Assessing family history of heart disease in primary care consultations: a qualitative study

Ruth Hall[a], Paula M Saukko[a], Philip H Evans[b], Nadeem Qureshi[c] and Steve E Humphries[d]

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Background. Current primary prevention guidelines recommend the assessment of family history of coronary heart disease (CHD) to identify at-risk individuals.

Objective. To examine how clinicians and patients understand and communicate family history in the context of CHD risk assessment in primary care.

Methods. A qualitative study. Patients completed a validated family history questionnaire. Consultations with clinicians were video recorded, and semi-structured interviews conducted with patients after consultation. The participants were 21 primary care patients and seven primary care clinicians (two practice nurses, five GPs). Four practices in South West England.

Results. Patients and clinicians usually agreed about the patient's level of risk and how to reduce it. Patients were mostly satisfied with their consultations and having their family history assessed. However, three issues were identified from the consultations which contributed to concerns and unanswered questions for patients. Problems arose when there were few modifiable risk factors to address. Firstly, patients' explanations of their family history were not explored in the consultation. Secondly, the relationship between the patient's family history and their other risk factors, such as smoking or cholesterol, was rarely discussed. Thirdly, clinicians did not explain the integration of family history into the patient's overall cardiovascular disease risk.

Conclusions. Clinicians appeared to lack a rhetoric to discuss family history, in terms of capturing both genetic and environmental factors and its relation to other risk factors. This created uncertainties for patients and carries potential clinical and social implications. There is a need for better guidance for primary care clinicians about family history assessment.

Keywords. Communication, coronary heart disease, family history, primary care.

Background

Epidemiological and genetic studies indicate that a family history of premature heart disease is an independent risk factor for coronary heart disease (CHD).[1,2] Assessing family history could enable better identification and management of individuals at increased risk.[3] The National Service Framework[4] and Joint British Societies' guidelines[5] recommend assessing family history in primary prevention of CHD.

European physicians rarely assess family history of CHD systematically and risk assessment tools seldom include family history.[6]

While the epidemiological significance of a family history of CHD is clear, the precise reasons for this are not. Family history reflects complex gene–environment interactions. Both genetic and environmental factors related to heart disease aggregate in families,[2] and family history can interact synergistically with other risk factors, such as smoking to produce multiplicative effects.[7]

Family history forms an integral part of lay people's understanding of heart disease and is thought to differ significantly from clinical understandings.[8–10] Epidemiologists classify family history by genetic relatedness, age of disease onset and number of relatives affected. Lay people consider the degree of similarity to relatives (in terms of lifestyle or physical resemblance), and are influenced by emotional experiences and closeness to relatives in assessing their own family history.[8,9,11] It has been suggested that these differences between lay and medical understandings of familial risk could create misunderstandings in the clinical encounter,[8,9] but no research has examined this. This study examines patient–clinician communication and patients' understandings of family history in the context of CHD risk assessment in UK primary care, using a validated family history tool.[12]

Participants and methods

Recruitment
Patients were recruited from four general practices in South West England (Exeter). Eligible patients were aged 18–55 (men) or 18–65 (women) without a history of heart disease, diabetes or familial hypercholesterolaemia and not prescribed statins. Patients were recruited directly by clinicians when requesting a cholesterol test, or patients whose cholesterol had been tested in the previous 3 months were recruited by post.

Methods
All patients completed a family history questionnaire (FHQ) about heart disease among first- and second-degree relatives.[12] A clinical member of the team (NQ) evaluated the FHQs and the impact on CHD risk was categorized to one of three risk categories (Table 1). These categories incorporated the Joint British Societies' guidelines (premature CHD in first-degree relatives increases absolute risk by 1.5 times) but additionally incorporated the number of first-degree relatives affected and premature CHD in second-degree relatives.[1,13] The use of the FHQ sought to provide clinicians with more detailed and systematic information about the risk associated with family history than is ordinarily obtained from asking patients about their family history during consultations. Clinicians were sent their patient's familial risk stratification results with recommendations for adjusting cardiovascular disease (CVD) absolute risk scores.

