

Original Article

Holding blame at bay? 'Gene talk' in family members' accounts of schizophrenia aetiology

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Abstract We provide the first detailed analysis of how, for what purposes and with what consequences people related to someone with a diagnosis of schizophrenia use 'gene talk'. The article analyses findings from a qualitative interview study conducted in London and involving 19 participants (mostly women). We transcribed the interviews verbatim and analysed them using grounded theory methods. We analyse how and for what purposes participants mobilized 'gene talk' in their affectively freighted encounter with an unknown interviewer. Gene talk served to (re)position blame and guilt, and was simultaneously used imaginatively to forge family history narratives. Family members used 'gene talk' to recruit forebears with no psychiatric diagnosis into a family history of mental illness, and presented the origins of the diagnosed family member's schizophrenia as lying temporally before, and hence beyond the agency of the immediate family. Gene talk was also used in attempts to dislodge the distressing figure of the schizophrenia-inducing mother. 'Gene talk', however, ultimately displaced, rather than resolved, the (self-)blame of many family members, particularly mothers. Our article challenges the commonly expressed view that genetic accounts will absolve family members' sense of (self-)blame in relation to their relative's/relatives' diagnosis.

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Introduction

Psychiatric genetics has grown in prominence over the last two decades. As the field has grown, social scientists and clinicians have wondered whether the models proposed by psychiatric genetics are affecting how diagnosed individuals and family members conceptualize the aetiology of mental illness (for example, Barr and Rose, 2008). There has been particular interest in the possibility that an emphasis on biogenetic factors might reduce the stigma of mental illness, as well as the blame and guilt frequently experienced both by those who receive psychiatric diagnoses and those related to them (Austin and Honer, 2007; Rüsçh *et al*, 2010). Although there are growing indications that biogenetic accounts do not reduce stigma at a population level (Read *et al*, 2006; Angermeyer *et al*, 2011), it is likely that such accounts carry different implications for different groups of people. Some qualitative studies have shown that some people either with a psychiatric diagnosis or related to someone with a psychiatric diagnosis believe that biogenetic accounts reduce – or *might* reduce – stigma and blame, through shifting responsibility and/or culpability away from the individual and towards heredity (Meiser *et al*, 2005; Laegsgaard *et al*, 2010; Potokar *et al*, 2011). In relation to family members, the stigma researcher Jo Phelan has surmised that the use of genetic accounts ‘may be most beneficial to parents and most harmful to younger relatives, such as siblings, children, and cousins of the ill person’. Such accounts ‘may absolve parents of causal responsibility’ but make the issue of ‘genetic contamination’ particularly salient for younger relations who might well be searching for partners and/or in the ‘risk period’ for developing the illness themselves (Phelan, 2005).

There has, nonetheless, been surprisingly little in-depth, qualitative research that has investigated if and how ‘gene talk’ is employed by people related to someone with a psychiatric diagnosis. We contribute to what we hope will become a larger, empirically grounded literature by providing a fine-grained analysis of a subset of data deriving from a qualitative interview study conducted in London. The article explores how individuals related to at least one person with a diagnosis of schizophrenia employ ‘gene talk’ when accounting for the presence of schizophrenia within their families. That the diagnosis of schizophrenia is one of the most stigmatized of psychiatric diagnoses (Knight *et al*, 2003; Crisp *et al*, 2005) is likely to bring into sharp relief any ‘beneficial’ or ‘harmful’ consequences (to use Phelan’s terminology) that may arise from family members’ use of genetic accounts.

We borrow the phrase ‘gene talk’ from Evelyn Fox Keller who, in investigating how ‘gene talk’ influenced biological research over the last century, emphasized the importance of understanding what gene talk *was for*. (She pointed to its effectiveness as a tool of persuasion as well as an operational shorthand for scientists working in particular experimental contexts (Fox Keller, 2000).) We focus on exploring the moments at which family members mobilize ‘gene talk’, in order to investigate what the attractions of such talk might be for those wanting to discuss the presence of schizophrenia within their family, as well as if and how such talk addresses or negotiates questions of blame and guilt. In other words, rather than assuming that family members’ use of the word ‘gene’, ‘genes’ or ‘genetic’ straightforwardly installs their adherence to a genetic model of mental illness, we investigate the logic and characteristics of ‘gene talk’, in order to understand the ‘symbolic and social functions’ (Duden and Samerski, 2008) carried out through such talk. It is worth emphasizing that we take seriously the configuration of the intersubjective space in which such gene



talk unfolded in our study: participants were discussing, face to face, the presence of schizophrenia within their family with a researcher whom they did not previously know.

Our article draws on findings from a qualitative interview study, which involved 37 research participants (19 family members and 18 service users) and was conducted between 2008 and 2010. The primary objective of the study was to investigate participants' views regarding participation in genetic research on schizophrenia. In this article, we focus solely on the family members, in both the methods and the analytical sections, so as to be able to pursue in greater detail lines of theoretical inquiry concerning their use of gene talk.

Methods

Family members (who were not related to one another) were recruited using e-mail lists, local posters and visits to family/carer support groups. Some purposive sampling was used to ensure that we captured the perspectives of family members who were not university educated. Family members were related (first or second degree) to at least one person who had received a diagnosis of schizophrenia; they could not have received a diagnosis of schizophrenia or bipolar disorder themselves, and were 18 years or older; all interviews were conducted in English. The participants were predominantly female, ranged in age from mid-20s to early 60s, included individuals from a range of countries and ethnicities, and were relatively diverse in their levels of education. While the majority were siblings of someone with a diagnosis of schizophrenia, the sample also included parents, children, nieces, first cousins and one ex-spouse (the only person not related by consanguinity). (See Table 1 for demographic details.) We use familial terms – for example, 'mother', 'brother' – to denote the relation that the interviewed family member has to the person(s) with a schizophrenia diagnosis. Six participants had more than one relative with this diagnosis: it is likely that this was the result of self-selection, given the topic of the research study.

