A Systematic Review of Genetic Testing and Lifestyle Behaviour Change: Are We Using High-Quality Genetic Interventions and Considering Behaviour Change Theory?

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Keywords
Health behaviour · Genetics · Nutrition · Physical activity · Smoking · Personalized healthcare · Nutrigenomics · Lifestyle genomics

Abstract
Background: Studying the impact of genetic testing interventions on lifestyle behaviour change has been a priority area of research in recent years. Substantial heterogeneity exists in the results and conclusions of this literature, which has yet to be explained using validated behaviour change theory and an assessment of the quality of genetic interventions. The theory of planned behaviour (TPB) helps to explain key contributors to behaviour change. It has been hypothesized that personalization could be added to this theory to help predict changes in health behaviours. Purpose: This systematic review provides a detailed, comprehensive identification, assessment, and summary of primary research articles pertaining to lifestyle behaviour change (nutrition, physical activity, sleep, and smoking) resulting from genetic testing interventions. The present review further aims to provide in-depth analyses of studies conducted to date within the context of the TPB and the quality of genetic interventions provided to participants while aiming to determine whether or not genetic testing facilitates changes in lifestyle habits. This review is timely in light of a recently published “call-to-action” paper, highlighting the need to incorporate the TPB into personalized healthcare behaviour change research. Methods: Three bibliographic databases, one key website, and article reference lists were searched for relevant primary research articles. The PRISMA Flow Diagram and PRISMA Checklist were used to guide the search strategy and manuscript preparation. Out of 32,783 titles retrieved, 26 studies met the inclusion criteria. Three quality assessments were conducted and included: (1) risk of bias, (2) quality of genetic interventions, and (3) consideration of theoretical underpinnings – primarily the TPB. Results: Risk of bias in studies was overall rated to be “fair.” Consideration of the TPB was “poor,” with no study making reference to this theoretical framework.
Introduction

Since decoding the entire human genome in 2003 [1], there have been considerable advances in genetic research and the clinical utility of genetic testing. The terms nutrigenomics or nutritional genomics describe the study of how genes interact with the foods, beverages, and supplements consumed to influence health outcomes [2]. Currently, there are no generally accepted or standardized terms describing the study of how genes interact with physical activity, sleep, or smoking to influence subsequent health outcomes. These gene-lifestyle interactions can be referred to using the broad term lifestyle genomics. Despite the lack of a standardized terminology, research pertaining to nutrigenomics and other emerging genomic sciences continues to advance. Specifically, behaviour change guided by genetic testing results or other personalized healthcare information is emerging as a priority area of research, with several reviews on this topic published in recent years [3–6].

Genetic testing is increasingly used in clinical practice to provide personalized information and recommendations about health risks and lifestyle habits at a relatively low cost [7]. However, studies assessing whether or not genetic testing promotes changes in lifestyle habits have conflicting findings [8–11]. Given that chronic diseases can often be managed through lifestyle interventions alone, or a combination of lifestyle interventions and medication [12–14], genetic testing's potential to provide personalized lifestyle recommendations holds considerable promise.

Behaviour change is a multifactorial, complex area of research and clinical practice. The theory of planned behaviour (TPB) is arguably the most widely accepted behaviour change theory in academia [15]. This theory posits that attitudes, subjective norms, and perceived behavioural control are key constructs that can be used to predict behaviours. Actual behavioural control, which typically refers to factors such as income, educational level, and other social determinants of health for the purposes of healthcare research, further contributes to one’s likelihood of performing a behaviour [15, 16]. It is important for genetic testing behaviour change research to consider validated theories in order to control for a number of confounding factors that could significantly influence the results of a study.

Despite the complexity of behaviour change, genetic testing behaviour change studies do not often use any theoretical underpinnings to inform their study design, or for the analysis and interpretation of their data. This is concerning, as it implies that these studies did not report whether they considered the many confounding factors impacting behaviour change, including but not limited to attitudes, subjective norms, and perceived and actual behavioural control [17]. Consideration of such factors could help explain why some studies conclude that genetic testing facilitates health behaviour change, while others conclude that it does not. For example, a study may find that genetic testing has a positive influence on attitudes and subjective norms, but it is only when behavioural control is high (for example, with a higher income or education level) that genetic testing facilitates health behaviour change. The importance of such considerations has been highlighted in a recent call to action for personalized healthcare behaviour change research, which recommended the completion of a systematic review with perspective from the TPB as an important next step in advancing knowledge in personalized healthcare behaviour change literature [18].

Systematic reviews and meta-analyses are typically considered the highest quality of scientific evidence and, notably, often guide clinical practice [19]. When it comes to systematic reviews assessing behaviour change as a result of genetic testing interventions, a simple risk-of-bias assessment is not sufficient to develop the most meaningful conclusions; yet it is often the only quality assessment conducted in this type of work [3, 5, 6]. It is further im-
important to consider the delivery of a health/genetic intervention (such as considering the provision of disease risk estimates vs. actionable behaviour change recommendations) and to consider behaviour change theories [18]. Therefore, the development of more comprehensive methods for reviewing and compiling the primary research articles conducted to date related to genetic testing behaviour change is needed.

The present review provides an in-depth analysis and summary of the current body of knowledge, thus presenting the most robust and comprehensive review of genetic testing behaviour change research conducted to date. Overall, the purpose of this comprehensive systematic review is to use these novel perspectives to answer the following research questions: Are we considering validated behaviour change theory (particularly the TPB) in genetic testing behaviour change research? Are we using high-quality genetic interventions in genetic testing behaviour change research? What is the impact of genetic testing on behaviour change pertaining to four lifestyle factors: nutrition, physical activity, smoking, and/or sleep? These four lifestyle factors were chosen as they have all been shown to have a significant impact on chronic disease management [20–24]. Behaviour change is challenging, and it is important to find strategies that effectively facilitate beneficial lifestyle changes related to nutrition, physical activity, smoking, and/or sleep. Genetic tests may provide information on disease risk, which can be mitigated through specific alterations in lifestyle habits such as improving nutrition, optimizing physical activity habits, quitting smoking or smoking less, and engaging in healthful sleep-related behaviours.

