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What is This?
Individuals’ experiences of, and responses to, a negative genetic test result for familial hypercholesterolaemia

Jennifer Hilgart¹, Jenny Mercer² and Kathryn Thirlaway²

Abstract
This study aimed to explore the responses of individuals who have undergone genetic testing for familial hypercholesterolaemia (FH) where no genetic mutation has been identified. Semi-structured interviews were conducted with 11 patients and interpretative phenomenological analysis (IPA) was employed. This article describes three inter-related themes: ‘feeling in limbo’, ‘exploring causes of raised cholesterol’ and ‘contradictions in talk about diet’. Although participants generally adhered to medication and engaged in healthy lifestyles, the findings have clinical implications for how genetic test results are communicated.

Keywords
coronary heart disease, genetic testing, interpretative phenomenological analysis, responses, risk

Introduction
Familial hypercholesterolaemia (FH) is a common inherited condition which results in elevated cholesterol levels. FH is estimated to affect around 1 in 500 people, at least 75% of whom are undiagnosed in the United Kingdom (Neil et al., 2000). People with FH, if untreated, are at an increased risk of early coronary heart disease (Humphries et al., 2006), but this risk can be significantly reduced through treatment with cholesterol lowering medication, such as statins, and the adoption of a healthy lifestyle (Simon Broome Steering Committee, 1991).

FH can be diagnosed by the presence of a combination of clinical indicators, such as high blood cholesterol levels, cholesterol deposits on the tendons, knuckles or knees, premature coronary heart disease and a family history of heart disease or high cholesterol (Simon Broome Steering Committee, 1991). A clinical diagnosis of FH can now also be confirmed with genetic testing, which identifies mutations in the genes currently known to cause FH. At present, not all mutations likely to cause FH have been identified (Taylor et al., 2010), and if...
no genetic mutation is found it could mean that high cholesterol is due to other FH genes which cannot yet be tested for, or it could mean that high cholesterol levels are not due to FH.

Unlike other genetic conditions, such as Huntington’s Disease or hereditary cancer, people with FH can be effectively treated with statins, which may explain why a majority of FH patients regard FH as highly controllable and downplay their risk of heart disease (Claassen et al., 2010; Senior et al., 2002). In a study by Weiner and Durrington (2008), patients with a clinical diagnosis of FH who had not undergone genetic testing did not explain their FH in genetic terms, but also regarded FH as controllable and heart disease as avoidable. Although many individuals with FH underestimate their risk of heart disease, up to 96% of FH patients receive and adhere to long-term treatment with statins (Hadfield et al., 2008; Pijlman et al., 2010). It has also been shown that those with a confirmed genetic diagnosis of FH do not become more anxious after receiving their test results (Marteau et al., 2004) and have similar long-term quality of life scores to those who screen negative for FH (Van Maarele et al., 2003).

With the exception of Senior et al. (2002), most of the qualitative research conducted to date with FH patients has involved those with a confirmed genetic diagnosis of FH (e.g. Agard et al., 2005; Frich et al., 2006). Therefore, individuals who have a clinical diagnosis of FH, but in whom no genetic mutation has been found, represent an interesting group whose experiences have not been fully explored in the qualitative studies conducted so far. One quantitative study did consider those with a negative result, finding that participants believed their FH and risk of heart disease was less controllable with medication and lifestyle changes, compared with those in whom a genetic mutation was found (Marteau et al., 2004). Marteau et al. (2004) also concluded that while there may be clinical and psychological advantages of confirming a FH diagnosis using a DNA test, there may be some small, adverse psychological effects of unsuccessful searches.

The present study adds to the growing literature on the psychosocial implications of genetic screening for FH, by conducting in-depth qualitative interviews with individuals who have a clinical diagnosis of FH, but in whom no genetic mutation has been found. As there is limited research about these individuals’ experiences of genetic testing, the findings of this study will increase understanding of the impact of negative genetic test results and may have implications for the information and support these individuals receive after testing. The increasing ability to test for genetic susceptibility for common diseases such as heart disease and diabetes means that it is especially important to understand people’s emotional and behavioural responses to genetic testing. When no genetic mutation is found from FH genetic testing, patients are informed that their diagnosis cannot be confirmed, either because current technology cannot identify all mutations in the FH genes, or because there are other FH genes for which testing is not yet available. A negative test result can also mean that the patient’s high cholesterol levels are not due to FH. Therefore, the ambiguous meaning of a negative test result is potentially confusing for the patient. The current study aims to explore this further by looking at individuals’ responses to FH genetic testing, where no genetic mutation has been found.