The study was approved by the local ethics committee. Following informed consent (for participation and use of data in publications), participants took part in semi-structured interviews lasting 30min to 2 hours (on average 45–60min), conducted by Felicity Callard and Jody Quigley (both social scientists). These researchers strenuously attempted to position themselves as having no stake in psychiatric research involving genetics, and conducted as many interviews as possible in participants' homes (and, if not, in a university setting). The interview addressed participants' experience of and feelings about involvement in mental health research; their thoughts regarding genetic research addressing schizophrenia; their reactions to a segment from a short excerpt from a television programme in which clinical researchers discussed genetic research on schizophrenia (which was presented towards the end of the interview); and their opinions on what influences people to be 'pro' or 'anti' genetic research.¹ The topic guide did not contain questions regarding schizophrenia aetiology. Each participant received £15 at the interview's close.

All interviews were recorded, transcribed verbatim and analysed using NVivo 8. Felicity Callard coded all interviews; Emma-Louise Hanif and Jody Quigley participated in the earlier

1 Through the coding process, it became clear that the 4-min television excerpt had very little impact on how participants used 'gene talk'. We therefore quote from material both before and subsequent to the presentation of the television excerpt.

Table 1: Demographics of participants ($n=19$)

| | |
|--|----|
| <i>Gender</i> | |
| Male | 2 |
| Female | 17 |
| <i>Age</i> | |
| ≤30 | 6 |
| ≤40 | 5 |
| ≤50 | 1 |
| ≤60 | 5 |
| Over 60 | 2 |
| <i>Ethnicity</i> | |
| White | 14 |
| Asian/Asian British | 2 |
| Black/Black British | 1 |
| Mixed heritage | 2 |
| <i>Education [highest level completed]</i> | |
| Up to 16 years | 4 |
| Up to 18 years | 2 |
| Undergraduate degree | 6 |
| Post-graduate degree | 7 |
| Relationship to family member with diagnosis of schizophrenia | |
| <i>The total exceeds the number of participants, since some participants had more than one family member with a diagnosis.</i> | |
| Mother | 5 |
| Father | 1 |
| Ex-wife | 1 |
| Sister | 10 |
| Brother | 1 |
| Daughter | 3 |
| Niece | 2 |
| First cousin | 2 |

phases of coding to cross-reference and refine codes. The analysis presented here was indebted to many of the principles and methods of grounded theory (including initial line-by-line coding (Glaser, 1978) and iterative memo writing), though departed from grounded theory by virtue of our prior formulation of certain overall objectives that shaped how the study was designed and carried out (see Charmaz, 2006). In the course of memo writing and refinement of codes, it became clear that participants, frequently spontaneously, introduced discussions of aetiology. We identified all passages within the transcripts in which interviewees used language relating to genes when expressing some formulation that explained the presence of schizophrenia and/or ‘mental illness’ within their family, and/or the cause of schizophrenia. We shuttled between close, line-by-line coding and engagement with select social scientific literature to develop our analytical framework.

There is significant, often critical, debate about the ontology and historicity of psychiatric diagnostic categories (Lakoff, 2005; Martin, 2007), including schizophrenia (Boyle, 2002). We refer to people ‘who have received a diagnosis of schizophrenia’ so as to remain agnostic about the ontological status of schizophrenia. We followed our research participants in the



use of the term 'schizophrenia' during the interviews; while some family members expressed ambivalence about the concept of schizophrenia, most used the word to denote a discrete mental illness that their relative(s) had. In order to protect participants' anonymity, all names are changed and some narratives are composites.

Our analysis of findings is divided into two sections. First, we consider how family members use 'gene talk' in attempts to exculpate the immediate family of the diagnosed person from blame. Second, we explore the fragility of 'gene talk' in securely holding such blame at bay. While narratives were inflected by participants' gender, ethnicity, class and age, participants were largely united in their use of 'gene talk' to navigate blame; we therefore do not explicitly consider these factors – asides from our focus on the symbolic position of the mother – in this article.

We wish to emphasize, before moving to the specifics of our analysis, how affectively complex the 'gene talk' of our participants was. It is still somewhat uncommon for the emotional timbre of research encounters to be explicitly noted in academic writing, let alone a focus of analytical concern (though see Devereux, 1967; Davies and Spencer, 2010). It was clear during the time of our interviews that many of them were affectively intense experiences for many of the participants (as variously demonstrated through tears, through steely determination not to become 'emotional', and through clear manifestations of anxiety) – and indeed for us, too, as researchers. It nonetheless took a while for us to begin to understand and be able to interpret the diverse emotional valences of participants' 'gene talk'. Guilt and (self-)blame produce complex circuits of feelings. We argue that each family participant was, during the research interview, in an intersubjective relation not only with the interviewer (in relation to whom s/he might have manifested feelings of guilt and (self-)blame at that particular moment), but with (imaginary) others (whom s/he might have regarded as part of other circuits for any such feelings), and, indeed, with him or herself (since both guilt and blame can loop around and through the self). For example, it was not unusual for the interviewer to feel that there were (imagined) others in the room alongside the family member (often the relative with the schizophrenia diagnosis, or other family members) who were in some ways the addressees, alongside the interviewer herself, of the interviewees' speech. 'Gene talk' undoubtedly provided a flexible and fertile resource through which these intersubjective relations were shaped and negotiated. At times, as we demonstrate below, participants used 'gene talk' consciously and forcefully in attempts to repulse blame and guilt that might otherwise stick to themselves or to their family members (see Ahmed, 2004, for a powerful analysis of how certain emotions tend to 'stick' to particular subjects); at other times, 'gene talk' traced what we came to interpret as less conscious, and manifestly painful efforts to negotiate difficult family histories. In some participants' interviews, 'gene talk' was prodigious and affectively intense; for some participants, gene talk was prodigious and (at least on the surface) affectively neutral; and in a small minority of cases, gene talk was markedly rare.² Although we are unable in this article to give the diversity of these affective valences the analytic attention that it deserves, we endeavour

2 A forthcoming article uses exemplary moments from our interviews in order to place the affective transfers between researcher and research participant centre stage of the analysis. Both this forthcoming article and our analysis of 'gene talk' here start from the premise that it is unwise to assume that one knows with any degree of certainty that what transpires in the course of an interview (and, indeed, sometimes well after its completion) necessarily focuses on those areas that one set up the interview to address.

when providing excerpts from our interviews, below, at least to gesture to those excerpts' affective tenor. In so doing, we hope both to clarify the complex 'social and symbolic' functions that we identify in gene talk, as well as to substantiate our argument that such talk is ultimately fragile in its capacity to hold blame at bay.