Methods

Search Strategy

The systematic review protocol that was used to guide this review is detailed elsewhere [25]. In brief, the search strategy was guided by the PRISMA Flow Diagram [26]. From February to April 2017, the following databases were searched for relevant articles: PubMed, Scopus, and Nursing & Allied Health. Publications posted on the Food4Me website [27], as well as the reference lists of 4 recent review articles published on topics similar to those of the present review [3–6], were also screened for articles relevant to the research questions. After the number of records had been condensed through title and abstract screening, the full-text articles were reviewed to assess each one for eligibility according to predetermined inclusion and exclusion criteria. The complete search terms and search strategy were developed and approved by all authors, and they are detailed in Figure 1 and Figure 2, respectively.

Selection Criteria

To capture a comprehensive summary of the research conducted to date, the present review was not limited to a single, specific study design. We included primary research articles published in English in peer-reviewed journals from all years which assessed the impact of genetic testing on one or more of the four lifestyle habits of interest (nutrition, physical activity, smoking, and/or sleep). Both qualitative and quantitative studies were included. Studies were excluded if there was not at least one group of participants who underwent genetic testing and/or if the study did not provide follow-up data related to one or more of the lifestyle habits of interest after the participants had received the results of a genetic test. One author (J.H.) completed data extraction using piloted forms [28], which were tested on 4 studies, reviewed by another author (J.G.), and modified during the piloting process by two authors (J.H. and J.G.).

Analysis

The National Institutes of Health (NIH) Study Quality Assessment Tools were used to conduct a risk-of-bias assessment in quantitative research [29]. The Critical Appraisal Skills Programme Qualitative Research Checklist [30] was used to assess risk of bias in qualitative research. The quality of the genetic intervention was also assessed. To our knowledge, there currently is no tool available for assessing the quality of a genetic intervention. As such, we developed the first assessment tool for evaluating the quality of a genetic intervention provided to subjects (online suppl. Table 1; see www.karger.com/doi/10.1159/000488086 for all online suppl. material). The quality rating and general outline for this new tool was based on the format of the NIH Study Quality Assessment Tools [29]. The questions included were developed from a review of previously identified critiques and concerns related to genetic testing and health risk messages [11, 31–38].

Consideration of the main components of the TPB (attitudes towards a behaviour, subjective norms, behavioural control, and intention) [17], as well as consideration of theory more generally, was assessed using deductive content analysis of the manuscripts.
The deductive content analyses of consideration of the TPB and its key components in each study was then translated into a rating, based on the rating system generated in the NIH Study Quality Assessment Tools, whereby “good” indicates a robust consideration of the main components of the TPB, “fair” indicates intermediate consideration of the main TPB components, and “poor” represents little to no consideration of the main TPB components. An overall quality score was assigned to each article based on a point system, where “good” ratings were awarded 3 points, “fair” ratings were awarded 2 points, and “poor” ratings were awarded 1 point. The maximum possible overall quality rating was 9/9, upon consideration of all three assessments.

**Results**

The comprehensive electronic literature search returned a total of 32,783 results, with 26 studies meeting the predetermined inclusion criteria. In these 26 studies, the following outcomes were assessed: nutrition (n = 18), physical activity (n = 16), and smoking (n = 12) (Fig. 1), with 14 articles assessing more than one lifestyle habit of interest to this review. The vast majority of the literature has been published over the past decade, with a large spike in publications recently in 2015 (online suppl. Fig. 1). Consistent with recommendations for systematic reviews [25], our review was analytic and descriptive in nature and included: (a) a tabulation of the study characteristics and findings (Table 1); (b) a thorough and robust quality assessment (Table 2); and (c) a narrative synthesis. Research conducted thus far has focused on a variety of genes, as outlined in Table 3. It is concerning to note that 12 studies (46%) did not report whether or not the authors had a conflict of interest (COI). The vast majority of the literature has focused on genetic testing for determining the risk of developing certain diseases or conditions (88%; n = 23), while only a small number of studies have focused on nutrient metabolism (12%; n = 3), which indirectly affects the risk of developing diseases or conditions [40–42]. The three separate quality assessments completed on each study are summarized in Table 2. Risk of bias was overall rated as “fair.”

**Are We Using High-Quality Genetic Interventions?**

Although some risk of bias is apparent, the ratings for the quality of the genetic interventions were more con-
### Table 1. Summary of study characteristics and behaviour change findings