Clinicians incorporated the CHD family history results into their usual method of CVD risk calculation, and patients were given their 10-year risk score and discussed primary prevention. These consultations were video recorded.

The interviewer (RH) reviewed these consultations and 2 weeks later interviewed patients. The open-ended, audio-recorded interviews explored patients' understandings of their risk, views of the assessment process and any concerns.

Analysis
Consultations and interviews were transcribed. The videos were watched and the transcripts read both during and after data collection, and early emerging themes were explored in subsequent interviews. RH performed the primary analysis using constant comparative method.[14] Themes were identified through an iterative process, whereby themes emerging from the interviews were used to inform further analysis of the consultations and vice versa. NVivo software aided coding. RH and PMS met regularly to discuss emerging themes; the final stages of thematic analysis involved both authors.

Results

Twenty-one patients were recruited and seven clinicians participated (two practice nurses, five GPs). The response rate for direct recruitment was 16/23 (70%) and 5/20 (25%) for postal. Patients completed the FHQ to varying levels of detail, recording heart disease, hypertension, cholesterol levels, medication and lifestyle of relatives. The socio-demographic background and clinical features of patients are shown in Table 2. Of the 10 with average family history risk, two had an unclear family history and four had family histories of hypertension or high cholesterol which were reported to clinicians.

Consultation pattern and main themes
All consultations followed a similar pattern; absolute risk scores were calculated and lifestyle changes and medication were discussed. Clinicians often stated that absolute risk scores were not exact but may be an over or underestimate and adjusted them in light of the

TABLE 1   *Risk scores for family history*

Risk category	Combinations of relatives with premature CHD	Increase in absolute risk score
Strong	Two first-degree relatives or one first-degree relative + two second-degree relatives	Approximately two times
Moderately high	One first-degree relative or two second-degree relatives	Approximately 1.5 times
Average	One second-degree relative (or insufficient information)[a]	No increase

[a]Insufficient information would include unclear diagnosis of relative (e.g. sudden death at 45) and/or patient aware of only very few relatives' health status.

TABLE 2 *Clinical, social and demographic features of participants*

Clinical features		Family history result	
Absolute risk score (%)[a]	Average	Moderately high	Strong
0–10	7	5	
10–20	3	1	4
20–30		1	
Social and demographic features			
	Number		
Age			
20–30	1		
30–40	0		
40–50	14		
50–60	6		
Sex			
Male	12		
Female	9		
Social class			
Class I and II	10		
Class III and IV	10		
Class V	1		

[a]Including family history score.

patient's lifestyle. Most participants had thought about their family history prior to completing the FHQ and often found it illuminative, 'seeing it in black and white' gave them a better idea of their family history (008). In most consultations, patients and clinicians agreed about the level of risk and how to reduce it. Patients spoke positively about the experience, retained information about overall levels of risk and were mostly satisfied with consultations. However, three interrelated issues occasionally created communication problems in the consultation, and contributed to patients' unanswered questions and concerns.

Explanations of relatives' heart disease
In the consultation, patients often spontaneously contributed information about cardiac events in their family and their explanations of these events. Similarities and dissimilarities from their affected relatives were highlighted in terms of the patient's own cholesterol and blood pressure levels and in terms of 'the things I do' (for example, exercise or diet) and 'the type of person I am' [for example, 'stressy' (012) or 'active' (010)]. Patients viewed this information about relatives as relevant to their own risk, so weighing up both their overall risk and the risk from the family history itself.

Clinicians responded to patients' accounts of family cardiac events with sympathy. However, patients' explanations about relatives' heart disease were less easily accommodated. For example, clinicians focused on relatives' age of disease onset and the number of affected relatives, but did not explore information about relatives' lifestyle, blood pressure or cholesterol:

> Patient: They all died suddenly … that was a while ago and their diet and lifestyle was different to

what I have, they were smokers, I've never smoked.

> Clinician: Right. And had they had heart disease before that?

> Patient: Yeah, well, they said that my father's blood pressure was high, but it'd never been treated … they put my mother's [hypertension] down to a nervous disposition and hadn't realised her blood pressure was high until very late ….