'Gene Talk' and Schizophrenia Aetiology

Not one family member disputed the role of genetics in the aetiology of schizophrenia. This does not mean that each endorsed a solely genetic model of identity and aetiology: rather, family members overwhelmingly took pains to emphasize that genes interacted with other factors. This has important consequences for how 'gene talk' served to address questions of blame (see section 'Holding blame at bay?'). Many used a language of combination and/or of triggering through which to link genes (frequently used loosely to connote 'heredity' or 'disposition') to the influence of other factors:

Gemma (*daughter and sister*) spoke of: '*a combination of predisposition with family dynamics, plus current affairs going on [...] the twentieth century was a funny time and a bit of that coming in, like some real catastrophes happening in the background which were quite terrifying you know, [and] a bit of marijuana in there too*'.

Jane (*daughter and sister*) invoked the language of current molecular genetics: '*environment and genetics ... a particular gene may switch [schizophrenia] on [...], because of some sort of environmental factors*'.

Karen (*mother*) spoke of something that '*perhaps would trigger the onset of something like schizophrenia*' – invoking, in the case of her own child, the '*make up of the person*' and '*heavy cannabis [use]*'.

Family members' talk of predisposition, combination and/or triggering bore similarities to scientists' 'diathesis-stress' models of schizophrenia (Rosenthal, 1970), and has been found in other studies investigating family members' and service users' explanatory models for psychosis (for example, Meiser *et al*, 2005). Our interviews were designed, as we have noted, to investigate views regarding participation in genetic research, and we therefore did not press family members to provide reasons for why they were committed to these combinatorial models – rather than, say, a more strictly 'genetic' account of schizophrenia. It is possible to conjecture, however, that participants desired to tell a cogent life narrative that could in some way account for the diagnosis of their family member(s), and that such a narrative would, perhaps, be harder to shape if it employed a solely 'genetic' formulation. The frequency with which participants referred to critical events during the adolescence or early adulthood of the diagnosed relative(s) (including drugs, parental divorce, the death of relatives or friends), for example, suggests that it may well have been difficult for family members to forgo the salience of this period of life when accounting for the formation of an individual's subjectivity. A combinatorial model provides one way in which to draw these salient events (the '*heavy cannabis [use]*' that Karen mentions, or the '*real catastrophes happening in the background*'



referred to by Gemma) into articulation with genes or predisposition. In the two sub-sections that follow, we clarify how genes were invoked within these combinatorial models.

Telling a family history of schizophrenia: Recruiting antecedents

'Genes' and/or 'genetics' were most commonly mentioned in attempts to provide coherence for the story of a family beset by mental illness. There was a strong impulse from most family members to tell, spontaneously and often with great conviction, the interviewer this history. The history always extended at least one generation back from the person narrating it. For interviewees related to more than one person with a diagnosis of schizophrenia, it was easy to enroll these family members into the history:

Vivienne (*mother and sister*), when asked why she was interested in participating in the interview study, replied, with sorrow and a degree of urgency: 'Because it's genetic and I want to know what lies behind the genetic because I had three generations of mental illness and I know that it is in the family'.

But what was particularly striking was how family members whose families contained *no* other known or clear examples of mental illness (for example, psychiatric diagnoses, psychiatric admissions, suicides) used 'gene talk'. While a small number of participants explicitly commented that there was 'no family history' beyond the family member they were speaking about, much more common was the urge to use 'gene talk' to enumerate *potential* instances of schizophrenia (and/or other related mental health problems).

Karen (*mother*): there must be a genetic link [in schizophrenia] because [...] I've got no real proof [...], on my ex's family, [...] they had aunts who [...] had [...] lived on their own for donkeys' years. And you think, well maybe, I know that my grandfather on my mother's side used to drink a lot and I don't know if that was because, if there was a reason for that. So maybe looking back [...] I think, [...] maybe there is something that comes through in the genes.

Giulia (*niece*): [schizophrenia] should be like kind of erm transmitted, genetic transmitted, like disease. But I just not sure because [...] in my family [my uncle's] the only one who has [schizophrenia] or maybe he's the only one who like, kind of like, how can I say? More clear symptoms (laughs)? Maybe you have and you don't know because you don't have like so exacerbated?

John (*father*) noted that while on his side of the family 'there's no history of mental illness', and on his wife's side 'no diagnosed [...] illness', he and his wife now believe that her mother had schizophrenia: 'She was a very difficult person [...] a very unusual person and [...] she would behave in a way which was quite extreme, ... which now seems like it was probably schizophrenia'.

Jane (*daughter and sister*) saw herself as the one in her family who could identify cases of schizophrenia. While there was 'still a lot of debate in [Jane's] family as to, "No! They don't suffer from schizophrenia",' in Jane's mind, 'They're exhibiting the very same symptoms of- that- to- that to me say that it is schizophrenia'. From this, she

concludes that, 'It's not surprising' that 'there could be some sort of mutation of gene that does get passed on through family members'.

Each of these family members is familiar with the scientific language of psychiatry and each uses, in the interview, formulations indebted to current psychiatric genetics (for example, mentions of gene mutations, neuroscience, environmental 'switches'). Each is committed to installing some role for genetics in relation to schizophrenia (note Karen's 'must be' and Giulia's 'should be', regarding the question of genetic transmission), yet none has an explicit example of another diagnosed person whom s/he can provide as evidence of this genetic 'link' or 'transmission'. This leads each of them to conjecture that other people in the family *might well have had or have* psychosis or psychosis-like symptoms – whether it be individuals with less clear symptoms (Giulia), or 'the very same symptoms' (Jane), or aunts who had 'lived on their own for donkeys' years' (Karen). They foreground their own expertise with psychiatric symptoms, diagnosis and models to give credence to their analysis of the presence of mental illness within their or their spouse's family history.