<table>
<thead>
<tr>
<th>First author [Ref.], date</th>
<th>Participants (n baseline; n follow-up)</th>
<th>Intervention group(s)</th>
<th>Comparison group(s)</th>
<th>Target diseases/ conditions (genes tested)</th>
<th>Follow-up</th>
<th>Lifestyle habits assessed</th>
<th>Outcomes (p values); conclusions</th>
<th>Ranking</th>
<th>COI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Boeldt [45], 2017</td>
<td>Young female adults (n = 57; n = 56)</td>
<td>Genetic testing</td>
<td>No genetic testing</td>
<td>Health effects related to omega-3 intake (FADS1)</td>
<td>3 months</td>
<td>Nutrition (omega-3: EPA and DHA)</td>
<td>NS change in omega-3 intake in the genetic testing group compared to the control group (no genetic testing)</td>
<td>1</td>
<td>No</td>
</tr>
<tr>
<td>Marsaux [10], 2016</td>
<td>Adults (n = 265; n = 130)</td>
<td>High-risk genetic result</td>
<td>Non-risk genetic result</td>
<td>Overweight/obesity (FTO)</td>
<td>6 months</td>
<td>Physical activity</td>
<td>NS change in subjective or objective physical activity with provision of FTO genotype risk info</td>
<td>3</td>
<td>Yes</td>
</tr>
<tr>
<td>Meisel [44], 2015</td>
<td>Young adults (n = 1,916; n = 279)</td>
<td>Genetic testing</td>
<td>No genetic testing</td>
<td>Obesity (FTO)</td>
<td>1 month</td>
<td>Nutrition (adherence to a variety of eating behaviours) and physical activity</td>
<td>NS changes in nutrition and physical activity (pooled) between groups</td>
<td>1</td>
<td>No</td>
</tr>
<tr>
<td>Boeldt [45], 2015</td>
<td>Adults working at health technology companies (NR, n = 2,037)</td>
<td>Genetic testing</td>
<td>None</td>
<td>23 conditions including heart attack, Alzheimer disease, type 2 diabetes, obesity, colon cancer, and cervical cancer (NR)</td>
<td>2 weeks</td>
<td>Nutrition (dietary fat and physical activity)</td>
<td>NS (significance level NR) change in nutrition and physical activity following genetic testing</td>
<td>4</td>
<td>No</td>
</tr>
<tr>
<td>Hietanen-Luoma [9], 2015</td>
<td>Adults (n = 122; n = 113 at 12 months)</td>
<td>Genetic testing</td>
<td>No genetic testing</td>
<td>Cardiovascular disease (apoE)</td>
<td>6 months</td>
<td>Nutrition (fat quality, and consumption of vegetables, berries, fruits, and fatty and sugary foods) and physical activity</td>
<td>Improved dietary fat quality in the high-risk genetic result group vs. the control group at 2 weeks (p &lt; 0.05) and 6 months of follow-up (p &lt; 0.05); decreased intake of high-fat, high-sugar foods in the non-risk genetic result group vs. the control group at 12 months (p &lt; 0.05)</td>
<td>1</td>
<td>No</td>
</tr>
<tr>
<td>Voilu [46], 2015</td>
<td>Veterans (n = 691; n = 506 at 3 months, n = 472 at 6 months)</td>
<td>Genetic testing</td>
<td>No genetic testing</td>
<td>Type 2 diabetes (TCF7L2, PPARγ, and KCNJ11)</td>
<td>3 months</td>
<td>Nutrition (calories, carbohydrates, protein, fat, saturated fat, MUFA, and PUFA) and physical activity</td>
<td>Reduced calories and fat (MUFA and PUFA) in the genetic testing group vs. the no-genetic-testing group (p &lt; 0.05) at 3 months; NS changes in nutrition between the groups at 6 months; NS changes in physical activity at either time point</td>
<td>1</td>
<td>Yes</td>
</tr>
<tr>
<td>Marsaux [47], 2015</td>
<td>Adults (n = 1,607; n = 1,233 with subjective data at 6 months, n = 730 with objective data at 6 months)</td>
<td>Genetic testing</td>
<td>No genetic testing</td>
<td>Overweight/obesity (FTO)</td>
<td>3 months</td>
<td>Physical activity</td>
<td>NS changes in physical activity with the addition of genetic information</td>
<td>1</td>
<td>Yes</td>
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<tr>
<td>Nielsen [11], 2014</td>
<td>Adults (n = 138; n = 130 at 3 months, n = 123 at 12 months)</td>
<td>Genetic testing</td>
<td>No genetic testing</td>
<td>Caffeine metabolism (CYP1A2, vitamin C utilization (GSTT1 and GSTM1), sweet taste perception (TAS1R2), and sodium sensitivity (ACE))</td>
<td>3 months</td>
<td>Nutrition (caffeine, vitamin C, added sugar, and sodium)</td>
<td>The high-risk genetic result group (for the ACE gene) had reduced sodium intake to a greater extent than the control group by the 12-month follow-up (p = 0.008); NS changes in caffeine, vitamin C, and added sugar intake at each follow-up time point; NS changes in sodium intake at the 3-month follow-up</td>
<td>1</td>
<td>Yes</td>
</tr>
<tr>
<td>First author [Ref.], date</td>
<td>Participants (n baseline; n follow-up)</td>
<td>Interventions</td>
<td>Comparison group(s)</td>
<td>Target diseases/conditions (genes tested)</td>
<td>Follow-up</td>
<td>Lifestyle habits assessed</td>
<td>Outcomes (p values); conclusions</td>
<td>Ranking of study design</td>
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<tr>
<td>Egglestone [8], 2013</td>
<td>Adults who had purchased a DTC genetic test or were considering purchasing a test or who were awaiting their results (n = 275)</td>
<td>Genetic testing</td>
<td>No genetic testing</td>
<td>NR (NR)</td>
<td>Varied</td>
<td>Nutrition (healthier diet, vitamins/supplements, caffeine, fibre, salt, fat, and fruits/vegetables), physical activity, and smoking</td>
<td>Greater health behaviour scores in the genetic testing group vs. the control group (p = 0.02 for pooled nutrition, physical activity, and smoking); the most common changes were “healthier diet,” “more exercise,” and “taking vitamins or supplements”; more often reported “sufficient fruit and vegetable intake” in the genetic testing group (p = 0.03); NS changes in smoking individually</td>
<td>2</td>
<td>NR</td>
</tr>
<tr>
<td>Bloss [48], 2013</td>
<td>Adults working at health and technology companies (n = 3,639; n = 2,037 at 3 months, n = 1,325 at 14 ± 1.3 months)</td>
<td>Genetic testing</td>
<td>None</td>
<td>Deep vein thrombosis, melanoma, sarcoidosis, haemochromatosis, lactose intolerance, breast cancer, prostate cancer + 20 other conditions not listed (variable)</td>
<td>3 months 14±1.3 months</td>