Method

Phenomenological approach

A phenomenological methodology was utilized to enable a detailed examination of people’s lived experience and how they make sense of their experiences with regards to FH. With its grounding in phenomenology and hermeneutics, interpretative phenomenological analysis (IPA) attempts to understand the participant’s experience through their personal account of it, while acknowledging that this is necessarily an interpretative process. Chapman and Smith (2002) suggested that IPA’s flexible and detailed methodology is a particularly useful approach for examining the psychological aspects of
‘new genetics’. Indeed, IPA has been successfully applied in other qualitative studies of genetic testing and risk perceptions such as in Huntington’s Disease (Smith et al., 2002) and hereditary breast and ovarian cancer (Buckmaster and Gallagher, 2010; Etchegary et al., 2009).

**Participants**

Participants were recruited from the Welsh FH Cascade Screening service in South Wales, United Kingdom. Males and females over 18 years old, who were sufficiently proficient in English to take part in the interview, and who had undergone genetic testing for FH but in whom no mutation was found, were eligible for participation in the study. Eleven participants, five males and six females, aged between 40 and 79 were interviewed for this study. Participants’ names and identifying information have been changed to protect their anonymity. Although this relatively small sample may not be representative of all patients in the FH service, the method of analysis used in this study (IPA) is reportedly most suited to a small, homogeneous group of no more than 15 people (Smith and Osborn, 2003). The number of participants involved in this study is also comparable with other methodologically similar studies (e.g. Senior et al., 2002; Weiner and Durrington, 2008), as well as through consultations with clinical staff from the FH service. In the interviews themselves, information about family history of heart disease and high cholesterol was elicited after participants were asked about their perceptions of FH, in order to avoid pre-framing FH in a genetic way. With the participants’ consent, each interview was recorded with a digital audio recorder and transcribed verbatim. The transcripts of the interviews served as the raw data to be analysed using IPA.

**Analysis**

In the first stage of analysis, each transcript was read several times to achieve familiarity with the data. Initial notes of any observations or reflections that occurred while reading were recorded in the margin of the transcript. The second stage involved reading each transcript and the initial comments to identify emergent themes. Themes were constructed inductively, using the participants’ accounts, as well as deductively, based on the interpretations of the researcher. Patterns and connections between the themes were considered. During this stage some themes were discarded and others were reorganized into clusters or concepts. The clustering of themes into associated groups then led to the development of a label for that cluster of themes (a ‘super-ordinate’ theme). A table of the super-ordinate themes and subordinate themes that emerged from the analysis was created for each participant. As suggested by Smith et al. (2009), identifying quotations reflecting each theme were included in order to aid the
organization of the analysis. This process was repeated with each transcript and new themes were allowed to emerge with each case. The next step involved looking for patterns across cases and noting any similarities and differences between participants’ accounts. In particular, it was noted where themes or narratives from a single interview contrasted with the data more generally. The transcripts were re-read throughout this process to ensure the analysis was fully grounded in the data. Themes which did not capture an essential aspect of the phenomenon under scrutiny or which were not well represented in the text were discarded.

Results

For each participant, FH genetic testing was seen as a means of confirming the cause of both their own and their family’s raised cholesterol and/or heart disease. The term ‘heart disease’ encompasses a number of different heart conditions, including angina, heart failure and heart attack. These terms were used interchangeably by participants throughout the interviews to describe various heart conditions present in their family. Most participants underwent FH genetic testing as they were anxious to confirm the cause of the heart problems they had witnessed in their families and, in some cases, in themselves too. These illnesses had been significant events in the participants’ lives, and had led to a heightened awareness of their personal vulnerability. Three inter-related themes regarding participants’ responses to their negative FH genetic test result are highlighted in this article: ‘Feeling in limbo’; ‘exploring causes of raised cholesterol’; and ‘contradictions in talk about diet’. In accordance with Smith (2011), each theme was present in at least half of the interviews.