Other studies have shown how people use 'genes' to gesture to a (frequently vaguely understood) process of inheritance or transmission that links the illness of one family member to other familial instances (for example, Lock *et al*, 2006; Everett, 2011). The illnesses in these studies (for example, heart disease, cancer, arthritis) have tended to be less nosologically unstable than many psychiatric diagnoses (for example, Lindenmeyer *et al*, 2008). In contrast, the participants in our study construe socially unusual or unwelcome behaviour as either contiguous with or interpretable as a symptom of mental illness – such that this behaviour can be interpreted as indicative of undiagnosed familial schizophrenia/mental illness. Here, 'gene talk' gives shape to a subterranean family history, one that draws these members of the family into alignment with the diagnosed family member through an imagined story of shared mental illness.

This use of 'gene talk' serves, we argue, several functions. First, by enumerating likely forebears who are linked genetically to the diagnosed family member, it renders the presence of schizophrenia in the interviewee's immediate family more comprehensible and less shocking. (In this regard, it was common for family members to talk of resistance to and/or bewilderment at the arrival of schizophrenia within their family (see Jungbauer *et al*, 2004).

Second, it shifts the 'origins' of the schizophrenia to a temporal location before that of the immediate family. (Notably, there were no instances in which a parent interpreted his/her own – or his/her spouse's – odd behaviour as providing evidence of undiagnosed schizophrenia, so as to ground a genetic narrative about schizophrenia transmission. There was only one possible example of a sibling (Paolo) interpreting his parent's behaviour in such a way – and we discuss this sibling's narrative later in the article as exemplifying a clear departure from the predominant mode of telling a family history.) This temporal relocation of origins ensures that the cause of schizophrenia *does not lie within* the immediate family – a point we take up in the next section.

Third, 'gene talk' also contributes to the creation of particular kinship ties (Featherstone *et al*, 2006; Chilibeck *et al*, 2011). 'Genetic transmission' is like a key to a puzzle: with it, pieces of a family's history can be excavated and retold – in the form of unmasked tales of mental illness. In this regard, we disagree with the conclusions of Laegsgaard and colleagues in their study of the implications of psychiatric genetic knowledge among people with multiple



cases of depression in the family. They argue that participants 'recognized that depression had already been present several generations ago, and that behaviours by particular family members which had otherwise appeared inexplicable *were now put into their proper context*' [emphasis added] (Laegsgaard *et al*, 2010). The phrase 'proper context' implies that these families' understanding of their family history is now – appropriately and belatedly – catching up with robust scientific knowledge. Here, it is 'the proper context' of psychiatric genetics that provides the correct framework through which to decode the behaviours of various family members. We suggest the reverse. Participants mobilize their own understandings of psychiatric genetics, and, with this, piece together an interpretative frame through which they retrospectively 'diagnose' the behaviours and life decisions of forebears as indicative of the genetically transmitted illness of schizophrenia. In other words, they use their interpretations of current scientific genetic thinking *to create* their own kind of classificatory framework, rather than genetics comprising the *already created* 'proper context'.

Monica Konrad, in her research on predictive genetic testing for Huntington's Disease (a single gene disorder, in contradistinction to the polygenic models circulating in current biomedical schizophrenia research), has argued how 'the genetic tie can make or break relations' – for example, by both 'mak[ing] and re-invent[ing] the terms by which kin imagine that they can get on and get by with one another' (Konrad, 2005, p. 149). In this way, genetic knowledge has the capacity to reorganize a family's telling of its own history to itself and to others – through genealogies that are produced through scientific knowledge as well as (re)imagined kin relations. In our study, there was no *scientifically* substantiated 'genetic tie': no family participant had undertaken genetic testing for schizophrenia, and, in any case, the polygenic models of schizophrenia gainsay the possibility of any straightforward genetic test for schizophrenia. Rather, participants produced a variety of genetic genealogies that frequently built upon their invocations of supposed genetic links between certain family members. While these genealogies served, as they did in Konrad's research, to bring certain family members into closer proximity to one another, what was particularly noticeable was their reliance on certain well-worn cultural codes through which to identify and interpret individual family members' 'odd' behaviour (for example, the picking out of one family member's heavy drinking, or of another family member's life as an unmarried woman). It should, nonetheless, be emphasized that there are many historically and culturally variable ways in which a mother's 'extreme behaviour', a grandfather's extensive consumption of alcohol and an unmarried aunt's choice to live alone could be interpreted – ways that might, or might not, have some presumed relation with mental illness.³

Telling a family history of schizophrenia: The ghost of the schizophrenia-inducing mother

How might we account for the fact that so many family members – both parents and siblings – vigorously emphasized that the origins of schizophrenia were located before the temporal

3 The appearance of the figure of the single, unmarried woman is fascinating here, given the rich history of reading such women as existing outside of heteronormative relations (for example, as lesbians, or as celibate women) (Franzen, 1996). The potential overlap between an imagined genealogy that foregrounds individuals with presumed dissident sexualities and one that foregrounds individuals with presumed psychopathology demands further investigation.

span of their immediate, nuclear family (whether by enrolling family members with no diagnosis of mental illness, or by enumerating, unbidden, instances of mental illness within prior generations)? In order to make sense of this insistence, it is helpful to recall the twentieth-century history of the diagnosis of schizophrenia.