> Clinician: Because neither of them had heart disease below the age of 65 for your mum and 55 for your dad you come out as being of average risk.

> Patient: Yeah, and like I say, their lifestyle … they were all smokers and my father loved fried bacon and fried everything, which is totally what I don't do. (Consultation, patient 015: Average family history risk, 10–20% absolute risk)

Family history was typically incorporated as a fixed numerical adjustment to the risk score by clinicians and then placed aside as 'non-modifiable'. Modifiable risk factors, such as diet and exercise, were used to adjust the absolute risk score and suggest a course of action. During this process, patient's explanations of relatives' heart disease were either not explored or reinterpreted in terms of the patient's own modifiable risk factors such as diet, exercise or stress:

> Patient: My family is quite a nervy bunch and stress was a big component part I think to my father's early demise and I don't know how to reduce that because I just naturally find myself in work that you know encourages that … I think that that can actually do a lot of damage to your heart. But I don't know how you get around that ….

> Clinician: I've got a few sort of non-evidence based things and thoughts on that and I suppose … you can't live an essentially stress-free life … but I reckon if you can get out and do exercise … (Consultation, patient 003: Strong family history, 10–20% absolute risk)

Patients were generally content to have their overall risk score adjusted according to their lifestyle. Where there was little to modify, however, overlooking the patient's explanation of their relatives' heart disease contributed to strained communication.

Not exploring patient's explanations about their relatives' heart disease reflected clinicians' orientation towards modifiable risk factors and revealed uncertainty about the clinical relevance of this information. When patients did not volunteer information about relatives' heart disease, clinicians did not raise the issue, and when patients asked questions, clinicians provided few answers.

In the subsequent interviews, patients continued to make sense of their risk partly through explaining relatives' heart disease. They weighed up which risk factors were 'inherited' and reflected on how 'in control' of their family history they were. The inherited risk was not described as simply genetic; multifarious explanations of the role of genes and environment in causing relatives' heart disease were expressed:

[Completing the FHQ] I suddenly thought, gosh there's an awful lot of heart disease. My family were overweight and, you know, there was an awful lot of sugar and fat involved in the cooking at that time … it's so easy to go down the generations and not be aware of what's behind you …. It's just a case of, I really want to try and stop the disease progressing because, I think you have enough problems in life without having the family history that's possibly your genes going to come down, and I was hoping would get weaker and weaker as the generations go on … (Interview, patient 001: Strong family history, 10–20% absolute risk)

Making sense of relatives' heart disease was occasionally difficult for patients. Two patients whose family history was 'average' still regarded their family history as significant. The lack of clarity received from clinicians about why their relatives had developed heart disease meant uncertainties about their own or other family members' risk persisted:

[My Dad and] his brother had the same thing [heart attacks and 'nervous problems'] … and they both died in exactly the same way …. I thought, is it hereditary, then? … or was it just something that happened to them, a co-incidence? Was it something that happened to them because they had the same upbringing? …. And my son's had, I suppose, a similar thing to my dad, panic attacks …. And I just wonder … if that is going to be hereditary … whether it's been passed to him, rather than me. (Interview, patient 012: Average family history, 0–10% absolute risk)

As the above quote shows, this patient held an ambiguous notion of 'heredity' whereby traits such as 'nervousness' or heart disease might have been passed from her father to her son but 'skipped' her. Her uncertainty about risk persisted after the consultation partly because this causal model was not addressed by the clinician.

Interaction of risk factors
In the consultation, patients related risk factors to each other and sought to understand their own cholesterol and blood pressure results. Concerns about these results were often because of family history. However,

clinicians rarely related family history to other risk factors.

In the consultations, clinicians sometimes stated that cholesterol and blood pressure had 'a genetic component' (016) and often explained that cholesterol is only partly modified by diet. However, this genetic component was not linked to family history. On one occasion, the clinician attributed the patient's high cholesterol to chance rather than family history:

Nurse: So you know now that [your cholesterol is] 8.3?

Patient: Yeah … I bumped into a friend of mine last night and he's a big lad all right … his cholesterol is 2.6!

Clinician: What does he do differently to you then? Exercise do you think?