Feeling in ‘limbo’

The main features that characterized the feeling of ‘limbo’ were the lack of a conclusive test result and an uncertainty about what would happen next. Many participants expected that the FH genetic test would detect a mutation because of their family history of heart disease, and reported feeling both ‘relieved’ and ‘disappointed’ in response to their negative genetic test result. Jane, for example, was reassured that a genetic mutation had not been found but also, like many other participants, she remained uncertain about the true cause of her raised cholesterol:

Well I just felt pleased that it wasn’t positive in a way because obviously it’s something that you haven’t got wrong with you [laughs], but also I’m back where I started several years ago not knowing why I’ve got high cholesterol.

Feelings of uncertainty about the genetic test result were conveyed in nearly all of the participants’ narratives, which appeared to stem from the ambiguous nature of the negative result. Many interpreted their result as meaning that they did not have FH or a genetic condition, which conflicted with their firmly held beliefs regarding the hereditary nature of their condition. Participants did not regard the genetic test result as conclusive evidence that there was no genetic aspect to the heart problems inherent in their families. For example, Gethin described his uncertainty about his genetic test result and the implications for his children:

I’m still sceptical because there’s just so much of it in my family … I know this, what the results are saying there isn’t, it’s not hereditary, but I’m still not going to take anything for granted. It may be that my kids do still have it, I don’t know.

Jackie also described a strong family history of heart problems and due to her own recent diagnosis of heart failure, Jackie believed she had a ‘rarer form of FH’ that was not identified by the genetic test. As illustrated in the following quotation, she had a desire to ‘label’ the cause of the cardiac problems in her family, believing that this would help reduce the uncertainty and anxiety surrounding her condition:
I’m still in this quandary as to have I got this rarer one, or haven’t I? … and given my history now, it’s most probably I have … I mean it’s easier to put a label on it, because you can say oh right I’ve got duh duh duh, rather than thinking well have I got it or haven’t I? … ‘Cos I sort of feel in a bit of a limbo really.

Glynn’s family history of heart disease was less salient. Unlike the majority of other participants, he readily accepted the result as meaning that there was no genetic aspect to his raised cholesterol:

JH: So how did you feel when you got the genetic test result?

Glynn: Well I mean I didn’t really feel any different but um, it’s nice to know that it’s not hereditary you know.

Eight individuals reported concerns about the implications of the genetic test result for their own treatment and for their children’s treatment in the future. They reported being unsure as to ‘what the next step is’:

I don’t know where it goes from here now you know does, do I see [the FH nurse] again? You know, I might have to do more bloods, I don’t know … all I know is she told me I wasn’t carrying it. (Barbara)

Christopher and Michael questioned whether their children were at less risk of developing heart disease, and also whether they would be entitled to the same healthcare as they would have, had a genetic mutation for FH been identified:

It would be assuring to know that they would be watched and monitored as they are growing up … I don’t know now, because I haven’t had a positive test, if they have the same level of care. (Christopher)

Presumably if there is any sort of different kind of treatment or longer term treatment … if it’s definitely a sort of family link, that would have been nice, but unfortunately it wasn’t, so I can’t do anything about it. (Michael)

The lack of a clear diagnosis or label appeared to make it difficult for participants to have clear expectations about their own, and their children’s, future health and healthcare eligibility, in relation to FH. Michael states a feeling of lack of control over his future care (‘I can’t do anything about it’), while Terry, although feeling ‘in limbo’, was reassured that his cholesterol was being monitored and managed:

… does it make you feel better to know that you haven’t got the gene or worse? And the outcome is it does leave you in limbo, you don’t know why you’ve got the defect. The most important thing is that it’s been detected and you’re being treated for it isn’t it?