<td>Nutrition (dietary fat) and physical activity</td>
<td>NS changes in nutrition or physical activity at 3 months (significance level NR) or 14±1.3 months</td>
<td>4</td>
<td>No</td>
</tr>
<tr>
<td>Kaufman [49], 2012</td>
<td>Adult customers of DTC genetic testing companies (n = 3,167; n = 1,048)</td>
<td>Genetic testing and High-risk genetic result</td>
<td>Non-risk genetic result</td>
<td>Variable (variable)</td>
<td>2-8 months</td>
<td>Nutrition (change diet) and physical activity</td>
<td>The participants who considered themselves at high risk of colon cancer were significantly more likely to change their diet (p = 0.02) and start exercising more (p = 0.01) than those who considered themselves at low risk of colon cancer; 10% of all participants reported they changed a supplement, 33% reported being more careful about their diet, and 14% reported exercising more</td>
<td>2</td>
<td>NR</td>
</tr>
<tr>
<td>Hollands [50], 2012</td>
<td>Adults with 1st-degree relatives with Crohn disease (n = 497; n = 426)</td>
<td>Genetic testing and High-risk genetic result</td>
<td>No genetic testing and Non-risk genetic result</td>
<td>Crohn disease (NOD2)</td>
<td>6 months</td>
<td>Smoking</td>
<td>NS changes in smoking cessation between the genetic testing and the no-genetic-testing group; NS changes in smoking cessation between the high risk and the non-risk genetic result group (significance level NR)</td>
<td>1</td>
<td>No</td>
</tr>
<tr>
<td>Bloss [36], 2011</td>
<td>Adults working at health and technology companies (n = 3,639; n = 2,037)</td>
<td>Genetic testing</td>
<td>None</td>
<td>23 conditions including breast and prostate cancer (NR)</td>
<td>5.6±2.4 months</td>
<td>Nutrition (dietary fat) and physical activity</td>
<td>NS changes in nutrition and/or physical activity following genetic testing</td>
<td>4</td>
<td>NR</td>
</tr>
<tr>
<td>Vernarelli [51], 2010</td>
<td>Adults with at least one parent who developed Alzheimer disease (n = 279; n = 272)</td>
<td>High-risk genetic result</td>
<td>Non-risk genetic result</td>
<td>Alzheimer disease (apoE)</td>
<td>6 weeks</td>
<td>Nutrition (dietary supplement use) and physical activity</td>
<td>The high-risk genetic result group was more likely to take supplements than the non-risk genetic result group (p = 0.0001)</td>
<td>3</td>
<td>No</td>
</tr>
<tr>
<td>First author [Ref.], date</td>
<td>Participants (n baseline; n follow-up)</td>
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<tr>
<td>Hishida [52], 2010</td>
<td>Adult smokers (n = 562; n = 533)</td>
<td>Genetic testing</td>
<td>No genetic testing</td>
<td>Lung and oesophageal cancer (L-myc)</td>
<td>12 months</td>
<td>Smoking</td>
<td>NS changes in smoking cessation between the genetic testing and the no-genetic-testing group</td>
<td>1</td>
<td>No</td>
</tr>
<tr>
<td>Quach [53], 2009</td>
<td>Adults with a personal and/or family history of breast and/or ovarian cancer (n = 120; NR)</td>
<td>Genetic testing</td>
<td>None</td>
<td>Breast and ovarian cancer (BRCA1/2)</td>
<td>6 months</td>
<td>Nutrition (healthy diet and vitamin use) and physical activity</td>
<td>NS changes in nutrition, vitamin use, or physical activity after genetic testing (significance level NR)</td>
<td>4</td>
<td>NR</td>
</tr>
<tr>
<td>O'Neill [54], 2008</td>
<td>Adult females (NR; n = 115 at 1 month and 6 months)</td>
<td>High-risk genetic result</td>
<td>and</td>
<td>Breast cancer (BRCA1/2)</td>
<td>1 month</td>
<td>Nutrition (saturated fat, fruit/vegetables) and physical activity</td>
<td>NS differences between groups in nutrition or physical activity at baseline and 1 month or 6 months following genetic testing</td>
<td>3</td>
<td>NR</td>
</tr>
<tr>
<td>Chao [55], 2008</td>
<td>Adult with parent who developed Alzheimer disease (n = 162; n = 147)</td>
<td>Genetic testing and</td>
<td>and</td>
<td>Alzheimer disease (apoE)</td>
<td>12 months</td>
<td>Nutrition (changes in diet, changes in vitamin/supplement use) and physical activity</td>
<td>The high-risk genetic result group was more likely to report a nutrition or physical activity change than the non-risk genetic result group (p = 0.003) and the no-genetic-testing group (p = 0.03); most common was a change in medication/supplement use (specifically vitamin E)</td>
<td>1</td>
<td>NR</td>
</tr>
<tr>
<td>Sanderson [56], 2008</td>
<td>Adult smokers (NR; n = 61)</td>
<td>Genetic testing</td>
<td>and</td>
<td>Lung cancer (GSTM1)</td>
<td>1 week 2 months</td>
<td>Smoking</td>
<td>Fewer cigarettes smoked (p = 0.009) and greater quit rates (p = 0.009) at the 1-week follow-up in the high-risk genetic result group than in the no-genetic-testing group; NS differences at the 2-month follow-up between the groups for cigarettes smoked and quit rates</td>
<td>1</td>
<td>No</td>
</tr>
<tr>
<td>Rees [57], 2007</td>
<td>Adult females (n = 23)</td>
<td>Genetic testing</td>
<td>None</td>
<td>Breast cancer (BRCA1/2)</td>
<td>Varied – up to 18 months</td>
<td>Nutrition (dietary changes), physical activity, and smoking</td>
<td>Few women reported a significant impact on nutrition, physical activity, and/or smoking as a result of receiving genetic testing results and counselling (significance level not applicable)</td>
<td>Qualitative</td>
<td>NR</td>
</tr>
<tr>
<td>Rief [58], 2007</td>
<td>Adults (n = 294)</td>
<td>Genetic testing and consultation</td>
<td>and</td>
<td>Obesity (NR)</td>
<td>6 months</td>
<td>Nutrition (restraint eating)</td>
<td>NS changes to restraint eating in the genetic testing group compared to the no-genetic-testing groups</td>
<td>1</td>
<td>No</td>
</tr>
</tbody>
</table>
Concerning, since overall the ratings were “poor” and only 6 of the 26 studies (23%) received a “good” rating. Thus, it is clear that the studies did not provide high-quality interventions to their participants, which helps to explain why the majority of studies did not report that genetic interventions facilitated lifestyle behaviour change.