Various theories from the twentieth century positioned the immediate family – and in particular, the mother – as central to the aetiology of schizophrenia. (These theories include Fromm-Reichmann’s psychoanalytically derived model of the schizophrenogenic mother (Fromm-Reichmann, 1948); anthropologist Gregory Bateson’s double bind model (Bateson *et al*, 1956); Laing and Esterson’s antipsychiatry ‘scapegoat’ formulations (Laing and Esterson, 1970); and psychiatrist Lidz’s model of problematic familial communication (Lidz *et al*, 1965). The impact of such theories was wide-ranging and profound: they exerted their grip on significant swathes of the Anglo-American populace (see Hartwell, 1996; Staub, 2011), even though today they have received widespread scientific rejection. The notion of the ‘schizophrenogenic family’ – and particularly the ‘schizophrenogenic mother’ – casts, we suggest, a long shadow over today’s discussions of schizophrenia aetiology, in ways that are perhaps not sufficiently acknowledged (Ferriter and Huband, 2003). Even as people may not be aware of the *theory* of the schizophrenogenic mother, ‘mother blame’ in relation to children’s upbringing is culturally prevalent and deep-rooted. A rich tradition of feminist theory and history has interrogated complex ideologies of motherhood, which often cleave the ‘good’ mother from the ‘bad’, and which in so doing hold mothers as responsible not only for their own children’s future, but that of the future citizenry (for example, Rich, 1976; Blum, 2007; Plant, 2010). Ilna Singh has demonstrated, in relation to the ‘mother-blame’ that surrounds diagnoses of Attention Deficit Hyperactivity Disorder (ADHD), that the long history of mothers being implicated in their child’s psychopathology means that there is no easy way out from this culturally dominant narrative (Singh, 2004). In her research, mothers turned to a ‘brain-based’ model of ADHD; while this exonerated them from the blame of *causing* their sons’ behaviour, Singh shows how they were still captured within a logic of maternal responsibility and culpability by virtue of being responsible for *preventing* sons’ additional behavioural and psychological problems. There are analogies between mothers’ use of ‘brain-based’ models of ADHD and family members’ use of ‘gene talk’ when discussing the presence of schizophrenia in their family.

Feelings of blame (both self-blame and that from others) and guilt featured extensively in our interviews. While some participants explicitly used the words blame and guilt, we were also struck by what we interpreted as diverse affective and behavioural manifestations and traces of such feelings – whether through a participant’s use of wry laughter, or through her emphatic disclosure of how the diagnosed relative had in no way received less love as a child than his siblings. Traces of such feelings were particularly noticeable in interviews with parents (see Ferriter and Huband, 2003; Moses, 2010), but were certainly not confined to these. Notably, the mother was frequently mentioned on these occasions (for example, daughters stressing their mother’s ongoing feelings of guilt). At times, blame was raised in biogenetically informed discussions that exonerated the diagnosed relative:

Charlotte (*sister*) emphasized that her sister’s schizophrenia ‘is biological’ and that ‘it wasn’t her fault she developed it. She didn’t deliberately go out and live a massive drug-fuelled lifestyle, she was just unlucky’.



John likewise used a biogenetic account to install a divide between someone who is implicitly culpable (by taking substances that are seen to lead to schizophrenia) and someone – like his child – who was not at fault. When the interviewer asked him whether he found ‘genetic accounts’ more or less ‘comforting’ than other kinds of explanations, he responded:

We worried at first whether he was abusing substances, whether it's his lifestyle when he was younger when he seemed to go off the rails and therefore, erm, it's his own behaviour which is causing the mental health problems. But knowing that there's a possible fa- family link then means that it's not his fault, we can't, there's no blame we can lay on him for it. Erm, I think there's a lot of people who are involved in the blame game and I think, erm, we want to try and avoid that as much as possible.

The ‘possible family link’ allows putative blame to be removed from John's son's actions. This moment of the interview followed John's enumeration of the ‘behavioural problems’ of some of the members of prior generations, which he had interpreted as indicative of mental illness. In this way, ‘gene talk’ – here denoted by the phrase ‘family link’ – allowed John to interpret his son's behaviour not as the *generator* of his mental health problems, but rather as a kind of unwilling *replay* of the behavioural patterns of members of previous generations. Rather than schizophrenia being caused by the son's ‘own behaviour’, this behaviour is in some way *not exactly his own* – but rather a recapitulation of the behaviour of family forebears.

Although some family members hastened to remove blame and responsibility from the diagnosed individual(s), far more common were instances in which blame was shown to fester around the mother of the diagnosed individual(s). Sharon (a mother) wanted to clarify with the interviewer that cognitive remediation therapy for schizophrenia (a possible treatment option) was ‘not like regressing’ because:

I hate the idea that it's – which [has] been accepted for quite a long time, really, that it's always the parent's fault (laughs), that these things ... something horrendous, y'know, that's happened or something horrendous that you did, and I'm not saying horrendous things don't go on, [but] [...] it's pretty awful [...] when [...] you find yourself being questioned (laughs) [...] and fingerpointing, you know.

Sharon seems to imply that regression therapy further embeds the assumption that it is parents who have caused their child's illness (perhaps via the culturally prevalent and mistaken assumption that all psychological therapies focus on an individual's childhood and thereby tend to locate the ‘origin’ of the mental illness within the family). While she notes that ‘horrendous things’ do go on within families, she strenuously wishes to distance herself from the category of mothers who could be seen to have contributed ‘something horrendous’. Her anxious laughter betrays the intensity of her feelings at having been placed in this category by others.

We argue that it is only by grasping the cultural prevalence of this imagined category of parent – *and, in particular, this category of schizophrenia-inducing mother* – that it is possible to understand the emotional fuel that lies behind much of the emphatic use of ‘gene talk’. Mothers, oftentimes their spouses, and frequently their children wish to ensure that the ghost of the schizophrenia-inducing mother is not seen to have taken up

residence in their household. To substantiate this claim, we present two contrasting examples. The first is Marge, the mother of three diagnosed children, whose transcript was one of the most full of ‘gene talk’ of any in the study. The second is that of Paolo; he has two siblings with a diagnosis of schizophrenia, and has also received a (non-psychotic) psychiatric diagnosis himself. His transcript was the least full of ‘gene talk’. (It should be emphasized, therefore, that there was no *necessary* connection between prevalence of psychiatric diagnoses within a family and the turn to ‘gene talk’ to account for this.)