Exploring causes of raised cholesterol

Most participants interpreted the negative FH genetic test result as meaning that their condition was not genetic, and also expressed uncertainty about the cause of their condition. However, in response to this uncertainty, some individuals engaged in the process of exploring alternative causes to their raised cholesterol. Ten participants still attributed their raised cholesterol to genetics, or to a combination of genetics and lifestyle factors. Those who were strongly aware of their family history were especially convinced of ‘a link of sorts’ to account for their family and personal history of raised cholesterol and heart disease. By identifying causal factors which contributed to their cholesterol levels, some participants were able to make sense of their condition and their risk of heart disease, regardless of the inconclusive FH genetic test result. For example, in Terry’s family, adherence to healthy behaviours did not seem to reduce vulnerability to heart disease. The use of ‘I still consider’ suggests he has drawn upon his previously held beliefs about the genetic nature of his family’s heart conditions:
It just seems strange doesn’t it … if it was one generation you might say well, we all got the same lifestyle, we all drink too much … But when you’ve got in those nine [siblings with heart conditions], smokers, non-smokers, overweights, underweights, there’s no rules there anywhere at all. Technically I still consider there’s got to be something within our genes. (Terry)

In other participants’ accounts, it was thought that high-fat diets, sedentary lifestyles and smoking could explain the heart problems in older generations of the family but could not fully account for their own raised cholesterol, and it was this that led to the belief in a genetic aspect to their condition. In fact, many participants were surprised when they found out they had high cholesterol, as they already adhered to a healthy lifestyle and maintained a healthy weight. Therefore, it seemed to them that the only plausible explanation for their cholesterol levels could be genetic:

I weigh eight and a half stone which isn’t much … I’ve always exercised and my diet’s good … I feel that there must be something that I’m, in, in to me that’s giving me six point five [cholesterol level]. (Gloria)

it’s got to be, as they say, a gene in my mother and me because I certainly don’t eat fats. (Marie)

By contrast, Barbara’s knowledge of her family history was limited and she reported being uncertain about the cause of her raised cholesterol and her family’s heart disease:

‘is it FH or something that’s genetic through the family or is it because we’re big? Is it one of those things? I don’t know.’

Glynn was also unsure about his family history, and was the only participant to interpret the FH genetic test result as meaning his high cholesterol was not hereditary, therefore concluding that dietary factors must have played a role:

‘if it’s not hereditary it was obviously my diet, what I was eating’.

The above quotations suggest that those who perceived their diet and lifestyle to be healthy were less willing to dismiss genetic explanations for their raised cholesterol than were those who reported having a poor diet. For many, the belief in a genetic cause was further reinforced by the ineffectiveness of dietary change in reducing cholesterol levels. Regardless of this, participants generally reported that they adhered to a healthy lifestyle.

Contradictions in talk about diet

Although no FH genetic mutation had been identified from the genetic test, all participants had raised cholesterol. For many, concerns over establishing a genetic cause of their FH were overtaken by concerns about managing their current and future health status. All participants believed that their condition could be controlled through both cholesterol-lowering medication and a healthy diet, but were dependent upon statins because they produced observable reductions in their cholesterol levels. The apparent lack of efficacy of ‘going on things like low-fat foods’ was a recurrent theme in many participants’ accounts. All participants reported having attempted to lower their cholesterol by adhering to a healthy diet and exercising, or else had always maintained a healthy lifestyle. However, many reported that this did not produce any reduction in their cholesterol levels:

‘I met the dietician and I had a go on the chicken and God knows what and eat all the good things in life, but it didn’t really make much difference, didn’t make any difference at all’ (Glynn).

While it was thought that a low-fat diet may help reduce ‘other cholesterol’, many believed it would fail to control FH or ‘inherited cholesterol’. As a result, participants commonly believed that they were not personally culpable for their raised cholesterol. The following quotation illustrates how Terry distinguished between normal and inherited raised cholesterol, as he accounted for the poor efficacy of dietary modifications:
the thing is about inherited FH, inherited cholesterol, it does not matter what you do, you will not bring it down. That’s conclusive. You might vary it from 8 to 7.8 between each time, but you’re never going to get it down to the limits or the acceptable limit of 4 … only a statin works.

Interestingly, despite giving genetic explanations for their FH and maintaining that dietary modifications were ineffective in reducing cholesterol levels, all participants still felt obliged to demonstrate that they were leading a responsibly healthy lifestyle. For example, Gethin believed that any changes in his diet were unlikely to have any influence on his cholesterol level:

My understanding of it is that even if you did change your diet and I went on a really healthy diet it’s still never going to get my cholesterol down to the proper levels that it should be. It may get it down from say 6.5 to 5.8, but it’s not going to take you from 5.8 down to 4.1.

