on small trials or hypothetical risk estimates. Large randomised control trials are required using risk estimates based on genetic or hereditary risk information to determine the extent to which individual risk influences perception of control and motivation to adopt health behaviours that ameliorate risk for major depressive disorder.

5. Conclusions

This is the first study to provide data from a large national cohort in which motivation to change health behaviour in response to hypothetical depression genetic risk testing has been investigated. It is likely that causes attributed to mental illness influence perceptions about what kind of interventions might be effective in reducing risk or preventing disease. The results suggest that informing people of their genetic susceptibility to major depressive disorder may motivate individuals to engage in risk reducing behaviour, although this may not occur as a direct result of genetic testing. Rather than facilitating protective behavioural change some evidence suggests that genetic risk information could induce fatalistic attitudes about modifiability of disorders with associated genetic susceptibility, thus inhibiting willingness to engage in protective health behaviours (Marteau et al., 2004; Marteau and Lerman, 2001; Senior et al., 1999; Senior et al., 2000). In particular, the study has identified that individuals who perceive themselves to be at increased risk for major depressive disorder and who endorse gene–environment interactions as a cause are likely to be motivated to engage in various protective interventions at a pre-symptomatic stage. The study has shown that mental health interventions that facilitate learning of effective coping skills are likely to be well-received as preventive strategies.

These findings now require investigation in a prospective study to evaluate how the impact of actual risk estimates may differ from the hypothetical scenario posited in this survey. Studies of risk reduction behaviours amongst target healthy adults following provision of risk estimates for a psychiatric disorder are required to inform the planning and monitoring of health promotion and risk-reduction strategies associated with genetic testing.

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Conflict of interest

None declared.

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