Patient: No. He barely exercises, I would say his diet is worse than mine … he tends to have fried food which I don't ….

Clinician: Life's not fair then is it? (Consultation, patient 019: Average family history, but family history of high cholesterol, 10–20% absolute risk)

Opportunities to discuss the relationship between patients' family history and their current risk factors were partly obscured by the tendency to address risk factors individually. For instance, smoking was discussed in isolation without it being related to the presence of a family history:

Clinician: So, I suppose what we are saying is, you have got a family history which might contribute to some risk over the next 10 years. Blood pressure and weight's alright, cholesterol's alright, so the only thing that you can really alter out of that is the old cigarettes … you quite enjoy them I guess don't you?

Patient: Yeah, with the amount of driving and the road conditions it's either fags or valium. (Consultation, patient 017: Strong family history, 10–20% absolute risk)

In the interview, it was apparent that not relating his strong family history to smoking enabled this patient to maintain a view of heart disease as down to chance:

[My] old man packed up work because he had heart problems and me mum, she's always been a heavy smoker. Me oldest sister … she started having heart problems and that's what surprised me because she's a non-smoker and, like I said to the doctor, she's the fit healthy one … if it had been me, you know, I would have understood it, but not her. And it's the smokers logic that if she's

got it and she's never sparked up, and I'm perfectly OK ... I know smoking's bad for you, but, 100% of non-smokers are going to die ... I haven't got an inkling to give up smoking. (Interview, patient 017: Strong family history, 10–20% absolute risk)

A frequent comment in the interviews was that consultations did not explain the relation of family history to other risk factors. Patients drew their own conclusions. One way involved using family history to explain blood pressure or cholesterol results. Frustration and moral indignation were expressed by patients who were 'doing all the right things' but whose blood pressure or cholesterol remained high; some concluded that it must be due to 'something in the family history'. When patients saw themselves as healthy, family history was regarded more as a genetic than environmental influence. This did not, however, engender a fatalistic attitude, patients still strived to reduce their cholesterol or blood pressure:

[I'm] leading a fairly healthy lifestyle, how else would you explain high cholesterol levels? Other than it being genetic .... So I guess I came away with knowing where I am today ... it would be useful to perhaps try a particular diet for a specific period to see the impact on the cholesterol result. You know, as opposed to saying 'oh, it's genetic, that's life.' It would be nice to feel that you had an input over it, something preventative .... (Interview, patient 018: Moderately high family history, 0–10% absolute risk)

Patients also conversely interpreted their blood pressure or cholesterol results as 'markers' of whether the family history was having an impact. Patients with a positive family history but normal cholesterol and blood pressure readings concluded that their family history was probably not a problem in itself, as it had not manifested in high cholesterol or hypertension. However, a nagging sense of 'what's really going on in my body' persisted for some patients who were concerned that there was some damage to their heart or arteries undetected by cholesterol or blood pressure tests:

If I had high cholesterol problems or high blood pressure and if I had weight problems and a sedentary lifestyle then this would scare me, but I don't ... I think he was saying ... carry on as you are ... I just feel possibly there were some other tests that would be quite interesting to undertake, you know have a heart monitor and some stress, you know, body exercises just to really evaluate the true state of one's heart. (Interview, patient 003: Strong family history, 10–20% absolute risk)

Understanding the overall risk

Reaching general agreement with the clinician involved integrating all the results with a message about the 'overall risk' and lifestyle and medication options. In some cases, the way family history was related to the overall risk clouded this agreement and produced apparently incongruous messages about the overall level of risk and how to reduce it.

Family history was not explained explicitly by clinicians; however, two models of its relation to the overall risk emerged conveying different messages about the nature and relative importance of family history. In one model, family history was described as the 'genetic' element that 'you can't change'. This produced a message that family history constituted a fixed risk and that overall risk could only be reduced by focusing on other modifiable elements. When there was little to modify, this could be contradictory and the message that family history is a separate genetic factor overstated:

Patient: Would you say it's quite a high risk? Considering everything else?

Clinician: [Your risk] is not because of how you are now ... it's what's in your genes that's changing your risk.