However, he still described at length his attempts to improve his diet and reduce his alcohol intake:

I don’t smoke, I don’t drink much nowadays, so yeah I’m feeling a lot happier about it than say I was three years ago … in terms of what I’m putting in my mouth, you know, it’s not all the real fatty foods … like it used to be.

Medication was identified as the main source of control over cholesterol levels but lifestyle and diet were also constructed as critical factors that can mean the difference between health and ill-health. All participants reported self-surveillance by monitoring their eating habits and exercise. In this way, participants were assuming responsibility for taking care of themselves and, in some cases, their families. This ethic of care was believed to be a crucial benefit for their overall health. The following extract suggests that maintaining a healthy diet absolved Gloria of blame for any future increases in her cholesterol levels:

‘I am personally though making an effort with my diet, my exercise and my weight, so that I can’t blame myself for that’ (Gloria).

Several participants believed that a high-fat diet could ‘override’ the beneficial effects of their medication. In these cases, adherence to a healthy lifestyle seemed to provide an extra sense of control, allowing participants to feel that they were doing everything possible to reduce their risk of illness:

I’m just pleased the cholesterol is right down low. And it can make you think oh well I can rely on the pills, I can eat all the sugary things and just rely on the pills, but I’m not doing that. So you can get into that phase of mind can’t you, but no [I’m] watching what I eat and that. (Barbara)

**Discussion**

The themes presented here highlight responses to a negative FH genetic test result. For most participants, genetic testing failed to provide a conclusive explanation for the cause of their hypercholesterolaemia. Many did not understand that a negative test result does not represent conclusive proof that high cholesterol is not inherited, and interpreted their own test result as meaning their condition was not genetic. Many participants, following their negative test result, also expressed feelings of uncertainty about the associated risks for their children. These findings are comparable to those from studies of women undergoing genetic testing for breast and ovarian cancer, where it has been found that many patients view genetic testing as important in allowing them to be certain about their own risk and their children’s risk (Meiser et al., 2000), to confirm their perceptions of the genetic aetiology of the disease (Hallowell et al., 2004), and also in granting them eligibility for a high level of specialist care (Buckmaster and Gallagher, 2010). A study of genetic testing for familial adenomatous polyposis (FAP), which causes potentially cancerous polyps in the bowel, also showed that a negative test result failed to reassure patients, who still perceived a continued risk to themselves and their children (Michie et al., 2003).
In the present study, the results surrounding participants’ uncertainty about the nature of their condition have implications for how FH genetic test results are communicated to patients, since it was found that feelings of uncertainty were exacerbated by a lack of information. For example, those who received their result by letter felt they would benefit from discussing their result with a health professional from the lipid clinic, in order to clarify what the test implied for their future care. Similarly, in the study by Michie et al. (2003), it was found that many patients expressed a desire to continue with bowel screening, even though their genetic test result meant that screening was not clinically recommended. The present findings suggest that, prior to FH genetic testing, it would be helpful if patients were advised by clinicians to consider the likelihood of no genetic mutation being found. It may also be beneficial to patients for clinicians to explain what a negative genetic test result would mean in terms of the cause of their condition, how their FH healthcare would continue, and how their test result would impact upon their children’s options for treatment and surveillance.

In terms of Leventhal’s common-sense model of illness (CSM) (Leventhal et al., 2003), it appears that participants’ uncertainty was focused around the identity (symptoms and names) and the causes of their condition. Uncertainty about the cause of hypercholesterolaemia seemed to stem from the genetic test result conflicting with participants’ prior beliefs about there being a genetic component to their raised cholesterol. Such participants maintained a belief that their condition was genetic, even after receiving the negative test result. They justified this by drawing upon their previously held beliefs about the genetic nature of their condition and the fact that heart problems were so prevalent in their families. Other participants maintained that they had always led a relatively healthy lifestyle, and therefore believed that the presence of a genetic cause was the only plausible explanation for their raised cholesterol. However, while many felt certain of a hereditary component to the heart problems in their families, they also acknowledged that lifestyle factors could trigger or override hereditary risks. Participants’ causal representations of their condition were guided by their personal experiences of heart problems, which led many to believe in this multi-factorial model. Similar results have indeed been found in a study of people with a family history of heart disease (Walter and Emery, 2006), where participants believed that hereditary risks may be offset by other factors, such as diet and smoking. Also, in a focus group study on public understandings of genetic risks for heart disease, a majority of participants acknowledged that both environmental and genetic risk factors contribute to the onset of heart disease (Bates et al., 2003).