Marge, very near the start of the interview, and before she had said anything about her three children, was asked to explain why she thought genetics was ‘one of the most important things’ for schizophrenia research to focus on. She replied:

Well, I have a cousin She’s never had a proper job and spent a lot of time in the pubs. ... she was unwell before the age of 20. ... Then ... how old’s Martin? [Around 70.] I would say Martin was about 20 when his mum hanged herself Also my grandmother, when she was [2 secs] in her later stages [of Alzheimer’s], she also had mental illness and so did her sister. Well her sister had mental illness on and off through her life. [...] I have got the family tree upstairs ...

Marge saw no need *explicitly* to make a link between ‘genetic research’ and the enumeration of the various family members who were or had been mentally ill: the clear implication was that ‘genes’ would be able to clarify these instances. Her rapid and rather affectively flat enumeration of instances also demonstrated that the story she would go on to tell – an overwhelming account of her children each in turn developing schizophrenia – did *not* begin with her and her husband, but extended back at least two generations. For a mother engaging with an interviewer whose response to the existence of a schizophrenia diagnosis in three of her children she did not yet know, the stakes of such ‘gene talk’ were likely to be high. The interviewer might, after all, have considered her as responsible for the mental illness of not one, but three children.

Later in the interview, Marge spoke of the traumatic experience of leaving the first of her children to develop schizophrenia ‘in a mental health hospital, having needles stuck in their arm, not knowing that it’s not your fault because of being the hereditary’. Despite her strong assertion that ‘being the hereditary’ means that ‘it’s not your fault’, her interview was suffused with oblique as well as explicit references to the guilt that she felt in relation to her three children having a schizophrenia diagnosis. For example, Marge was aware that others might consider it likely that she would experience guilt: she stated that doctors explain her children’s illness by telling her that it’s ‘half and half’ (half heredity and half other things) – but then added, ‘Whether they tell me that to ease my conscience ... ’.

She also recreated a conversation with one of her children, which attempted to move the origins of schizophrenia away from her nuclear family. In the context of speaking about ‘traits’ running through the family, she said that her daughter:

knows about my aunt [who hanged herself]. She said, ‘Mum, it’s in the family [...] it’s got to come out somewhere’.

The phrase ‘got to come out somewhere’ implies that schizophrenia emerges, like an inevitable yet unpredictable eruption, in various corners of the family: its genesis was not linked to



Marge, the mother, herself. Marge attributes both the doctors and her daughter with a language of heredity, genes and traits, which are used to emphasize Marge's lack of culpability. Her insistence on telling such stories could be interpreted, nonetheless, as betraying her ongoing subjection to precisely such feelings. Her ventriloquization of her daughter – with the phrase 'it's got to come out somewhere' – results, we argue, in her daughter's words being used both to console herself and to convince the interviewer. Here, she movingly stages, in front of the unknown interviewer, one child granting her absolution from the fearful possibility of being the origin of all her children's schizophrenia.

Paulo began his interview, like Marge, by narrating, spontaneously, a family history of mental illness. In contrast, this was one in which 'gene talk' was almost entirely absent. His story started by describing how his two siblings 'that stayed living with my mum' have been in and out of the mental health system for years; a sibling who never lived with mum never received a diagnosis; and he, who lived with his mother for fewer years than his two siblings with a schizophrenia diagnosis, had a less severe psychiatric diagnosis. The narratives he told of the time he did live with his mother were full of extensive abuse (for example, his mother kicking and burning him). His mother he characterized as living in, and propagating to her children, a strange world that had little to do with reality. His mother, he emphasized, 'had her own traumas' – which Paulo explained as resulting from immigration, intense racism and severe marital problems.

Although Paulo affirmed that he did 'have empathy' for his mother, he quite clearly positioned her as the cause of her children's psychiatric illnesses. It is not coincidental, we argue, that he barely used 'gene talk'. He saw himself as having little to contribute vis-à-vis 'genetic research in schizophrenia' – the focus of the overall study – because to his mind, his family's history of mental illness was one characterized by the maternal transmission of trauma, and not via the genetic transmission of schizophrenia. He had no reason to evoke 'gene talk' and a genetic family history of schizophrenia: he had no interest in holding blame away from the symbolic position occupied by the mother.⁴

Holding Blame at Bay?

Are family members' invocations of 'gene talk' successful in the sense outlined by Phelan? Do they 'absolve parents of causal responsibility' (Phelan, 2005)? Or do they, analogously with Singh's argument as regards brain-based models of ADHD, relieve family members of guilt

4 The majority of siblings whom we interviewed were very unlike Paolo in that they clearly desired to keep blame away from their mother. One of the anonymous referees was intrigued as to why these siblings would be equally 'motivated to protect the mother's "innocence" as the mother would be to protect her own'. We were struck by this too, and note, tentatively, that for these siblings, there appeared to be a need either to protect or to identify with their mother, in relation to the blame and guilt – or 'spectre' of blame and guilt, as one sibling put it – that she had had to endure. One sibling recalled his mother, for example, suffering in the face of 'armchair Freudians' who were 'commonplace amongst the liberals of South West London' and who were 'quick to offer [her] rather nasty and judgemental "advice"' when his sister developed schizophrenia. Other siblings were themselves parents (and two of these had children with disabilities): it is likely that this intensified their identification with their mother – not least as regards the anxiety of being positioned as responsible for how their children turned out. We also note that it is possible that our methods of recruitment lessened the likelihood of our speaking to siblings who did not desire to 'protect [their] mother's "innocence"'.

and blame in certain respects, but reconfirm other kinds of judgement and responsibility (Singh, 2004)? In some respects, Phelan's conjecture is borne out. Similar to other studies, some family members expressed clear enthusiasm that genetics could provide relief from overwhelming feelings of responsibility:

Emma (*sister*) wanted to share news about genetic research with her mother, and noted that 'people talk about nurture so much with schizophrenia' and genetic research would 'make her [mother] feel less responsible from a nurture point of view'.