Patient: So, even though everything else is low, I could still walk out of here and have a heart attack?

Clinician: Yes. (013: Moderately high family history, 0–10% absolute risk)

In the second model, family history was treated as part of the overall risk but not explained as a risk in itself. The implicit message was that family history itself was not a problem, but that changing the other risk factors would reduce the whole risk. This message made sense when blood pressure and cholesterol results were normal or there was something specific to modify. When this was not so, the scope for the clinician's advice was more restricted. The following patient had high cholesterol and a strong family history but led a healthy lifestyle:

Patient: I would quite like to just see what I can do with diet ... after six months if it has made any difference.

Clinician: That sounds reasonable ... if you were fairly tight with the diet and the exercise and then do the cholesterol test again ....

Patient: Yes but I don't know ... is there anything else I can do?

Clinician: I think, your weight is right .... You have plenty of exercise, you don't smoke ... and eating healthily so you're doing all the right things

and it may well come down because your choles-
terol was lower than this in the past. Again there
is a limit to how much you can lower your choles-
terol with diet. (Consultation, patient 006: Strong
family history, 0–10% absolute risk)

These apparent incongruities produced uncertainty
about the relative importance of family history and
how to reduce risk. In the interviews, a main theme
was 'prevention is better than treatment'. Patients
tried to conclude either 'there's something I can do
about the risk' or 'I've minimized the risk already'.
The two patients above particularly struggled to draw
either conclusion:

Well, you know, how much more can you do, eat
healthily and she said try not to get stressed well,
that's impossible really … but you know I don't
know what else I could do to prevent it, to make
it like a hundred percent chance that nothing was
going to happen. (Interview, patient 013: Moder-
ately high family history, 0–10% absolute risk)

In less extreme cases, patients still expressed ambi-
guity about reducing their risk. This ambiguity partly
reflected clinicians' treatment of family history; the
family history risk was identified but its relative signif-
icance unexplained. Patients had also expected the
risk assessment to provide more personalized and
clear-cut risk information. Patients frequently com-
mented in interviews that heart disease is unpredict-
able. Family history assessment had been anticipated
to diminish this unpredictability:

There wasn't much of an explanation as to how
[family history] did fit in. Whether there's a predis-
position or whether it was lifestyle …. My father's
brother is five years older than my father, had an
incredibly stressful life, smokes like a chimney,
drinks like a fish but is still walking around ….
(Interview, patient 010: Moderately high family
history, 0–10% absolute risk)

Discussion

Summary of findings
This study found that patients value systematic assess-
ment of CHD family history. When patients and clini-
cians could focus on modifiable risks, patients were
satisfied with the assessment of family history. How-
ever, three issues were identified in the consultations
which contributed to concerns and uncertainty for pa-
tients: patients' explanations of their family history
were not explored, the relationship between family
history and other risk factors was not discussed and
family history was not explicitly explained in relation
to the overall risk. In the study, these three issues were

important for effective communication, but also pose
clinical implications.

Comparison with existing literature
It is widely thought that effective clinician–patient
communication depends in part upon an appreciation
of patients' lay models of disease.[15] Lay people hold
complicated understandings about inheritance of heart
disease; it can be 'passed down' alongside other physi-
cal traits or mediated through inheritance of personal-
ity or constitution.[10] Recent work demonstrated that
lay beliefs and experiences influence individuals' rec-
ognition of their own family history and suggested that
misunderstandings could arise due to differences in
medical and lay understandings.[8,9,11] Patients in our
study expressed similar ideas about family history of
heart disease to those in previous research.

The three issues we identified in this study can be
partially explained as resulting from divergent patient
and clinical models of family history. In the consulta-
tions, clinicians attributed the risk associated with
family history to 'genes', or treated it as a numerical
figure, whereas patients held more complicated mod-
els. More fundamentally, however, patients and clini-
cians were 'doing' different things in the consultation.