*Are We Considering Validated Behaviour Change Theory?*

Consideration of the TPB and/or one or more of the theory’s three key components had mode overall ratings of “poor.” The deductive content analyses of the theoretical underpinnings mentioned in the studies are summarized in online supplementary Table 2. Fifteen studies (58%) did not make reference to any specific behaviour change theory or model within the text. When a theory was included, it was generally only briefly mentioned and was not thoroughly incorporated into the study design, or expanded upon in the discussion. No study specifically referred to the TPB, suggesting that researchers have yet to consider this important theory in their study design or interpretation of findings. Several studies incidentally considered certain aspects of the TPB in the development of their scientific methods or within the text, such as the consideration of *behavioural control* by assessing one or more social determinants of health, such as income [64]. Overall, behaviour change theory is not being thoroughly incorporated into genetic testing behaviour change research.

### Table 1 (continued)

<table>
<thead>
<tr>
<th>First author [Ref.], date</th>
<th>Participants (n baseline; n follow-up)</th>
<th>Intervention group(s)</th>
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</tr>
</thead>
<tbody>
<tr>
<td>Carpenter [59], 2007</td>
<td>Adult smokers (n = 729; n = 199)</td>
<td>High-risk genetic result</td>
<td>Non-risk genetic result</td>
<td>Emphysema (AAT)</td>
<td>3 months</td>
<td>Smoking</td>
<td>Those with high-risk genetic results made significantly greater quit attempts than the non-risk genetic result group (p = 0.004)</td>
<td>3 NR</td>
<td></td>
</tr>
<tr>
<td>Ito [60], 2006</td>
<td>Adult smokers (n = 697; n = 369 with data for baseline, 3 and 9 months)</td>
<td>Genetic testing</td>
<td>No genetic testing</td>
<td>Lung and oesophageal cancer (L-myc)</td>
<td>3 months 9 months</td>
<td>Smoking</td>
<td>NS differences in smoking cessation between groups at 3 months (significance level NR) or 9 months</td>
<td>1 NR</td>
<td></td>
</tr>
<tr>
<td>Marteau [61], 2004</td>
<td>Adult probands and their adult relatives with familial hypercholesterolaemia (n = 341; n = 275)</td>
<td>Genetic testing</td>
<td>No genetic testing</td>
<td>Familial hypercholesterolaemia (NR)</td>
<td>6 months</td>
<td>Nutrition (total fat and unsaturated fat), physical activity, and smoking</td>
<td>NS impact on nutrition, physical activity, or smoking with genetic testing</td>
<td>1 NR</td>
<td></td>
</tr>
<tr>
<td>McBride [62], 2002</td>
<td>Adult smokers (n = 557; n = 412 at 6 months, n = 356 at 12 months, n = 487 with data from all 3 time points)</td>
<td>Genetic testing</td>
<td>No genetic testing</td>
<td>Lung cancer (GSTM1)</td>
<td>6 months 12 months</td>
<td>Smoking</td>
<td>Greater smoking cessation in the genetic testing group than in the no-genetic-testing group (p &lt; 0.006) at 6 months; NS smoking cessation rates at 12 months</td>
<td>1 NR</td>
<td></td>
</tr>
<tr>
<td>Audrain [24], 1997</td>
<td>Adult smokers (n = 550; n = 426)</td>
<td>Genetic testing</td>
<td>No genetic testing</td>
<td>Lung cancer (CYP2D6)</td>
<td>12 months</td>
<td>Smoking</td>
<td>Greater likelihood of quit attempts in the genetic testing group than in the no-genetic-testing group (p = 0.02); NS change in 30-day cessation between groups</td>
<td>1 NR</td>
<td></td>
</tr>
</tbody>
</table>

COI, conflict of interest; EPA, eicosapentaenoic acid; DHA, docosahexaenoic acid; NS, not statistically significant (p > 0.05 unless otherwise stated); NR, not reported; DTC, direct to consumer; MUFA, monounsaturated fatty acid; PUFA, polyunsaturated fatty acid. 1 The rank of the study design is as follows, based on the categories of the NIH Quality Assessment Tools [29] in combination with consideration of the hierarchy of evidence [63]: 1 = controlled intervention study; 2 = observational cohort/cross-sectional study; 3 = case-control study; 4 = pre-post study with no control group. 2 Note: significance levels for this group of participants are reported in Bloss et al. [36].
### Table 2. Summary of quality assessment ratings and impact of genetic testing on lifestyle factor(s) of interest