The present study demonstrates an absence of fatalism (the belief that little can be done to reduce genetic risks) in participants’ accounts. A lack of fatalistic responses to genetic testing has also been shown in other studies of FH patients (Senior et al., 2005; Weiner, 2009), and in a recent systematic review of responses to genetic risk information (Collins et al., 2011). Such findings dispute the hypothesis that genetic testing leads to fatalistic beliefs about control over the risk of illness (Marteau and Lerman, 2001; Senior et al., 1999). All participants in the current study felt that control over their cholesterol levels could be exerted through medication and lifestyle. Marteau et al. (2004) also concluded that when risk is modifiable, as it is with FH, information about a genetic aetiology does not decrease perceptions of control. However, Marteau et al. (2004) did find that a genetic diagnosis of FH weakened participants’ belief in the effectiveness of a low-fat diet in reducing cholesterol. This finding has been replicated in further studies, where identifying a genetic mutation for FH increased the perceived efficacy of medication and decreased the perceived efficacy of a healthy lifestyle (Senior and Marteau, 2007; Senior et al., 2005). Nevertheless, these studies have reported no relationship between FH genetic testing and patients’ adherence to medication and a healthy lifestyle.
Similarly, in the current study, participants believed they had an ability to reduce or control their familial risk of heart disease by engaging in health behaviours (low-fat diet, exercise, non-smoking), even though diet appeared to be relatively ineffective in reducing cholesterol levels. This contradiction in participants’ accounts conflicts with the CSM’s proposal that people will only comply with preventative recommendations (e.g. low-fat diet) if the recommendation corresponds with their representations of risk (e.g. the belief that diet is a causal factor). In the current study, participants talked about the lack of efficacy of dietary modifications in reducing their cholesterol levels, but also reported being careful about what they ate. This contradiction could be explained by participants’ belief in the potential for lifestyle factors to override hereditary risks. Also, while dietary change may not produce observable reductions in cholesterol levels, a healthy diet was considered beneficial for overall health. Maintaining a sense of being healthy seemed to assure participants that they were doing all they could to lower their chances of developing the heart problems that had been so prevalent in their families.

In the current study it was not possible to gauge fully whether awareness of FH or undergoing genetic testing changed people’s perceptions of the efficacy of risk-reducing health behaviours, as has been explored in longitudinal studies (e.g. Marteau et al., 2004). To extend the current study, it would be interesting to conduct interviews with patients both before and after FH genetic testing, to explore qualitatively the effect of providing genetic risk information on risk-perceptions, illness representations and health behaviours, of those both with and without a genetic mutation. Although some participants stated that they were already aware of a familial link before they were referred to the lipid clinic, it is not clear if knowledge and experience of FH and genetic testing increased participants’ focus on genetic and hereditary factors, as has been suggested in previous research (Marteau et al., 2004; Shiloh, 2006), and which would be a useful avenue to explore in future studies. Finally, all participants in the current study, except two, reported being highly adherent to their cholesterol-lowering medication, which is similar to the high rates of medication adherence described in other studies of FH patients (Classen et al., 2010; Umans-Eckenhausen et al., 2001; Van Maarle et al., 2002). The two participants in the current study who did not take their statins as recommended, rationalized their choice in terms of the side-effects of the statins. Previous FH studies have not explored the impact of the side-effects of statins on patients’ adherence to medication, which warrants this as an important area for further research.

Competing Interests
None declared.

References
Collins RE, Wright AJ and Marteau TM (2011) Impact of communicating personalized genetic risk information on perceived control over the