Not exploring patients' explanations, and not ex-
plaining explicitly what family history is, may in part
be explained by clinicians' lack of knowledge about
gene–environment interaction; GPs hold limited
knowledge about genetics.[16] However, what clinicians
in our study did with family history, and how they
talked about it, was also contingent on the clinical
context. In line with current guidelines,[5] clinicians
used an additive model, treated the family history risk
as independent, and followed a model of primary pre-
vention which focuses on immediate, modifiable risk
factors. Previous research has shown that clinicians of-
ten ignore patients' explanations of illness as clinically
irrelevant information.[17] Given the primary preven-
tion orientation, explanations of relatives' heart dis-
ease may appear clinically irrelevant to clinicians.
Moreover, the use of an additive model, and the focus
on the modifiable elements of risk, also meant there
was little available rhetoric to discuss with patients
how genes and environment interact.

Patients attempted to understand their 'whole risk'
by relating what had been passed down to them to
what was currently occurring in their bodies. Thus,
they tried to answer the question 'is it hereditary and
have I inherited it?'. Causal information about rela-
tives was, to the patient, relevant. People often try to
make sense of complex risk information by under-
standing disease causality; the past is relevant to un-
derstanding the future.[18] Furthermore, the idea of risk
is generally more subjective for the patient than it is
for clinicians.[19] Patients in our study also expected the
risk assessment to be more personalized than it was.

Avoiding misunderstandings depended more on patients and clinicians sharing an understanding of the preventability of heart disease than it did on shared understandings of family history. When there was little to modify, patients observed the anomalies between their own lifestyle and their clinical results without always understanding them. Discussion of the interaction between environmental and genetic factors could have assisted in understanding such anomalies.

Strengths and limitations of study

This study is the first to look at communication in primary care consultations using a validated CHD FHQ. This is timely given the current emphasis on assessing family history of CHD and other common chronic diseases.[4] Patients recruited to the study came from a range of social classes. All patients recruited were white; further research among different ethnic groups is needed. All but one patient in this study fell below the current 20% 10-year CVD risk statin-prescribing threshold. Although the main findings and recommendations should aid consultations across risk categories, further research among patients at higher risk might be required. In normal practice, a FHQ is not utilized, but a method of systematically collecting family history could enhance CVD risk assessment and, based on our findings, be positively received by patients. Current guidelines recommend GPs identify patients with premature CHD in first-degree relatives.[5] In the future, more complex family histories may need to be interpreted requiring electronic decision support similar to that for familial cancer risk identification.[20]

Implications for clinical practice

Family history assessment is anticipated to produce a more personalized approach to CHD risk management.[21] It could also diminish the chance of people disregarding simplistic public health messages because they observe 'exceptions' (individuals who have heart attacks in spite of their healthy lifestyle and vice versa).[22–24] Treating family history as a genetic, independent risk factor, or leaving it unexplained, misses this potential. This study highlights the need for clinicians to explore patients' explanations of their family history and to discuss the relationship between risk factors.

Family history is a proxy for complex gene–environment interactions and the recommended 1.5 times increase in absolute risk[5] is based on population-level epidemiological data. Information about patient's relatives may be of clinical relevance to their individual risk and could facilitate targeted health promotion.[25] For example, telling a smoker that family history and smoking together have a multiplicative effect could personalize smoking cessation advice. There is a need for clearer guidance about family history of CHD. Key points to address are that family history

assessment captures both genetic and environmental factors aggregating in families and the relationship between family history and other risk factors and its relative importance to overall risk.

Declaration

References

1 Hawe E, Talmud PJ, Miller GJ, Humphries SE. Family history is a coronary heart disease risk factor in the Second Northwick Park Heart Study. *Ann Hum Genet* 2003; **67:** 1–10.

2 Kardia SLR, Modell SM, Peyser PA. Family-centered approaches to understanding and preventing coronary heart disease. *Am J Prev Med* 2003; **24** (2): 143–151.

3 Yoon P, Scheuner MT, Peterson-Oehlke MS, Gwinn M, Faucett A. Can family history be used as a tool for public health and preventive medicine? *Genet Med* 2002; **4:** 304–310.

4 Department of Health. *National Service Framework for Coronary Heart Disease*. London, 2000.

5 British Cardiac Society, British Hyperlipidaemia Association, British Hypertension Society, British Diabetic Association. JBS 2: Joint British Societies' guidelines on prevention of cardiovascular disease in clinical practice. *Heart* 2005; **91(suppl 5):** v1–v52.