<table>
<thead>
<tr>
<th>Ranking of study design</th>
<th>First author [Ref.], year</th>
<th>Quality assessment rating</th>
<th>Key findings: impact of genetic testing on lifestyle factor(s) of interest</th>
<th>Source of genetic information</th>
<th>Specific lifestyle factors with significant improvement</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Roke [43], 2017</td>
<td>Good Fair Fair 7</td>
<td>Δ</td>
<td>Other</td>
<td>N/A</td>
</tr>
<tr>
<td>1</td>
<td>Hietaranta-Luoma [9], 2015</td>
<td>Fair Good Poor 6</td>
<td>√² (2 weeks) √³ (6 months) √⁴ (12 months)</td>
<td>HCP</td>
<td>Improved dietary fat quality (high-risk genotype vs. control at 2 weeks and baseline to 6-month follow-up in high-risk genotype group); decreased intake of high-fat, high-sugar foods (in low-risk genotype vs. control at 12 months)</td>
</tr>
<tr>
<td>1</td>
<td>Marsaux [47], 2015</td>
<td>Fair Fair Poor 5</td>
<td>Δ</td>
<td>DTC</td>
<td>N/A</td>
</tr>
<tr>
<td>1</td>
<td>Meisel [44], 2015</td>
<td>Poor Fair Fair 5</td>
<td>Δ Δ</td>
<td>DTC</td>
<td>N/A</td>
</tr>
<tr>
<td>1</td>
<td>Voils [46], 2015</td>
<td>Fair Good Poor 6</td>
<td>√⁵ (3 months) √⁶ (6 months)</td>
<td>HCP</td>
<td>Reduced calories and fat (MUFA and PUFA)</td>
</tr>
<tr>
<td>1</td>
<td>Nielsen [11], 2014</td>
<td>Good Fair Poor 6</td>
<td>Δ (3 months) √ (12 months)</td>
<td>DTC</td>
<td>Reduced sodium intake</td>
</tr>
<tr>
<td>1</td>
<td>Hollands [50], 2012</td>
<td>Good Good Poor 7</td>
<td>Δ</td>
<td>Other</td>
<td>N/A</td>
</tr>
<tr>
<td>1</td>
<td>Hishida [52], 2010</td>
<td>Poor Poor Poor 3</td>
<td>Δ</td>
<td>HCP</td>
<td>N/A</td>
</tr>
<tr>
<td>1</td>
<td>Chao [55], 2008</td>
<td>Fair Poor Poor 4</td>
<td>√+a √b √c</td>
<td>HCP</td>
<td>General improvements to nutrition and PA; vitamin E supplementation was the most common change reported</td>
</tr>
<tr>
<td>1</td>
<td>Sanderson [56], 2008</td>
<td>Poor Fair Fair 5</td>
<td>√ (1 week) √ (2 months)</td>
<td>HCP</td>
<td>Fewer cigarettes smoked and greater smoking cessation</td>
</tr>
<tr>
<td>1</td>
<td>Rief [58], 2007</td>
<td>Fair Good Poor 6</td>
<td>Δ</td>
<td>HCP</td>
<td>N/A</td>
</tr>
<tr>
<td>1</td>
<td>Ito [60], 2006</td>
<td>Poor Good Fair 6</td>
<td>Δ</td>
<td>Other</td>
<td>N/A</td>
</tr>
<tr>
<td>1</td>
<td>Marteau [61], 2004</td>
<td>Fair Fair Fair 6</td>
<td>√d (6 months) √ (12 months)</td>
<td>HCP</td>
<td>Greater smoking cessation</td>
</tr>
<tr>
<td>1</td>
<td>McBride [62], 2002</td>
<td>Poor Good Fair 6</td>
<td>√d (6 months) √ (12 months)</td>
<td>Other</td>
<td>Greater smoking cessation</td>
</tr>
<tr>
<td>1</td>
<td>Audrain [24], 1997</td>
<td>Fair Fair Fair 6</td>
<td>√d (quit attempts) √ (30-day cessation)</td>
<td>HCP</td>
<td>Greater likelihood of quit attempts</td>
</tr>
<tr>
<td>Summary² (n = 15)</td>
<td>Fair Fair Poor 5.6</td>
<td>3/8 0/6 3/7 1/1</td>
<td>Greater health behaviour scores; the most common changes were &quot;healthier diet,&quot; &quot;more exercise,&quot; and &quot;taking vitamins or supplements&quot;; more often reported &quot;sufficient fruit and vegetable intake&quot;</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

1. Quality assessment rating: Good, Fair, Poor
2. Source of genetic information: HCP (Healthcare Provider), DTC (Direct-to-Consumer)
3. Specific lifestyle factors with significant improvement:
   - Improved dietary fat quality
   - Reduced sodium intake
   - General improvements to nutrition and PA
   - Fewer cigarettes smoked and greater smoking cessation
   - Reduced calories and fat
   - Reduced health behaviour scores
### Table 2 (continued)

<table>
<thead>
<tr>
<th>Ranking of study design</th>
<th>First author [Ref.], year</th>
<th>Quality assessment rating</th>
<th>Key findings: impact of genetic testing on lifestyle factor(s) of interest</th>
<th>Source of genetic information</th>
<th>Specific lifestyle factors with significant improvement</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>nutrition, PA, smoking, nutrition, and PA, and smoking</td>
<td></td>
<td>&quot;Changed their diet&quot; and &quot;started exercising more&quot;</td>
</tr>
<tr>
<td>2</td>
<td>Kaufman [49], 2012</td>
<td>Fair – poor</td>
<td>4 ✓ ✓</td>
<td>DTC + optional HCP</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>2/2 2/2 0/1</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>1/1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Summary² (n = 2)</td>
<td></td>
<td>Fair – poor</td>
<td>3.5</td>
<td>DTC</td>
<td>N/A</td>
</tr>
<tr>
<td>3</td>
<td>Marsaux [10], 2016</td>
<td>Fair – poor</td>
<td>5 ✓</td>
<td>HCP</td>
<td>Greater changes in supplement use; vitamin E, vitamin C, botanicals, multivitamins, vitamin B, and fish oil/omega were the most common changes reported</td>
</tr>
<tr>
<td></td>
<td>Vernarelli [51], 2010</td>
<td>Good – poor</td>
<td>5 ✓</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>O’Neill [54], 2008</td>
<td>Good – poor</td>
<td>5</td>
<td>HCP</td>
<td>N/A</td>
</tr>
<tr>
<td></td>
<td>Carpenter [59], 2007</td>
<td>Fair – poor</td>
<td>4 ✓</td>
<td></td>
<td>Greater 24-h quit attempts</td>
</tr>
<tr>
<td>Summary² (n = 4)</td>
<td>Good – poor</td>
<td>Fair – poor</td>
<td>5.0</td>
<td>N/A</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>1/2 0/3 1/1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>Boeldt [45], 2015</td>
<td>Fair – poor</td>
<td>5 ✓</td>
<td>DTC + optional HCP</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Bloss [48], 2013</td>
<td>Fair – poor</td>
<td>5 ✓</td>
<td>N/A</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Bloss [36], 2011</td>
<td>Fair – poor</td>
<td>4 ✓</td>
<td>N/A</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Quach [53], 2009</td>
<td>Fair – poor</td>
<td>5 ✓</td>
<td>N/A</td>
<td></td>
</tr>
<tr>
<td>Summary³ (n = 4)</td>
<td>Fair – poor</td>
<td>Fair – poor</td>
<td>4.8</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>0/4 0/4</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>Qualitative Rees [57], 2007</td>
<td>Good – poor</td>
<td>5 ✓</td>
<td>N/A</td>
<td></td>
</tr>
<tr>
<td>Summary³ (n = 1)</td>
<td>Good – poor</td>
<td>Poor – poor</td>
<td>5.0</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>0/1 0/1</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Summary of all studies (n = 26)  
FAIR POOR POOR 5.2  
NUTRITION: 6/18 (33%)  
PA: 2/16 (13%)  
SMOKING: 4/12 (33%)  
Studies with significant beneficial health behaviour change(s): 7/93 (78%) provided actionable recommendations  
Studies with null findings: 7/14 (50%) provided actionable recommendations