Barbara (a mother) asserted that the origins of schizophrenia are 'largely' or 'at least significantly partly genetic', and that 'the cause of my daughter's illness are more to do with genetics than environmental things'. In recalling environmental factors mentioned in a lecture by a prominent psychiatric researcher – 'being an immigrant', 'being abused', 'abandoned as a child', 'smoking cannabis in adolescence' – she stressed that her daughter 'doesn't have any of them'. In comparison with the guilt and blame that she said she would feel if she hadn't supervised her daughter well enough to prevent cannabis use, or had exposed her to 'gross neglect and abuse', she argued that 'genes ... seem a whole lot better than that sort of scenario, right?' Barbara stressed her familiarity with current scientific research – specifically as regards environmental factors that contribute to schizophrenia. Her belief that the causes of her daughter's schizophrenia were largely genetic was based on her conviction that she as a mother has not exposed her to these environmental factors, and in turn served to reduce the guilt that she felt in relation to her daughter's schizophrenia.

Nonetheless, we argue that there were limits to how far the gene talk employed by the family members in our study did and could assuage blame and guilt. The transcripts indicated two significant reasons for this.

The challenge of gene–environment accounts

As noted earlier, no family member in our study endorsed a solely genetic aetiology for schizophrenia, and most presented a 'trigger' and/or 'combination' model of an interaction between genetic/dispositional and environmental factors. Some family members referred to current science (for example, mentioning newspaper articles, or their attendance at lectures on schizophrenia) when offering their thoughts regarding schizophrenia aetiology; for these participants, their 'combination' models were clearly in some ways influenced by current biomedical accounts that stress both genes and environment. For those who did not explicitly refer to current scientific or psychiatric models of schizophrenia, the temporal characteristics of schizophrenia (that is, the fact that most individuals receive a schizophrenia diagnosis after they have exited childhood) seemed to enhance the attraction of an aetiological model that left space for events or factors *beyond* genes or disposition. The predominance of these 'combination' models thereby left family members – most commonly parents – open to the possibility that they would feel responsible vis-à-vis exposing their family member(s) to those factors or events occurring during childhood or adolescence that they considered might contribute to the development of schizophrenia:

Karen (*mother*), while endorsing the role of genetics, repeatedly mentioned the deleterious effect of her divorce – which, notably, she emphasized that she had wanted – on her children, and its likely contribution to her daughter's illness: 'You think, maybe if



[the divorce] hadn't happened, maybe she wouldn't have got involved with the group that she did, and maybe she wouldn't have smoked the cannabis and maybe she would have been okay. It's all maybes, isn't it?'

Previously, we pointed out how the mother Barbara emphatically argued that her daughter's schizophrenia was produced by genetic rather than environmental factors since she did not expose her daughter to those environmental factors. But this was not, in fact, the whole story. Later in the interview, she turned to the fact that she had had toxoplasmosis while pregnant (a factor currently under investigation for potentially raising the risk of schizophrenia). While Barbara said that, 'You can't blame yourself for getting something like that decades ago', she then described how she is tortured by ongoing thoughts that she acquired it because, 'Maybe I was up to something that I shouldn't have been'. She returned, preoccupied, to this, a second time in the interview. Barbara cannot ultimately rest content with focusing on the 'genetic' side of the equation, and is plagued by the thought that, somehow, she has failed to be a good-enough mother, and that that failure has somehow resulted in her child's schizophrenia.

Vivienne, another mother, likewise revealed the 'gap' that gene talk left open. She praised genetic accounts for allowing her to move beyond castigating herself as a mother: Whereas she used to 'think and say, "What have I done wrong? Didn't I do enough for them? Didn't I give them enough love?"', she now knows, 'At the end of the day, it's not you'. But then, just at the moment at which it appeared that gene talk had provided absolution, she added, '*Or the majority of it is not you*' [emphasis added]. This statement is striking in its poignancy. Vivienne's, as well as other family members' transcripts, indicated that gene talk ultimately did not and could not do everything in accounting for the presence of schizophrenia in one's family. It left open the unresolved possibility that, rather than schizophrenia's origins being displaced to earlier forebears, part of the responsibility *might well* still lie with you.

Foresight in the face of a family history of mental illness

The very usefulness of gene talk – for example, its flexibility in underpinning a tale of a long family history of mental illness – posed additional challenges for some family members. The enumeration of that long family history, after all, threatened to beg the question of whether family members should have manifested greater foresight in the face of that family history.

Emma (*sister*) believed that, genetically speaking, schizophrenia had 'come down' through her father's side of the family. But it was her mother who experienced ongoing self-blame. Emma's father had told her mother 'that he thought there was madness in the family ... quite early on in their relationship'; her mother felt responsible, in that 'she could have known what was happening' – both in relation to her husband's (non-psychotic) psychiatric diagnosis and her child's schizophrenia.

Some family members attempted to ward off this further opportunity for self-blame or guilt. They suggested, for example, that they had not 'know[n] a great deal' about this family history, or that the family had retrospectively constructed its history of mental illness *subsequent* to their family member being diagnosed with schizophrenia.

Conclusion

We have provided the first detailed analysis of how, for what purposes and with what consequences family members related to someone with a diagnosis of schizophrenia use ‘gene talk’ when accounting for the presence of schizophrenia in their family (cf Silverman, 2008, as regards autism). For many family members in our sample, ‘gene talk’ provided a scaffold to support a narrative about a family history of mental illness, one that could extend backwards to previous generations, and one in which the point of origin of mental illness was thereby impossible for the current generation to know. We argue that gene talk attempted to relocate the guilt and sense of responsibility that continues to collect around those family members most closely related to the person with the schizophrenia diagnosis – and in particular, around the mother. Gene talk functioned in the service of expelling the ghost of the schizophrenogenia-inducing mother, by pushing back the origins of familial schizophrenia to previous, often already dead, forebears. Notably, such gene talk was flexible enough to enroll a wide variety of (undiagnosed) forebears – including the unmarried, the heavy-drinking, and the socially difficult – into this genealogy of schizophrenia. Further work needs to be carried out to determine if this talk differs from other modes of discussing inheritance and family history in relation to mental illness. Nonetheless, we tentatively suggest that our participants’ frequent combination of a language of genes and of psychiatric symptoms might have allowed them to interpret a greater number and wider range of behaviours in their relatives and forebears as phenotypic manifestations of underlying genotypic similarities than might have been the case with, say, a discourse of family madness. If so, this could have implications as regards the potency of psychiatric genetics in calling forth new bonds of kinship amongst members of a family within which there is at least one diagnosis of mental illness (cf. Chilibeck *et al*, 2011; Lock, 2011).