6 Sheridan S, Pignone M, Mulrow C. Framingham-based tools to calculate the global risk of coronary heart disease: a systematic review of tools for clinicians. *J Gen Intern Med* 2003; **18:** 1039–1052.

7 Jousilahti P, Puska P, Vartiainen E, Pekkanen J, Tuomilehto J. Parental history of premature coronary heart disease: an independent risk factor of myocardial infarction. *J Clin Epidemiol* 1996; **49:** 497–503.

8 Walter FM, Emery J. 'Coming down the line'—patients' understanding of their family history of common chronic disease. *Ann Fam Med* 2005; **3:** 405–414.

9 Hunt K, Emslie C, Watt G. Lay constructions of a family history of heart disease: potential for misunderstandings in the clinical encounter? *Lancet* 2001; **375:** 1168–1171.

10 Davison C, Frankel S, Smith GD. Inheriting heart trouble: the relevance of common-sense ideas to preventive measures. *Health Educ Res* 1989; **4:** 329–340.

11 Walter FM, Emery J. Perceptions of family history across common diseases: a qualitative study in primary care. *Fam Pract* 2006; **23:** 472–480.

12 Qureshi N, Bethea J, Modell B *et al*. Collecting genetic information in primary care: evaluating a new family history tool. *Fam Pract* 2005; **22:** 663–669.

13 Scheuner MT, Wang S-J, Larabell SK, Rotter JI. Family history: a comprehensive genetic risk assessment method for chronic conditions of adulthood. *Am J Med Genet* 1997; **71:** 315–324.

14 Glaser B. The constant comparative method of qualitative analysis. *Soc Probl* 1965; **12:** 436–445.

[15] Helman CG. Communication in primary care: the role of patient and practitioner explanatory models. *Soc Sci Med* 1985; **20:** 923–931.

[16] Kumar S, Gantley M. Tensions between policy makers and general practitioners in implementing new genetics: grounded theory interview study. *BMJ* 1999; **319:** 1410–1413.

[17] Gill VT. Doing attributions in medical interaction: patients' explanations for illness and doctors' responses. *Soc Psychol Q* 1998; **61:** 342–360.

[18] Cohn S. Risk, ambiguity and the loss of control: how people with a chronic illness experience complex biomedical causal models. In Caplan P (ed). *Risk Revisited*. London: Pluto Press, 2000: 204–225.

[19] Gifford S. The meaning of lumps: a case study of the ambiguities of risk. In Janes CR, Stall R, Gifford S (eds). *Anthropology and Epidemiology: Interdisciplinary Approaches to the Study of Health and Diseases*, Boston, MA: Kluwer Academic Publishers, 1986: 213–246.

[20] Emery J, Walton R, Coulson A, Glasspool D, Ziebland S, Fox J. Computer support for recording and interpreting family histories of breast and ovarian cancer in primary care (RAGS): qualitative evaluation with simulated patients. *BMJ* 1999; **319:** 32–36.

[21] Williams RR, Hunt SC, Heiss G *et al.* Usefulness of cardiovascular family history data for population-based preventive medicine and medical research (the Health Family Tree Study and the NHLBI Family Heart Study). *Am J Cardiol* 2001; **87** (2): 129–135.

[22] Davison C, Davey Smith G, Frankel S. Lay epidemiology and the prevention paradox: the implications of coronary candidacy for health education. *Sociol Health Illn* 1991; **13** (1): 1–19.

[23] Hunt K, Emslie C. Commentary: the prevention paradox in lay epidemiology—Rose revisited. *Int J Epidemiol* 2001; **30:** 442–446.

[24] McConnachie A, Hunt K, Emslie C, Hart C, Watt G. "Unwarranted survivals" and "anomalous deaths" from coronary heart disease: prospective survey of general population. *BMJ* 2001; **323:** 1487–1491.

[25] Rogers JC, Rohrbaugh M. The SAGE-PAGE trial: do family genograms make a difference? *J Am Board Fam Med* 1991; **4:** 319–326.