"Other" sources of genetic information: Roke et al. [43], 2017, used a researcher; McBride et al. [62], 2002, used "trained counsellors"; Ito et al. [60], 2006, used a "trained interviewer"; Hollands et al. [50], 2009, used a "trained research counsellor." No studies found a detrimental effect of genetic testing on lifestyle change. ✓, statistically significant beneficial behaviour change(s); ✗, no statistically significant behaviour change(s); blank cells, lifestyle factor(s) of interest was/were not assessed; N/A, not applicable; TPB, theory of planned behaviour; PA, physical activity; HCP, genetic intervention offered through a healthcare provider; DTC, genetic intervention offered direct to consumer; Other, another method was used to deliver the genetic intervention to the participants; MUFA, monounsaturated fatty acid; PUFA, polyunsaturated fatty acid.  
¹ The rank of the study design is as follows, based on the National Institutes of Health Quality Assessment Tools [29] and the hierarchy of evidence pyramid [63]: 1 = controlled intervention study; 2 = observational cohort/cross-sectional study; 3 = case-control study; 4 = pre-post study with no control group.  
² n = x indicates the total number of studies included in the summary; modes are reported for each of the three quality assessment ratings; x/x indicates the number of beneficial behaviour change findings/the total number of studies (note: several studies included multiple analyses such as those with more than one follow-up time point, and those assessing more than one lifestyle factor of interest); the overall quality score is represented as a mean.  
³ Three studies did not provide information about whether or not actionable recommendations were provided.  
⁴ High-risk genotype vs. control group.  
⁵ Baseline to 6-month follow-up in high-risk genotype group.  
⁶ Non-risk genotype vs. control group.  
⁷ Genetic testing group vs. control group.  
⁸ High-risk genotype group vs. non-risk genotype group.
Does Genetic Testing Impact Changes in Nutrition, Physical Activity, and/or Smoking Behaviour?

Overall. Given the heterogeneity of the literature and complexity of genetics-based behaviour change research, a cause-and-effect relationship between genetic testing and health behaviour change cannot be identified. Notably, it appears that it is unlikely that genetic testing has a “fatalistic” or negative impact on health behaviour change related to nutrition, physical activity, and smoking, since no study found that genetic testing negatively impacted the health behaviours of interest to the present review. Interestingly, 78% of the studies with health-promoting lifestyle behaviour change findings provided their participants with a genetics-based intervention that included actionable health behaviour recommendations. Examples of actionable recommendations provided to participants for each lifestyle factor included recommendations to reduce sodium intake (nutrition) [11], incorporate exercise into one’s daily routine (physical activity) [44], and quit smoking (smoking) [24]. Conversely, only 50% of the studies with null findings provided their participants with actionable health behaviour recommendations. Since an overarching cause-and-effect statement about the impact of genetic testing on behaviour change cannot be made, a best evidence synthesis is provided below.

Nutrition. Of the 18 articles that assessed a nutrition-related outcome, 6 (33%) showed a positive, health-promoting effect of genetic testing on behaviour change at one or more time points (both short term and long term, as further outlined in Tables 1 and 2). While this does not indicate that the majority of studies positively influenced nutrition, multiple studies of good quality have demonstrated that it is possible to facilitate healthier nutritional behaviours through the provision of genetic testing [8, 9, 11, 46, 49, 51].

Physical Activity. The provision of genetic testing to facilitate physical activity behaviour change does not appear to be as promising as behaviour change related to nutrition. Of the 16 studies that assessed physical activity-related outcomes independently, only 2 (13%) found positive influences of genetic testing on physical activity [8, 49], with follow-up periods ranging from 2 to 8 months in one study [49] and the periods not indicated in the other study (follow-up varied for each participant) [8]. However, these articles rated poorly in their overall quality assessment, with “poor” to “fair” quality ratings of 3 [8] and 4 [49].

Smoking. Similar to nutrition, 4 (33%) of the 12 genetic intervention studies had a positive influence on smoking-related behaviours. However, improvements in smoking-related behaviours were generally only sustained over a short-term period. The overall quality of these studies was “fair.”

Sleep. It is clear that sleep is an understudied area of genetic testing and behaviour change research, since our comprehensive search did not yield a single study that assessed sleep (sleep quality, hours of sleep, etc.) as a behaviour change outcome.

Pooled Analyses. Two studies completed pooled analyses of changes in more than one lifestyle factor. Chao et al. [65] did not find significant changes in nutrition or physical activity on their own, but when pooled together, there were significantly greater changes to nutrition and physical activity in the high-risk genetic testing group than in the non-risk and control groups. Additionally, in a pooled analysis of changes to nutrition, physical activity, or smoking, Egglestone et al. [8] found significant changes between the genetic testing group and the control group. However, their results should be interpreted with caution, as this study was awarded the lowest overall quality rating of 3 (Table 3).

Results from Controlled Intervention Trials

While it is important to be comprehensive and consider all studies conducted on the topic of interest regardless of the research methods chosen, controlled interventions should be further highlighted and reviewed separately from other study designs given that this is the highest possible level of evidence for the original research included in the present review.

In total, 15 controlled intervention trials have been conducted over the past two decades. Approximately half of these studies (n = 7; 47%) found significant changes in nutrition and/or physical activity or in smoking at 1–3 time points included in the study. Consistent with the overall analysis, the controlled interventions found that nutrition was the most promising area of behaviour change, followed by smoking (short-term only).