We also argue that it was unclear how successful, ultimately, such ‘gene talk’ was in holding (self-)blame and guilt at bay. As research on other illnesses has made clear, there are a variety of ways in which people can feel and express guilt – or not – in relation to genetic models of transmission and inheritance (for example, Hallowell *et al*, 2006). In relation to psychiatric diagnoses, Baart and Abma (2011) have mentioned how some family members who participated in a scientific psychiatric genetics project in the Netherlands initially resisted some of the non-monogenetic theories involving complex gene-environment models that emerged from the late 1990s onwards. These family members had wanted to embrace biological explanations for mental illness as ‘a way out of the feelings of guilt, stigma and responsibility created by the earlier psychodynamic theories’. Baart and Abma stress the difficulties that the newer models posed, since ‘environment as a risk factor (pregnancy, migration, cannabis, possibly trauma) is again back in the limelight, evoking the 1970s where family relations were held accountable for the development of schizophrenia’. In our study, family members did not shy away from discussing the environment as a risk factor. Indeed, their extensive reflections on social factors – including pregnancy, cannabis, migration and trauma – implied that they found solely genetic accounts of causality ultimately unsatisfying, and meant that their ‘gene talk’ left gaps into which their feelings of potential culpability (particularly if they were mothers) easily flowed back in. It is possible to conjecture that if these family members had used gene talk to endorse *solely* genetic models of schizophrenia aetiology, these feelings of potential culpability might have been reduced. However, given the



second problem that we located – the implicit demand to have foresight in the face of one's family history of mental illness – we suggest that it is unlikely that any model of genetic transmission would, for our group of family members, have provided absolution from a sense of guilt.

This was of course not a naturalistic study, and we do not claim that participants would use 'gene talk' in exactly the same way outside of our research interview. Indeed, we have argued that 'gene talk' was a powerful means through which family members shaped and structured the fraught, intersubjective space of a research interview with an unknown interviewer. It is likely, then, that participants used 'gene talk' more frequently than they might have done in other settings, both because of these intersubjective characteristics, and because they consented to an interview that they knew would explore their views regarding genetic research on schizophrenia. It is also possible that our sample was, overall, more knowledgeable about and/or favourable towards genetic research than many other family members in other settings might have been. While a significant proportion of our family members had not completed the equivalent of 'A' levels/a high school diploma and/or a university degree, the methods we used for recruitment, and the reputation the institution from which the study was conducted has for biomedical psychiatric research, might well have oversampled people with an interest in current scientific research regarding schizophrenia. Although these are limitations in some senses (Potokar *et al*, 2011), they do not detract from our primary focus on understanding how and why family members might employ 'gene talk'. These limitations in fact also strengthen our argument concerning the constraints of 'gene talk' in absolving family members of guilt and (self-)blame. If these family members, who largely enthusiastically endorsed the role of genetics in the aetiology of schizophrenia, were not, ultimately, wholly comforted or absolved from guilt by their own 'gene talk', then it is unlikely that biogenetic accounts of mental illness will, across larger samples, result in absolution for family members, particularly parents.

Our analysis here complicates the commonly held view that the schizophrenogenic mother and family have been ousted as schizophrenia has been repositioned as a brain disorder (Harrington, 2012). Many mental health advocacy organizations emphasize in their promotional material that 'Families do not cause schizophrenia or manic depression' (for example, Rethink, 2008). Such statements implicitly acknowledge, we argue, that familial blame for schizophrenia is still common – both on the part of family members themselves and on the part of the general public. Mothers – as a number of studies have made clear – are both frequently culturally positioned, and indeed just as frequently position themselves, as the location at which such blame concentrates (Ferriter and Huband, 2003; Singh, 2004; Moses, 2010; Lautenbach *et al*, 2012). 'Gene talk' in relation to schizophrenia aetiology can be understood, therefore, as one of a number of resources that family members marshal in a not altogether successful attempt to weaken the grip of a historically dense and still culturally prevalent ideology of motherhood as the crucible from which psychopathology issues. It is too easy to assume that the schizophrenogenic mother and family have been dethroned by the triumph of neuroscientific and polygenic models of schizophrenia. As Jonathan Metzl powerfully demonstrated in *Prozac on the Couch*, it is inaccurate to imagine that the recent history of psychiatry can be encapsulated by the replacement of psychoanalysis by biology, pharmacology and neuroscience. Mid-twentieth century models of psychopathology, motherhood and childhood development have not dissipated so easily, even as

biological psychiatry vociferously persists in announcing their supersession (Metzl, 2003). Some voices acknowledge that widespread assertions regarding this supersession of psychogenic models of schizophrenia have resulted in the creation of certain, unhelpful taboos – whether explicit or implicit – regarding the kinds of conversations it is possible to have about schizophrenia (for example, Johnstone, 1999; Harrington, 2012). In short, to use Harrington’s formulation, ‘Fear of giving any energy to discredited models of family blaming means that, today, cultural and psychosocial questions relating to schizophrenia are rarely discussed’ (Harrington, 2012, p. 1293). Given this context, it was particularly striking that many of the family members we interviewed addressed ‘cultural and psychosocial questions’; demonstrated ambivalence and doubt over whether the ghost of the schizophrenogenic mother has been laid to rest; and expressed feelings of responsibility – if not culpability – in relation to their family members’ schizophrenia. This indicates that it should not be assumed that family members necessarily uphold taboos regarding the kinds of conversations it is possible to have about schizophrenia. After all, as one of our participants, Karen, herself a mother, wryly noted, when discussing all the things that she might have done that could have led to her daughter acquiring schizophrenia, ‘I suppose mothers blame themselves for everything’. We suggest that, at best, gene talk works to displace rather than wholly to dissolve the blame and guilt that pervade family members’ discussions of schizophrenia aetiology.

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