The genetic interventions in the controlled intervention trials overall ranked “fair,” demonstrating that in comparison to the result of the pooled analysis of all study designs, these studies provided their participants with higher-quality genetic interventions. This may help explain why 47% of the controlled intervention studies found significant changes in lifestyle habits resulting from the genetic intervention, compared to 36% of the studies using other study designs. The overall ranking of these studies was “fair,” with a mean rating of 5.6 out of the highest possible score of 9. Risk of bias overall was
“fair” and consideration of the TPB was rated to be “poor,” which is consistent with the results of the analysis of all study designs combined.

### Discussion

Given that decoding the entire human genome was the primary focus of genetic research until 2003 [1], it is not surprising to find that the majority of studies included in the present review were published after this time, with only 2 studies published before 2003. Since then, much greater focus has been placed on genetic testing behaviour change research pertaining to nutrition, physical activity, and smoking. However, several studies included in the present review (46%) did not include a COI statement. Future research should ensure the inclusion of a COI statement given this concerning finding and given the increased emphasis in academia on the importance of considering COI in genetic testing and other research.

Improving one or more of the four lifestyle behaviours of interest to this review has been shown to have a beneficial effect on chronic disease management and general health and well-being [20–23]. The present review indicated that improvements to smoking habits were promising in the short-term. This finding was consistent with that of a previously published systematic review of the impact of genetic notification on smoking cessation [66].

While nutrition, physical activity, and smoking habits have been researched in multiple genetic intervention studies, sleep remains an understudied area of genetics and behaviour change. This is notable considering the substantial impact that sleep has on overall health and well-being. Current systematic reviews demonstrate a significant impact of sleep on cognition and emotion [67], glycaemic control [22], and overweight or obesity [23], to name a few. To our knowledge, little is known about the ability of sleep to modify gene-associated health risks. Thus, future research should seek to first determine gene-sleep interactions that may influence health outcomes using methodologies similar to those of nutrigenomics research, as opposed to a genome-wide association study approach. Upon determining ways in which sleep may mitigate genetics-associated health risks, future research should then seek to determine if genetic testing helps to motivate healthy sleep-related behaviours.

Of the studies that reported the specific genes tested in the genetic intervention, single nucleotide polymorphisms in 16 unique genes were tested, with apoE, BRCA1/2, FTO, and GSTM1 having the highest frequencies of use in the genetic intervention.
recommendations (e.g., the recommendation to reduce sodium intake [11]).

It is important to note that our risk-of-bias results are consistent with the previously published literature [3, 6], providing validation for the NIH quality assessment process completed in the current review. Effect sizes were not included in this review due to heterogeneity of the genetic interventions and study designs of the included articles that would have introduced potential flaws in effect size calculations and any conclusions drawn from such calculations. For randomized controlled trials, effect sizes have recently been presented elsewhere [3], although these should be interpreted with caution due to the significant heterogeneity of treatments (genetic interventions), measurements of outcomes, and populations studied. To our knowledge, we have developed and utilized the first quality assessment tool for evaluating and rating genetic interventions. Future research should seek to utilize this novel tool and significant contribution to the literature to assess the quality of genetic interventions in both primary research and systematic reviews. Furthermore, the components of this tool can be used in future genetic testing behaviour change study design to improve the quality of genetic interventions provided to participants (online suppl. Table 1). Although the genetic intervention quality assessment was based on previously published robust research and critical commentaries [11, 31–38], assessing the quality of evidence supporting the genetic tests provided to participants was beyond the scope of the present review. This is an important area of future research and is a notable ethical concern of genetic testing.

This review provides the most comprehensive analysis of genetic testing behaviour change research completed to date. However, some limitations to the present review exist. While this review summarized whether the genetic information was delivered direct to consumer or through a healthcare provider (Table 3), the practice of each provider is inevitably distinct. Some may incorporate behaviour change theory into their practice in order to maximally promote health behaviour change, while others may simply provide an explanation of the genetic results. This limitation further highlights the complexity of genetic testing behaviour change research. Additionally, the TPB was chosen as the key theory of interest given that it is one of the most widely accepted and validated theories of behaviour change, with over 4,500 publications referencing this theory and several meta-analyses finding that the key components of the TPB can be used to predict behavioural intentions with mean multiple correlations ranging from 0.59 to 0.67 [15, 68–73]. However, a number of other theories have been validated and are frequently used in behaviour change research, such as the transtheoretical model [74].

By improving upon genetic testing behaviour change studies, we anticipate the development of an algorithm that can be used to inform effective genetic testing behaviour change interventions for individuals who might benefit from this more personalized approach to healthcare. Indeed the limitations of genetic testing and the possible risk of harm [75] should be considered prior to an individual’s decision to undergo genetic testing, especially in situations where one may learn about their risk of developing a disease, where actionable strategies for mitigating the risk are currently unknown [75]. Given that behaviour change is complex and multifactorial and studies have yet to robustly incorporate validated theory and high-quality genetic interventions into their methods, we cannot conclude with a broad statement about the impact of genetic testing on behaviour change. However, it is clear that it is possible to facilitate behaviour change through the provision of high-quality genetic interventions. Incorporating behaviour change theory into future research is an important consideration to enhance our knowledge in this field. Specific recommendations for study design have recently been published elsewhere [18]. An interdisciplinary research team with expertise in genomics as well as behaviour change may be the optimal approach given the complexities of this field of study. Considerable future research is needed in this promising and exciting area of lifestyle behaviour change research.

Conclusion

The use of validated theory to inform a robust study design [18] and the provision of actionable, high-quality, genetics-based information and advice is recommended to test a behaviour change hypothesis in genetics research. Rather than using the traditional systematic review process of assessing solely risk of bias, we have demonstrated that factors beyond risk of bias influence research outcomes related to genetic testing and behaviour change. As more robust literature continues to be published, allowing for the determination of key components of genetic interventions that best facilitate behaviour change, lifestyle genomics behaviour change research has the potential to make a substantial impact on global health and well-being through the facilitation of personalized, health-promoting lifestyle behaviour change.
References


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Disclosure Statement
The authors declare that they have no conflicts of interest.
Genetic Testing and Lifestyle Behaviour Change


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